Annals of Agricultural and Environmental Medicine
www.aaem.pl

ORIGINAL ARTICLE

Cardiovascular Magnetic Resonance Imaging in Physically Fit Young Patients Sans Comorbidities Who Recently Recovered from Coronavirus Disease 2019 (COVID-19)

Agnieszka Pawlak[1,2,A-F] ✉, Mariusz Furmanek[1,3,B-E], Piotr Szymański[1,3,A-D], Piotr Ciechanowicz[1,B-D], Irena Walecka[1,3,B-C], Jerzy Walecki[1,3,B-C], Andrzej Fal[1,B-C], Artur Zaczyński[1,B,E], Zbigniew Król[1,A,E-F], Waldemar Wierzba[1,A,E-F], Katarzyna Gil[4,A,C], Robert Gil[1,2,A,E-F]

[1] *Central Clinical Hospital of the Ministry of the Interior and Administration, Warsaw, Poland*
[2] *Mossakowski Medical Research Centre, Polish Academy of Sciences, Warsaw, Poland*
[3] *Centre of Postgraduate Medical Education, Warsaw, Poland*
[4] *The Ohio State University Division of Cardiovascular Medicine, Columbus, United States of America*
A – Research concept and design, B – Collection and/or assembly of data, C – Data analysis and interpretation, D – Writing the article, E – Critical revision of the article, F – Final approval of the article

Pawlak A, Furmanek M, Szymański P, Ciechanowicz P, Walecka I, Walecki J, Fal A, Zaczyński A, Król Z, Wierzba W, Gil K, Gil R. Cardiovascular Magnetic Resonance Imaging in Physically Fit Young Patients Sans Comorbidities Who Recently Recovered from Coronavirus Disease 2019 (COVID -19). Ann Agric Environ Med. 2024;31(1):100–104. doi: 10.26444/aaem/183591

Abstract

Introduction and objective. Multiple studies showed that patients with a severe course of COVID-19 may develop cardiovascular complications. Assessment of the incidence of myocardial injury in young, physically fit male patients with no comorbidities, and asymptomatic/mild course of the disease who recovered from COVID-19.

Material and methods. A prospective, single-center, observational cohort study of 75 young (median[IQR] age 22 years) physically fit male patients, without comorbidities and smoking who recently recovered from COVID-19. Results were compared with a control group of age-matched, physically fit men with no comorbidities who tested negative for SARS-CoV-2.

Results. 19(25%) patients had possible COVID-19 related myocardial injury[PCRMI] on cardiovascular magnetic resonance [CMR] including definitive myocarditis (n=1;1.3%) and possible myocarditis (n=3;4%). Other abnormalities: mildly decreased (<50%) left ventricular(LV) ejection fraction (n=4;5%), increased LV end-diastolic volume index (n=8;11%) and LV mass index (n=9;12%). Patients with PCRMI had higher NT-pro-BNP level (29 vs 20pg/mL respectively, P=0.02) and lower LV ejection fraction (55% vs 59% respectively, P=0.03). PCRMI was demonstrated in 3(27%) volunteers from the control group based on the presence of LGE (2/18%) and decreased LV ejection fraction (1/9%). No volunteer from the control group was diagnosed with definitive or possible myocarditis.

Conclusions. PCRMI was a frequent finding in young, asymptomatic, physically-fit patients sans comorbidities relatively late after recovery from COVID-19. Whereas no definitive or possible myocarditis was found in the control group, LGE was relatively frequent suggesting that our findings might not be COVID-19 specific. This warrants a need for further investigation into the long-term cardiovascular consequences of COVID-19.

Keywords

Cardiovascular Magnetic Resonance Imaging, Coronavirus Disease 2019 (COVID-19), Myocardial injury

INTRODUCTION

The Coronavirus Disease 2019 (COVID-19) pandemic, caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), has posed many challenges for public healthcare systems worldwide [1]. Multiple studies have shown that myocardial infarction, cardiomyopathy, myocarditis, heart failure, arrythmias, and thromboembolic complications may occur in patients with severe course of the disease [2, 3, 4]. In a widely debated prospective observational cohort study published in JAMA Cardiology, Puntmann et al. demonstrated high prevalence of cardiovascular sequelae of COVID-19 using biomarkers of cardiac injury and cardiovascular magnetic resonance (CMR) imaging [5].

