Acute heart failure in the course of fulminant myocarditis requiring mechanical circulatory support in a healthy young patient after coronavirus disease 2019

Kamil Marcinkiewicz[1], Joanna Petryka-Mazurkiewicz[2, 4], Michał M Nowicki[1], Jarosław Kuriata[3], Zofia Dzielińska[2], Marcin Demkow[2], Anna Konopka[1]

[1]Department of Intensive Cardiac Therapy, The Cardinal Stefan Wyszynski Institute of Cardiology, Warszawa, Poland
[2]Department of Coronary and Structural Heart Diseases, The Cardinal Stefan Wyszynski Institute of Cardiology, Warszawa, Poland
[3]Department of Cardiac Surgery and Transplantology, The Cardinal Stefan Wyszynski Institute of Cardiology, Warszawa, Poland
[4]Magnetic Resonance Unit, The Cardinal Stefan Wyszynski Institute of Cardiology, Warszawa, Poland

Correspondence to:
Kamil Marcinkiewicz, MD, Department of Intensive Cardiac Therapy, The Cardinal Stefan Wyszynski Institute of Cardiology, Alpejska 42, 04–628 Warszawa, phone: +48 22 343 43 14, e-mail: marcinkiewicz.kamil@gmail.com

Kardiol Pol. 2021; 79 (5): 583–584; DOI: 10.33963/KP.15888

Received: February 4, 2021

Revision accepted: March 5, 2021

Published online: March 19, 2021

The novel coronavirus (SARS-CoV-2) responsible for coronavirus disease 2019 (COVID-19) not only affects the respiratory system but may have significant cardiovascular effects as well, causing arrhythmias, heart failure, myocarditis, and coagulation abnormalities [1].

A 20-year-old male with no prior cardiac medical history was admitted to our hospital with fever and dyspnea. Six weeks before admission he suffered from diarrhea shortly after his 10-year-old brother presented similar symptoms. Echocardiography performed 5 months earlier was unremarkable.

After admission, the patient deteriorated due to acute heart failure. Both NT-proBNP (26000 pg/ml [n <125 pg/ml]) and troponin T (850 ng/l [n <14 ng/l]) levels were significantly elevated on admission. Echocardiography revealed a severely lowered left ventricular ejection fraction (LVEF) of 15%. The patient required norepinephrine and dobutamine in increasing doses. Real-time PCR of his nasopharyngeal swab for the presence of SARS-CoV-2 RNA returned negative.

A blood test showed elevated inflammatory markers. Multiple tests were performed in search for the origin of the infection — however, both bacterial and viral causes were excluded.

Shortly after, an intra-aortic balloon pump (IABP) was implanted with mild improvement. The following day, due to complete cardiovascular insufficiency, veno-arterial extracorporeal membrane oxygenation (ECMO) was implanted. The patient improved quickly with this treatment and on the fourth day from ECMO implantation his LVEF increased to 48% and myocardium thickness increased to 20–21 mm, suggesting edema (Figure 1). Both ECMO and IABP were explanted after 6 days of therapy.

Magnetic resonance imaging revealed a small left ventricular cavity without regional wall motion abnormalities and a dynamic LVEF of 69% (Figure 1). On T2-weighted imaging, the myocardial signal was globally increased. Delayed late gadolinium imaging showed diffuse fibrosis in the anteroseptal and inferior walls. These findings were in keeping with acute myocarditis.

The diagnosis of fulminant myocarditis due to COVID-19 infection was confirmed (serological tests were positive for IgG and negative for IgM 8 weeks after first gastrointestinal tract symptoms).

Involvement of the cardiovascular system may occur in patients suffering from COVID-19 despite the absence of upper respiratory tract infection (URTI) symptoms. Several possible mechanisms of myocardial injury during COVID-19 infection have been discussed [2]. Surprisingly, myocarditis and other cardiovascular symptoms appear in COVID-19 patients after a prolonged period (up to 10–15 days) counting from the initial onset of URTI symptoms [3]. At this point, no viral particles may be detected. Myocarditis may be due to both plain viral invasion and an exaggerated secondary immune response. We hypothesize that the latter may be the pathomechanism of our patient's fulminant myocarditis.

