Annals of Case Reports
Gaizauskiene K, et al. Ann Case Rep: 7: 870.
www.doi.org/10.29011/2574-7754.100870
www.gavinpublishers.com

OPEN ACCESS

GAVIN PUBLISHERS

Case Report

Covid-19 Related Myocarditis: A Case Report

Karolina Gaižauskienė[1*], Darius Palionis[2], Sigita Glaveckaitė[3], Nomeda Valevičienė[2]

[1]Radiology and Nuclear Medicine Center, Vilnius University Hospital Santaros Klinikos, Vilnius, Lithuania

[2]Department of Radiology, Nuclear Medicine and Medical Physics, Institute of Biomedical Sciences, Vilnius University Faculty of Medicine, Vilnius, Lithuania

[3]Clinic of Cardiac and Vascular Diseases, Institute of Clinical Medicine, Vilnius University Faculty of Medicine, Vilnius, Lithuania

***Corresponding author**: Karolina Gaižauskienė, Radiology and Nuclear Medicine Center, Vilnius University Hospital Santaros Klinikos, Santariškių str. 2, LT-08661 Vilnius, Lithuania

Citation: Gaizauskiene K, Palionis D, Glaveckaite S, Valeviciene N. (2022) Covid-19 Related Myocarditis: A Case Report. Ann Case Report 7: 870. DOI: 10.29011/2574-7754.100870

Received: 11 June 2022; **Accepted:** 15 June 2022; **Published:** 17 June 2022

Abstract

Background: To date, about 524 million cases have been diagnosed with COVID-19 infection worldwide. Cardiovascular complications of the COVID-19 infection such as acute coronary syndrome, heart failure, cardiogenic shock, arrhythmias, and myocarditis have been reported.

Case Summary: A 29-year-old man presented with shortness of breath and swelling of the legs and genitals. Three months ago, the patient had a Covid-19 infection and pneumonia. Since then, shortness of breath has developed and progressed. ECG revealed sinus tachycardia and no ST-T elevation. Laboratory blood tests showed increased BNP, troponin I, D-dimer, and C-reactive protein levels. Transthoracic echocardiogram showed globally impaired left ventricular (LV) and right ventricular (RV) systolic function, enlargement of all heart chambers, fluid in the pericardial cavity, thrombus in the LV apex, and the right atrium (RA). Chest CTA revealed bilateral PE, LV, and RA thrombi, pericardial fluid, and bilateral lung infarcts. An intra-aortic balloon pump (IABP) was introduced. Later cardiac magnetic resonance imaging (MRI) was performed and revealed mid-myocardial and sub-epicardial late gadolinium enhancement (LGE) of the LV posterior and lateral walls and interventricular septum and signs of minimal focal edema in the midventricular segments. Patchy LGE was seen in the junction of both ventricles. Structural changes in the myocardium were characteristic of subacute-chronic myocarditis and dilated CMP.

Discussion: Myocarditis after COVID-19 infection has been the most common cardiovascular complication. Cardiac MRI is the first-choice non-invasive diagnostic method to evaluate structural myocardial changes in acute or chronic myocarditis.

Keywords: Myocarditis; COVID-19; Cardiac MRI; Cardiac imaging; Case report

Introduction

To date, about 523 million cases have been diagnosed with COVID-19 infection worldwide [1]. Cardiovascular complications of the COVID-19 infection such as acute coronary syndrome, heart failure, cardiogenic shock, arrhythmias, and myocarditis have been reported [2]. The cardiac injury occurs in 20% to 30% of hospitalized patients with COVID-19 infection [3,4]. Myocarditis after COVID-19 infection has been the most common cardiovascular complication accounting for 7%-23% of the cases and is 2 to 3 times higher than non-COVID-19 controls [5]. Acute

myocarditis occurrence is between 2.4 and 4.1 out of 1000 patients hospitalized for COVID-19 infection [6]. The study in the US reported that the association between COVID-19 and myocarditis is lowest in persons aged 25–39 years and higher among younger (<16 years) and older (≥50 years) age groups [7]. Acute myocarditis is associated with significant morbidity and mortality [2].