We expand these findings in a prospective cohort study of young, asymptomatic male patients without comorbidities, that have recently recovered from COVID-19 and had asymptomatic or mildly symptomatic course of the disease.

OBJECTIVES

Assessment of the incidence of myocardial injury in young, physically fit male patients with no comorbidities, and asymptomatic/mild course of the disease who recovered from COVID-19.

✉ Address for correspondence: Agnieszka Pawlak, Central Clinical Hospital of the Ministry of the Interior and Administration, Warsaw, Poland
E-mail: a.pawlak1@wp.pl

Received: 12.04.2023; accepted: 01.02.2024; first published: 17.06.2024

METHODS

Study Design & Population

This was a prospective observational cohort study that included 75 young, physically fit male patients without comorbidities and smoking who were diagnosed with COVID-19 by RT-PCR on swab test of the upper respiratory tract between April and May 2020. All participants were considered eligible after a minimum of 30, but less than 90, days following negative results of the swab test at the end of isolation. Follow-up studies were performed in the Central Clinical Hospital of the Ministry of the Interior and Administration in Warsaw (CCH MIA) between May and June 2020. Exclusion criteria were refusal to participate or provide informed consent or absolute contraindication to contrast-enhanced CMR imaging. Results were compared with a control group of age-matched, physically fit male volunteers without comorbidities who tested negative for SARS-CoV-2 (from the same firefighter training program as the study group). Control group was limited to 31 patients due to restrictions associated with COVID-19 surge. The enzyme-linked immunosorbent assay (ELISA) tests were used for qualitative detection of serum anti-SARS-CoV-2 IgM, IgA and IgG antibodies (kit produced by Vircell Microbiologist, Granada, Spain: ref. MA1032 and G1032) in the study and control group.

Clinical and demographic characteristics, blood test results, and imaging data were obtained using electronic data capture software on the day of CMR examination. All participants underwent venous blood sampling and echocardiography examination prior to CMR. Blood samples were processed using standardized, commercially available test kits for analysis of C-reactive protein (CRP), N-terminal pro-B-type natriuretic peptide (NT-pro-BNP), procalcitonin (Cobas 8000 System; Roche), and high-sensitivity troponin I (hsTnI) (Architect I 2000).

CMR Data

CMR examinations were performed with a 3T system (3.0T Ingenia, Philips Healthcare). Imaging protocol included short axis (SAX) and horizontal long axis (HLA) cine sequences (for function assessment), SAX and HLA T2 weighted and T2 STIR (short TI inversion recovery) images (for edema detection), SAX and HLA late gadolinium enhancement (LGE) (for detection of necrosis/fibrosis). LGE imaging was performed approximately 10 to 15 minutes after intravenous administration of 0.1 mmol/kg of body weight of gadobutrolum (Gadovist, Bayer AG). Assessment was performed using a professional workstation (QMass 7.1, Medis MIS Inc.). Left ventricular volumes, mass, and function were calculated using automated contour detection with manual correction. Edema and LGE were assessed visually by 2 independent readers. Possible COVID-19 related myocardial injury (PCRMI) was defined as the presence of LGE, edema, pericardial effusion (>5mm fluid layer at least two walls), or systolic wall motion abnormalities [6]. Definitive myocarditis was diagnosed based on Lake-Louis criteria [6]. Possible myocarditis was considered when edema or LGE and pericardial effusion or wall motion abnormalities were present.

The study protocol was approved by the Central Clinical Hospital of the Ministry of Interior and Administration in Warsaw (No. 89/2020). All procedures were performed in concordance with the Declaration of Helsinki and International Conference on Harmonization of Good Clinical Practice. All of the patients provided written informed consent to participate in the study.

Statistical Analysis

Quantitative variables are reported as mean and standard deviation (SD), or for non-normally distributed variables, the median and interquartile range (IQR). Normality of the variables was verified using the Shapiro–Wilk normality test. For categorical variables, the number of observations (N) with the corresponding percentage (%) is given.

To compare 2 independent groups, Student's t-test for quantitative variables with normal distribution or the non-parametric Mann–Whitney U test for non-normally distributed variables were used.

For categorical variables, Pearson's $\chi 2$ test, the maximum likelihood (ML) $\chi 2$ test or $\chi 2$ test with Yates's correction was applied.

The paired sample t-test (for normally distributed quantitative variables) or the nonparametric Wilcoxon signed-rank test (for non-normally distributed quantitative variables).

