VIA MEDICA

LETTER TO THE EDITOR

Cardiology Journal
2021, Vol. 28, No. 2, XXX–XXX
DOI: 10.5603/CJ.a2021.0028

ISSN 1897–5593
eISSN 1898–018X

Post-COVID-19 heart syndrome

Aleksandra Gasecka[1, 2], Michał Pruc[3], Katarzyna Kukula[3, 4], Natasza Gilis-Malinowska[5], Krzysztof J. Filipiak[1], Milosz J. Jaguszewski[5], Lukasz Szarpak[4, 6]

[1]1st Chair and Department of Cardiology, Medical University of Warsaw, Poland
[2]Department of Cardiology, University Medical Center Utrecht, The Netherlands
[3]Polish Society of Disaster Medicine, Warsaw, Poland
[4]Maria Sklodowska-Curie Medical Academy in Warsaw, Poland
[5]1st Department of Cardiology, Medical University of Gdansk, Poland
[6]Maria Sklodowska-Curie Białystok Oncology Center, Białystok, Poland

This paper was guest edited by Prof. Anna Tomaszuk-Kazberuk

To date, 92,111,432 of coronavirus disease 2019 (COVID-19), cases were confirmed worldwide and the number of asymptomatic patients remains largely unknown. There are emerging retrospective data implying that the COVID-19 infection has long-term complications, although there is still a paucity of large, prospective trials to investigate the true prevalence of these complications. Besides lung inflammation, myocardial injury is a typical COVID-19-related phenomenon, present in 20–30% of patients and contributing to 40% of deaths [1]. However, myocardial injury in the course of COVID-19 may be even more prevalent [2].

An autopsy study including 39 patients who had died due to COVID-19 showed features of myocardial abnormalities in patients, in whom the cardiac complications had not previously been diagnosed [3]. Histopathologic evaluation of the myocardium did not fulfil the criteria of acute myocarditis, but in 62% patients (24/39) the presence of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was confirmed within the myocardium. Among them, 67% of patients (16/24) demonstrated evidence of myocardial virus replication, as defined by a virus load above 1,000 virus copies per μg RNA. In addition, the cytokine response panel consisting of 6 proinflammatory genes was increased in those 16 patients, compared with patients without SARS-CoV-2 in the heart, but this had not (yet) been associated with an influx of inflammatory cells. As assessed using in situ hybridization, interstitial cells and infiltrating macrophages, but not cardiomyocytes were the most probable virus localization within the myocardium [3].

The silent but progressive myocardial injury in the course of COVID-19 might contribute to the development of heart failure and other cardiovascular complications following virtual recovery. This hypothesis is confirmed by the results of another study, where the authors performed cardiac magnetic resonance in 100 COVID-19 convalescents at 2 to 3 months following the acute phase of the disease [4]. Persistent cardiac involvement was observed in 78 (78%) patients and ongoing myocardial inflammation in 60 (60%) patients, which was independent of the severity and overall course of the acute disease and the time from the original diagnosis. Moreover, increased troponin concentration was demonstrated in 76 (76%) of patients without any clinically overt signs and symptoms of myocardial dysfunction.

In another study including 139 healthcare workers with confirmed past SARS-CoV-2 infec-

Address for correspondence: Aleksandra Gąsecka, MD, PhD, 1st Chair and Department of Cardiology, Medical University of Warsaw, ul. Banacha 1a, 02–097 Warszawa, Poland, tel: +48 22 599 19 51, e-mail: aleksandra.gasecka@wum.edu.pl

Received: 19.01.2021 Accepted: 22.02.2021

tion, cardiac magnetic resonance features of myocarditis were observed in 37% of the participants at a median of 10 weeks after infection [5]. Importantly, only half of the participants had symptoms of COVID-19, demonstrating that cardiac sequelae might be associated with an altered or delayed immune response, and that even asymptomatic patients and/or patients not aware of the infection may suffer from serious cardiovascular complication in the longer perspective.