Case Presentation

A 29-year-old man with no previously known cardiovascular disease and other comorbidities has presented to the emergency department with shortness of breath, swelling of the legs and genitals, and general weakness. In the anamnesis, three months ago the patient had a Covid-19 infection, and pneumonia, therefore was treated with antibiotics. Since then, shortness of breath has developed and progressed. Now about a week, the condition has greatly deteriorated. The patient was vaccinated with two doses of the Covid-19 vaccine. On admission, the general condition was severe, body temperature was 35,6°C, blood pressure 135/80 mmHg, heart rate 151 beats/min, and oxygen saturation 96% on room air. On auscultation, wet bilateral basal lung and right lung crackles were heard. An electrocardiogram (ECG) revealed sinus tachycardia, with a rate of 144 beats/minute, and no ST-T changes (Figure 1).

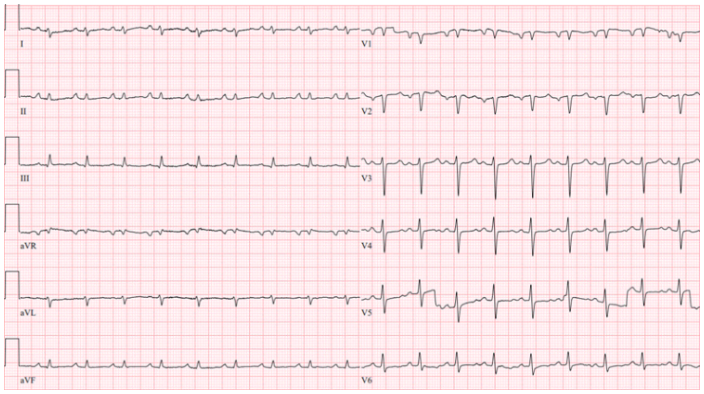

Figure 1: An electrocardiogram showed sinus tachycardia without ST-T changes.

Initial laboratory blood tests showed increased BNP level: 2897,7 ng/l, troponin I level: 221 ng/l (normal value (N) for man: ≤35 ng/l), D-dimer: 15670 µg/L (N: <250), WBC: 12,82 *10^9/l (N: 4-9,8), C-reactive protein: 116,3 mg/l (N: ≤5 mg/l), AST: 1015 U/l (N: <=40), ALT: 550 U/l (N: <=40), ALP: 159 U/l (N: 40-150), GGT: 162 U/l (N: <=36). SARS-CoV-2 (GeneXpert): negative. A patient underwent a transthoracic echocardiogram (TTE). It showed globally impaired left ventricular (LV) and right ventricular (RV) systolic function with ejection fraction (EF) of about 15-20%. Enlargement of all heart chambers with LV diameter of 7.5 cm. Mild mitral and mild-to-moderate tricuspid functional regurgitation. Fluid in the pericardial cavity at LV up to 1.2 cm. Thrombus in the LV apex up to 1.5 cm thick and in the right atrium (RA) measuring 3.5 x 2.2 cm. Signs of systemic venous stasis and ascites. A small amount of fluid in the right pleural cavity (Figure 2).

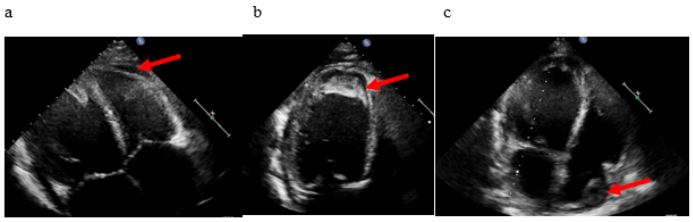

Figure 2: Transthoracic echocardiogram revealed (a) enlargement of all heart chambers, fluid in the pericardial cavity, (b) thrombus in the LV apex, and (c) thrombus in the right atrium. (a-c) apical four-chamber view, (b) apical two-chamber view.

Chest X-ray revealed right lower lung lobe consolidation. Emergency CTA was not performed for pulmonary embolism (PE) exclusion, because the patient was unable to lie down during the study. In the presence of acute heart failure, the patient was hospitalized in cardiac ICU for further management. After evaluation of all the data, a clinical diagnosis of acute myocarditis, acute heart failure, and suspected PE was made. The patient was consulted by a gastroenterologist, and it was concluded that ascites and elevations in liver enzymes are reactive in cardiac pathology. Laboratory blood test for CMV, Epstein- Barr, HIV, Coxsackievirus, Toxoplasma gondii, and Treponema pallidum was negative. Ultrasound examination of lower limbs revealed no thrombi in the deep and superficial veins of both legs. Allowing the condition, a patient underwent chest CTA. It revealed bilateral PE, LV and RA thrombi, pericardial fluid, a small amount of fluid in the pleura, and bilateral lung infarcts (Figure 3).