The results were considered statistically significant at $p < 0.05$. All the calculations were performed using the SPSS software version 13.0 (IBM).

The studied population was analyzed in relation to myocardial injury: patient with or without possible COVID-19 related myocardial injury. The sample size was estimated for the mean effect, ($f2 = 0.15$)

RESULTS

The study included a cohort of 75 young (median [IQR] age – 22 [21–23] years), physically fit male patients without comorbidities who were recruited from a single cluster (firefighter training program) and who recovered from COVID-19. 57 (76%) patients were asymptomatic, and 18 (24%) patients had mild COVID-19 symptoms (did not require hospitalization or treatment due to the presented symptoms). Dysosmia [11 (15%)], dysgeusia [8 (11%)], and atypical chest pain [8 (11%)] were the most frequent COVID-19 symptoms.

The median (IQR) duration between the positive and negative COVID-19 testing was 13 (12–16) days. The majority of patients were asymptomatic on the day of the CMR and laboratory testing (Table 1). Only one patient (n=1; 1.3%) complained of atypical chest pain. Other reported symptoms included rhinitis (n=2; 2.6%), dysgeusia (n=1; 1.3%), and dysosmia (n=4; 5.3%. The median (IQR) duration between the negative COVID-19 testing and the CMR examination was 64 (49–77) days. CMR results are presented in Table 2.

Nineteen (25%) patients had PCRMI including definitive myocarditis (n=1; 1.3%) and possible myocarditis (n=3; 4%) (Figure 1). LGE was present in 16 (21%) patients, most often in the inferior and inferolateral walls (Figure 2, Table 2). Edema was present in 1 (1.3%) and pericardial effusion in 3 (4%) patients. Wall motion abnormalities (LVEF<50%) were observed in 4 (5%) patients. Other abnormal CMR findings included increased LV volume index (n=8; 11%), and LV mass index (n=9 12%) (Table 2). LV ejection fraction was significantly lower (55% vs 59%, P=0.02) and NT-pro-BNP level was significantly higher (29 vs 20 pg/mL, P=0.02) in patients with PCRMI.

Table 1. Patient characteristics

Characteristics	Control group n=31	Study group		*P** value	*P*** value
		Isolation n=75	Follow-up n=75		
Patient characteristics, median (IQR)					
Age, y;	23 (22–24)	22 (21–23)	22 (21–23)	0.99	0.21
Male, No.(%)	11(100)	75 (100)	75 (100)	1.0	1.0
BMI	24(24–25)	24 (23–25)	24 (23–26)	0.31	0.27
Blood pressure, mmHg					
Systolic	120 (114–131)	128 (118–135)	127 (119–137)	0.21	0.23
Diastolic	64 (58–77)	71 (65–75)	71 (66–76)	0.31	0.18
Heart rate, beats per min	61 (55–70)	69 (60–76)	68 (60–74)	0.26	0.12
COVID-19 symptoms, No.(%)					
Fever	N/A	1 (1.3)	0 (0)	0.4	0.7
Cough	N/A	2 (2.6)	0 (0)	0.2	0.5
Shortness of breath	N/A	1 (1.3)	0 (0)	0.4	0.7
Dyspnea	N/A	1 (1.3)	0 (0)	0.4	0.7
Muscular pain	N/A	1 (1.3)	0 (0)	0.4	0.6
Chest pain	N/A	8 (10.7)	1 (1.3)	0.02	0.3
Sore throat	N/A	1 (1.3)	0 (0)	0.4	0.5
Rhinitis	N/A	3 (4.0)	2 (2.6)	0.6	0.5
Dysgeusia	N/A	8 (10.7)	1 (1.3)	0.02	0.26
Dyssomnia	N/A	11 (14.7)	4 (5.3)	0.1	0.18
Asymptomatic	N/A	57 (76)	67 (89)	0.01	0.02

BMI – body mass index; *P study group during isolation vs follow-up; **P study group vs control group.