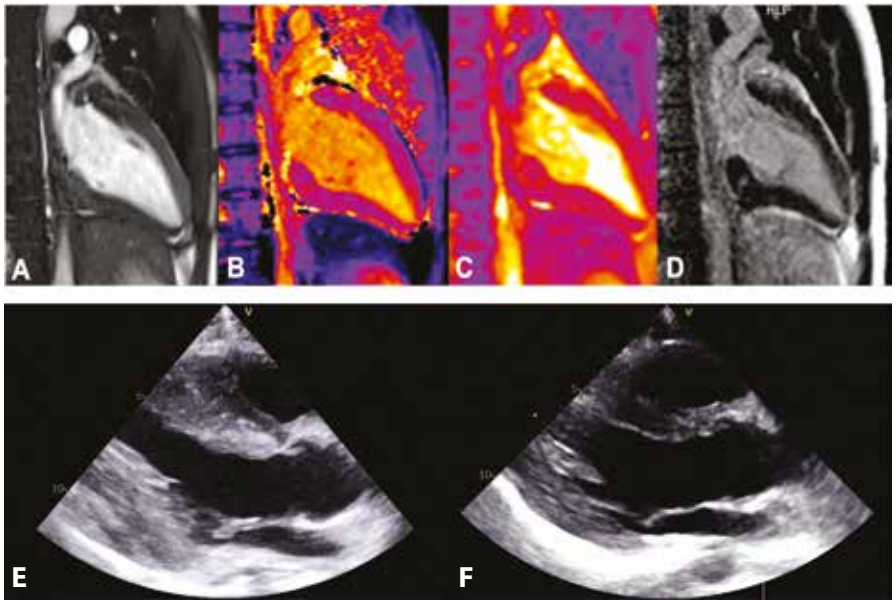

Figure 1. A–D. Post COVID-19 myocarditis on cardiac magnetic resonance — two-chamber images: **A.** cine; **B.** T1-mapping; **C.** T2-mapping; **D.** late gadolinium imaging. Images **B–D** show increased mid-wall signal in the basal inferior segment and increased subepicardial signal in the basal anterior segment. **E–F.** Wall thickness dimensions in the parasternal LAX view by TTE. **E.** Thickened LV wall (during hospitalization). **F.** Normal LV thickness (at discharge). The time difference between those two images is 13 days

SARS-CoV-2 induced fulminant myocarditis is an uncommon clinical presentation, with a mortality rate of approximately 40%–70% [4]. Nonetheless, the application of circulatory support systems, including IABP, Impella implantation, or ECMO might be beneficial for these patients. The hemodynamic rule of unloading the inflamed myocardium, which reduces wall stress and decreases myocardial oxygen requirements, supports myocardial recovery and is a viable treatment option in patients with fulminant myocarditis [5].

Our case report provides a unique insight into the traits of acute heart failure caused by fulminant myocarditis after a SARS-CoV-2 infection highlighting the value of mechanical circulatory support in these patients.

Article information

Conflict of interest: None declared.

Informed consent: The authors certify that they have obtained the appropriate patient consent form. In the form, the patient has given his consent for his clinical information to be reported in the journal.

How to cite: Marcinkiewicz K, Petryka-Mazurkiewicz J, Nowicki MM, et al. Acute heart failure in the course of fulminant myocarditis requiring mechanical circulatory support in a healthy young patient after coronavirus disease 2019. Kardiol Pol. 2021; 79(5): 583–584, doi: 10.33963/KP.15888.

REFERENCES

1. Guzik TJ, Mohiddin SA, Dimarco A, et al. COVID-19 and the cardiovascular system: implications for risk assessment, diagnosis, and treatment options. Cardiovasc Res. 2020; 116(10): 1666–1687, doi: 10.1093/cvr/cvaa106, indexed in Pubmed: 32352535.
2. Shi S, Qin Mu, Shen Bo, et al. Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. JAMA Cardiol. 2020; 5(7): 802–810, doi: 10.1001/jamacardio.2020.0950, indexed in Pubmed: 32211816.
3. Zhou F, Yu T, Du R, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. Lancet. 2020; 395(10229): 1054–1062, doi: 10.1016/S0140-6736(20)30566-3, indexed in Pubmed: 32171076.
4. Ammirati E, Veronese G, Brambatti M, et al. Fulminant versus acute nonfulminant myocarditis in patients with left ventricular systolic dysfunction. J Am Coll Cardiol. 2019; 74(3): 299–311, doi: 10.1016/j.jacc.2019.04.063, indexed in Pubmed: 31319912.
5. McCarthy RE, Boehmer JP, Hruban RH, et al. Long-term outcome of fulminant myocarditis as compared with acute (nonfulminant) myocarditis. N Engl J Med. 2000; 342(10): 690–695, doi: 10.1056/NEJM200003093421003, indexed in Pubmed: 10706898.