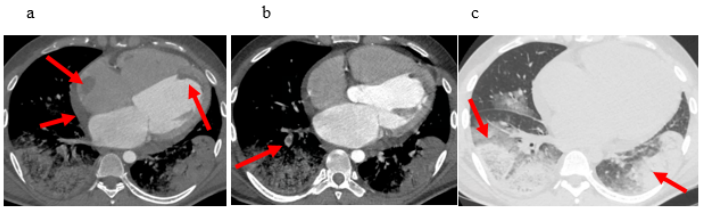

Figure 3: Chest CTA revealed (a) left ventricular and right atrium thrombi, pericardial fluid (b) pulmonary thrombi in the segmental branch of the right pulmonary artery, and (c) bilateral ground glass and consolidation zones showing lung infarcts.

On the fourth day of hospitalization, the patient's hemodynamics became unstable. Therefore, an intra-aortic

balloon pump (IABP) was introduced, and noradrenaline and dobutamine were administered. Hemodynamics stabilized and after nine days IABP was removed. Cardiac magnetic resonance imaging (MRI) was performed to evaluate myocardial structural changes in suspected acute myocarditis. Cardiac MRI revealed enlargement of all heart chambers, globally impaired LV, and RV systolic function with EF <20%. Thrombi in LV apex and superior vena cava. Fluid in the pericardium and pleural cavities. The late gadolinium enhancement (LGE) sequence showed mid-myocardial enhancement in the interventricular septum and sub-epicardial enhancement of the LV posterior wall of the basal segments, also focal mid-subepicardial LGE of the LV lateral wall of the mid-ventricular and basal segments with signs of minimal focal edema in the midventricular segments. Patchy LGE was seen in the junction of both ventricles. Structural changes in the myocardium were characteristic of subacute-chronic myocarditis and dilated CMP (Figure 4). Endomyocardial biopsy was not performed.

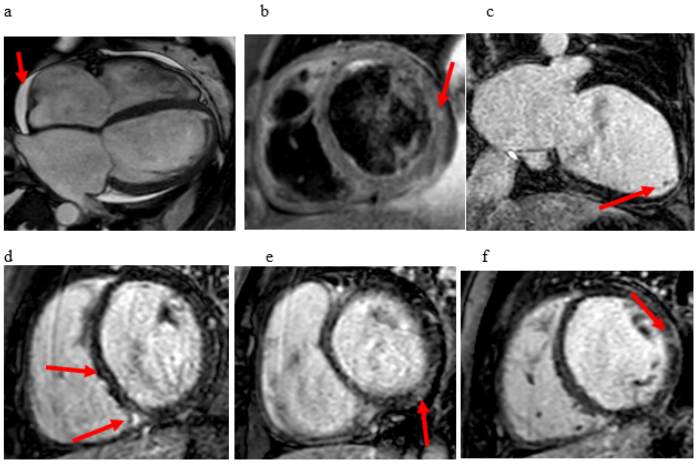

Figure 4: Cardiac magnetic resonance imaging: (a) four-chamber view shows an enlargement of all heart chambers, fluid in the pericardium, (b) T2_STIR sequence shows focal edema in the midventricular segment of lateral LV wall, (c) two-chamber view LGE sequence shows thrombus in the LV apex. Short-axis views, LGE sequence: (d) shows patchy LGE in the junction of both ventricles, mild mid-myocardial enhancement in the interventricular septum, (e) sub-epicardial enhancement of the LV posterior wall, (f) focal mid-subepicardial LGE of the LV lateral wall of the mid-ventricular segments.

The patient was treated in the ICU for eighteen days, and later in the department of Cardiology for eight days. The patient received medical treatment and IABP was introduced. During treatment the patient's condition improved significantly, shortness of breath decreased, and exercise tolerance and laboratory results improved. The patient was discharged for further outpatient treatment and follow-up by a cardiologist.