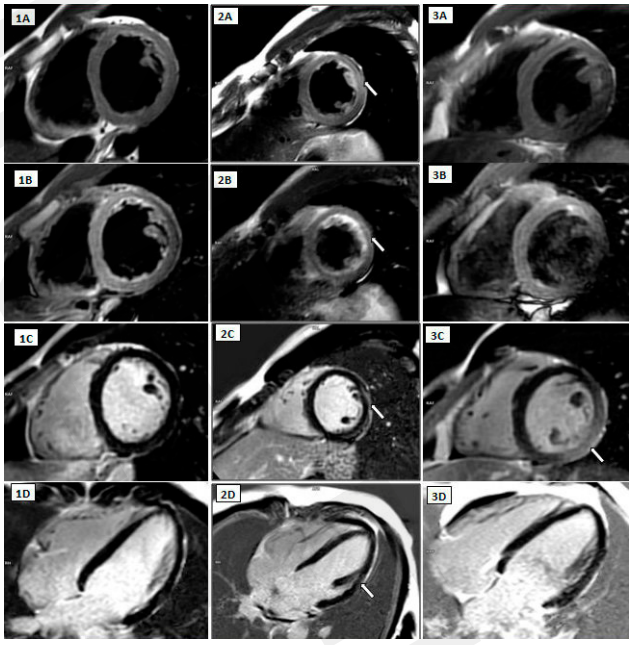

Figure 1. Panel A – SAX T2-weighted images, panel B – T2 STIR images, Panel C – SAX LGE images, Panel D – HLA LGE images.
Panel 1A-D demonstrates images obtained from a patient with no myocardial edema or late gadolinium enhancement.
Panel 2A-D demonstrates images obtained from a patient with PCRMI with visible myocardial edema (Panel 2A-2B; arrows) and subepicardial to midmyocardial late gadolinium enhancement in the mid anterolateral wall (Panel 2C-2D; arrows).
Panel 3A-D demonstrates images obtained from a patient with PCRMI with no visible edema (Panel 2A-2B) and near transmural late gadolinium enhancement in the inferolateral wall (Panel 3C; arrow).
SAX – short axis; HLA – horizontal long axis; STIR – short TI inversion recovery; LGE – late gadolinium enhancement; PCRMI – possible COVID-19 related myocardial injury

Table 2. CMR and laboratory findings

CMR findings	Control group n=31	Study group n=75	p
LVEF, %	57 (54-60)	59 (58-60)	0.13
LVEF below 50%, No.(%)	2(6)	4 (5.3)	0.43
LVEDV index, mL/m²	100 (96-106)	98 (89-106)	0.58
LVEDV index >110, No.(%)	1 (3.2)	7 (9.3)	0.65
LV mass index, g/m²	67 (62-74)	63 (56-67)	0.27
LV mass index > 70, No.(%)	3 (9.6)	9 (12)	0.28
PCRMI, No. (%)	4(13)	19 (25)	0.06
Myocardial LGE, No.(%)	4(13)	16 (21)	0.07
epicardial	1(3)	6 (8)	0.7
epicardial/midwall	2(7)	5 (6.7)	0.54
transmural	1(3)	5 (6.7)	0.54
Edema, No.(%)	0 (0)	1 (1.3)	0.7
Pericardial effusion, No.(%)	0 (0)	3 (4)	0.5
Pericardial LGE, No.(%)	0 (0)	0 (0)	0.8
Wall motion abnormalities, No. (%)	1 (3.2)	4 (5.3)	0.5
Definitive myocarditis, No.(%)	0 (0)	1 (1.3)	0.7
Possible myocarditis, No.(%)	0 (0)	3 (3.9)	0.5
Echocardiography parameters			
LVEF, Mean (SD)	59.0 (2.9)	59.2 (3.6)	0.8
LVEdD, Mean (SD)	51.1 (4.3)	51.8 (3.7)	0.4
IVSdD, mean (SD)	9.7 (1.0)	9.7 (1.1)	0.9
PWdD, mean (SD)	9.8 (1.1)	9.8 (1.1)	0.9
Blood test results			
CRP, mg/dL	0.6 (0.2-0.8)	0.7 (0.2-5.9)	0.8
hsTnI, pg/mL	3.2 (1.8-4.5)	4.1 (2.2-11.2)	0.6
NT-proBNP, pg/mL	22 (16-27)	24 (20-27)	0.6
CKMB, IU/l	16 (12-20)	19 (11-28)	0.7