The long-term health consequences of COVID-19 were also evaluated in 1733 patients with COVID-19 in Wuhan, China [6]. Six months following hospital discharge, the main persisting symptoms were fatigue or muscle weakness (1038/1655, 63%), sleep difficulties (437/1655, 26%) and anxiety or depression (367/1733, 23%). In addition, 76% of patients (1265/1655) declared at least one persisting symptom. In addition, 13% (107/822) participants without acute kidney injury and with normal estimated glomerular filtration rate (eGFR more than 90 mL/min/1.73 m^2) in the acute phase had eGFR less than 90 mL/min/1.73 m^2 at follow-up, implying the COVID-19-induced kidney injury [6]. Although cardiovascular imaging was not a part of this study, it is likely that at least a part of patients who reported the fatigue and muscle weakness might have developed cardiac dysfunction.

Altogether, emerging results from the hitherto studies indicate that SARS-CoV-2 infection may be associated with the long-term extrapulmonary organ manifestations, with cardiac involvement being one of the most prevalent. The long-term impact of COVID-19-associated cardiac dysfunction remains unknown. Hence, it is relevant to evaluate the presence of the potential myocardial damage in patients with a history of SARS-CoV-2 infection, even if the course was asymptomatic. Moreover, it is crucial to focus on the group of patients who were not aware of the infection, as the post-COVID-19 heart syndrome might be the first indicator of past infection [7]. In the societal perspective, there is a risk that SARS-CoV-2 might further increase the cardiovascular morbidity and mortality [8]. Further long-term studies are required to determine the incidence and clinical course of myocardial damage caused by COVID-19 in order to implement a routine cardiac imaging screening that allows for the treatment of post-COVID-19 heart syndrome.

Conflict of interest: None declared

References

1. Akhmerov A, Marbán E. COVID-19 and the Heart. Circ Res. 2020; 126(10): 1443–1455, doi: 10.1161/CIRCRESAHA.120.317055, indexed in Pubmed: 32252591.
2. Gąsecka A, Filipiak KJ, Jaguszewski MJ. Impaired microcirculation function in COVID-19 and implications for potential therapies. Cardiol J. 2020; 27(5): 485–488, doi: 10.5603/CJ.2020.0154, indexed in Pubmed: 33165898.
3. Lindner D, Fitzek A, Bräuninger H, et al. Association of cardiac infection with SARS-CoV-2 in confirmed COVID-19 autopsy cases. JAMA Cardiol. 2020; 5(11): 1281–1285, doi: 10.1001/jamacardio.2020.3551, indexed in Pubmed: 32730555.
4. Puntmann VO, Carerj ML, Wieters I, et al. Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from coronavirus disease 2019 (COVID-19). JAMA Cardiol. 2020; 5(11): 1265–1273, doi: 10.1001/jamacardio.2020.3557, indexed in Pubmed: 32730619.
5. Eiros R, Barreiro-Perez M, Martin-Garcia A, et al. Pericarditis and myocarditis long after SARS-CoV-2 infection: a cross-sectional descriptive study in health-care workers. , doi: 10.1101/2020.07.12.20151316.
6. Huang C, Huang L, Wang Y, et al. 6-month consequences of COVID-19 in patients discharged from hospital: a cohort study. Lancet. 2021; 397(10270): 220–232, doi: 10.1016/s0140-6736(20)32656-8.
7. Lorente-Ros A, Monteagudo Ruiz JM, Rincón LM, et al. Myocardial injury determination improves risk stratification and predicts mortality in COVID-19 patients. Cardiol J. 2020; 27(5): 489–496, doi: 10.5603/CJ.a2020.0089, indexed in Pubmed: 32589258.
8. Li G, Saguner AM, An J, et al. Cardiovascular disease during the COVID-19 pandemic: Think ahead, protect hearts, reduce mortality. Cardiol J. 2020; 27(5): 616–624, doi: 10.5603/CJ.a2020.0101, indexed in Pubmed: 32789839.