Discussion

After evaluating all the data, the above-described case illustrates COVID-19-related myocarditis. Unfortunately, an endomyocardial biopsy was not performed. Patient symptoms typically include fatigue, dyspnea, chest pain, chest tightness, in severe cases tachycardia, acute-onset heart failure with cardiogenic shock, arrhythmia, and sudden cardiac death, signs of right-sided heart failure [2,8]. Most patients present with elevated inflammatory markers, troponin levels, brain natriuretic peptide (BNP), and N-terminal prohormone BNP (NT-proBNP) could be elevated [8]. In most patients with COVID-19-related myocarditis, an ECG may reveal sinus tachycardia, ectopic beats, ST elevation, T-wave inversion, PR depression, new-onset bundle branch block, QT prolongation, pseudoinfarct pattern, premature ventricular complexes, and bradyarrhythmia with an advanced atrioventricular block [4,8]. In the cases of acute myocarditis, TTE may reveal normal or reduced LV EF, increased wall thickness, mild segmental hypokinesia, LV diastolic dysfunction, abnormal tissue Doppler imaging, LV thrombus, mild RV dysfunction, pericardial effusion, and abnormal myocardial echogenicity [2,9]. Cardiac MRI is an important non-invasive imaging tool to confirm myocarditis. The revised Lake Louise Criteria (LLC) defines cardiac MRI-specific diagnostic requirements [4,10]. The diagnostic findings of myocarditis include edema, seen as an increased signal intensity in T2-weighted images and increased T1 and T2 mapping values, hyperemia, seen as an uptake in early gadolinium enhancement images, and necrosis, seen as an uptake in late gadolinium enhancement (LGE) images [2,9,10]. Increased ECV shows edema (extracellular), hyperemia/capillary leak, necrosis, and fibrosis [10]. Cardiac MRI reveals typical mid-subepicardial non-ischemic LGE in myocarditis [10]. Cardiac MRI can give additional information, such as ventricular EF, quantitation of heart chamber volumes, quantitation of blood flow, and ventricular mass. Cardiac MRI is a useful tool for patients who are followed up after acute myocarditis. Usually, cardiac MRI is performed 7 to 10 days after the onset of acute disease. Edema usually reduces 4 weeks after disease onset and disappears, while LGE generally persists, but its extent reduces [9].

Conclusion

Myocarditis after COVID-19 infection has been the most common cardiovascular complication and is associated with significant morbidity and mortality. Cardiac MRI is the first-choice non-invasive diagnostic method to evaluate structural myocardial changes in acute or chronic myocarditis. Patients should be followed up by a cardiologist to assess long-term consequences after COVID-19-related myocarditis.

References

1. Coronavirus (Covid-19).
2. Adeboye A, Alkhatib D, Butt A, Yedlapati N, Garg N (2022) A Review of the Role of Imaging Modalities in the Evaluation of Viral Myocarditis with a Special Focus on COVID-19-Related Myocarditis. Diagnostics 12: 549.
3. Ismayl M, Abusnina W, Thandra A, Sabri A, Groh D, et al (2022) Delayed acute myocarditis with COVID-19 infection. PROC (BAYL UNIV MED CENT) 35: 366-368.
4. Chimenti C, Magnocavallo M, Ballatore F, Bernardini F, Alfarano M, et al (2022) Prevalence and Clinical Implications of COVID-19 Myocarditis. Card Electrophysiol Clin 14: 53-62.
5. Priyadarshni S, Westra J, Kuo YF, Baillargeon JG, Khalife W, et al (2022) COVID-19 Infection and Incidence of Myocarditis: A Multi-Site Population-Based Propensity Score-Matched Analysis. Cureus 14: e21879.
6. Ammirati E, Lupi L, Palazzini M, Hendren NS, Grodin JL, et al (2022) Prevalence, Characteristics, and Outcomes of COVID-19–Associated Acute Myocarditis. Circulation. 145: 1123-1139.
7. Boehmer TK, Kompaniyets L, Lavery AM, Hsu J, Ko MY, et al (2021) Association Between COVID-19 and Myocarditis Using Hospital-Based Administrative Data-United States, March 2020–January 2021. MMWR Morb Mortal Wkly Rep. 70: 1228-1232.
8. Siripanthong B, Nazarian S, Muser D, Deo R, Santangeli P, et al (2020) Recognizing COVID-19–related myocarditis: The possible pathophysiology and proposed guideline for diagnosis and management. Heart Rhythm. 17: 1463-1471.
9. Ammirati E, Frigerio M, Adler ED, Basso C, Birnie DH, et al (2020) Management of Acute Myocarditis and Chronic Inflammatory Cardiomyopathy. Circ Heart Fail. 13: e007405.
10. Ferreira VM, Menger JS, Holmvang G, Kramer CM, Carbone I, et al (2018) Cardiovascular Magnetic Resonance in Nonischemic Myocardial Inflammation: Expert Recommendations. J Am Coll Cardiol. 72: 3158-3176.