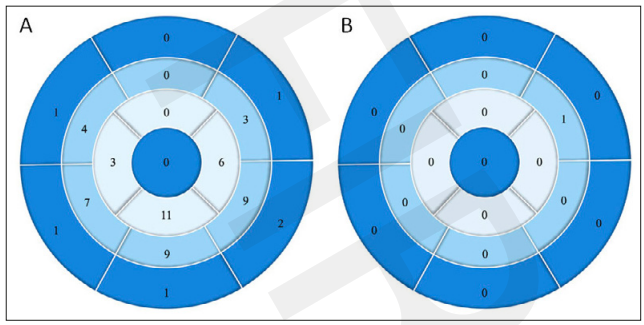

Figure 2. Distribution of segments with myocardial LGE (A) and edema (B) in the AHA 17 segments' model in 16 patients with PCRMI.
LGE – late gadolinium enhancement; AHA – American Heart Association; PCRMI – possible COVID-19 related myocardial injury

There were no statistical differences between the study group and control group and the study group with or without PCRMI, with regard to LV size and function (LVEF, LVEDV, LV mass) and laboratory findings (hsTnI, CKMB, CRP) (Table 3). PCRMI was demonstrated in 4 (13%) volunteers from the control group due to presence of LGE (n=4;13%), decreased LV ejection fraction (n=2; 6%) and wall motion abnormalities (n=1; 3.2%). Pericardial effusion/enhancement on LGE, myocardial edema were absent in the control group. None of the volunteers was diagnosed with definitive or possible myocarditis.

Table 3. CMR and laboratory findings in patients from the study group with (PCRMI+) and without (PCRMI-) possible COVID-19 related myocardial injury (PCRMI)

	PCRMI(+) n=19	PCRMI(-) n= 56	*p* value
LVEF, %	55 (52-59)	59 (57-62)	0.02
LVEF below 50%, No.(%)	4 (21)	0 (0)	0.08
LVEDV index, mL/m^2	97 (85-103)	99 (91-108)	0.8
LVEDV index >110, No.(%)	2 (11)	6 (11)	0.7
LV mass index, g/m^2	64 (57-67)	64 (57-67)	0.8
LV mass index > 70, No.(%)	0 (0)	9 (13)	0.4
CRP, mg/dL	0.4 (0.2-0,9)	0.3 (0.25-0.5)	0.4
hsTnI, pg/mL	1.2 (0.9-2.4)	1.5 (0.9-2.9)	0.1
NT-proBNP, pg/mL	29 (14-43)	20 (10-30)	0.02
CKMB, IU/l	13 (12-17)	15 (12-16)	0.6

DISCUSSION

To the best of our knowledge, this is a first prospective study including young, physically fit male patients without comorbidities, no or mild COVID-19 symptoms, who underwent assessment of myocardial injury at least 30 days following the negative results of a COVID-19 swab test. Previously published studies included either a smaller group of asymptomatic young patients undergoing earlier evaluation with no control group, older patients with comorbidities and COVID-19 severity ranging from mild to severe and no ongoing cardiac symptoms, or patients with ongoing cardiac symptoms [5, 7, 8, 9].

Interestingly, our study demonstrated a relatively common presence of PCRMI in healthy physically-fit young male patients despite relatively long-term follow-up after recovery from COVID-19 and lack of cardiac symptoms. Nevertheless, prevalence of PCRMI was lower than reported by Puntmann et al. in a cohort of older patients with comorbidities and more severe course of COVID-19 (49% patients with mild to moderate symptoms, 33% patients requiring hospitalization) with lower percentage of LGE (21% vs 32%) as well as pericardial effusion (4% vs 20%) [5]. As expected, PCRMI was also less common than reported by Huang et al. in a group of patients with ongoing cardiac symptoms (21% vs 58%) with lower percentage of myocardial edema (1% vs 54% on STIR images) and LGE (21% vs 31%) [7]. Our findings also show lower prevalence of myocarditis than Rajpal et al., who tested a small group of competitive athletes with no or mild COVID-19 symptoms earlier after recovery from COVID-19, and found myocarditis in 15% of patients according to updated Lake Louis criteria and LGE in 46% of patients [8].

Since our CMR study protocol did not include T1 and T2 mapping, complete direct comparison of myocardial edema/inflammation with recent studies is limited [5, 7, 8]. It is, however, worth mentioning that T2 measurements in the study of Puntmann et al. that revealed a high percentage of myocardial edema/inflammation in asymptomatic patients was limited to the septum in diastole where measurements can be influenced by partial volume artifact, especially in patients with thin myocardial walls and relatively long anterior septal perforator arteries [5, 10]. The above mentioned study also does not report the number of patients with definitive/possible myocarditis and LGE location, which might be crucial since LGE in the basal septum might reflect contrast enhancement of the anterior septal perforator arteries [11]. Therefore, it appears possible that the actual number of patients with myocardial injury might have been slightly lower.

All together these findings might indicate that in asymptomatic patients' presence of myocardial injury on CMR is determined by comorbidities, severity of COVID-19 symptoms, and length of the follow-up. This is further supported by prior myocarditis studies that showed that the area of LGE significantly decreases over time and completely resolves in some patients [12]. Despite the structural changes of myocardium (LGE, edema, pericardial effusion) on CMR, there was no significant increase in troponin level. However, elevated NT-proBNP might indicate myocardial damage that is no longer acute. The findings correspond with the findings of Eiros at al. study but not of Puntmann et al. (significant troponin increase in 15% of patients) [5, 13]. Similar to myocardial injury, length of observation and severity of COVID-19 seem to determine troponin release [14, 15]. Mildly elevated LV volume and mass in a few patients were not necessarily related to myocardial injury, and rather to competitive training.

The clinical significance of our findings is unknown, as it did not result in any significant sequelae such as arrhythmia or heart failure besides mild LV dysfunction. Whereas no definitive or possible myocarditis was found in the control group, LGE was relatively frequent suggesting that our findings might not be COVID-19 specific. This finding does not stand in agreement with Puntmann et al. who did not find LGE but only T1 and T2 elevation in some patients from a control group of age-matched and sex-matched of healthy volunteers. To the best of our knowledge, LGE presence was not assessed in control groups in other recently published studies on COVID-19.

LIMITATIONS

This was a single center study including a relatively large and homogenous group of patients. Lack of CMR obtained prior to COVID-19 was partially overcome by comparison with a control group of age-matched and sex-matched physically fit healthy volunteers. Control group was limited to 31 patients due to restrictions associated with COVID-19 surge.

Myocarditis was diagnosed based on original Lake Louis criteria [6]. Early gadolinium enhancement (EGE) was not assessed but recent data indicate that removing EGE from the original Lake Louis criteria does not significantly reduce diagnostic accuracy for myocarditis [16]. Since CMR study did not include T1 and T2 mapping, updated Lake Louis criteria could not be applied thus limiting the assessment of discrete lesions and direct complete comparison of myocardial edema/inflammation with recently published studies. None of the findings were confirmed by endomyocardial biopsy.

INTERPRETATION

PCRMI was a relatively common finding in asymptomatic young, physically fit male patients without comorbidities

relatively late after recovery from COVID-19. Presence of LGE in young healthy volunteers might suggest that these findings might not be COVID-19 specific. These findings warrant the need for further investigation of the long-term cardiovascular consequences of COVID-19 and comparison with other viral infections.

CONCLUSIONS

PCRMI was a frequent finding in young, asymptomatic, physically-fit patients sans comorbidities relatively late after recovery from COVID-19. Whereas no definitive or possible myocarditis was found in the control group, LGE was relatively frequent suggesting that our findings might not be COVID-19 specific. This warrants a need for further investigation into the long-term cardiovascular consequences of COVID-19.

LVEF, left ventricular ejection fraction; LVEDV, left ventricular end diastolic volume; LV, left ventricular; LGE, late gadolinium enhancement; LVEF – left ventricle ejection fraction; LVEdD – left ventricle end diastolic diameter, IVSdD – intraventricular septum diastolic diameter, PWdD – posterior wall diastolic diameter; CRP, C-reactive protein; hsTnI, high-sensitivity troponin I; NT-pro-BNP, N-terminal pro-b-type natriuretic peptide; CKMB, Creatine kinase -MB.

REFERENCES

1. Huang C, Wang Y, Li X, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020;395(10223):497–506. doi:10.1016/S0140-6736(20)30183-5
2. Long B, Brady WJ, Koyfman A, et al. Cardiovascular complications in COVID-19. Am J Emerg Med. 2020;38(7):1504–1507. doi:10.1016/j.ajem.2020.04.048
3. Shi S, Qin M, Cai Y, et al. Characteristics and clinical significance of myocardial injury in patients with severe coronavirus disease 2019. Eur Heart J. 2020;41(22):2070–2079. doi:10.1093/eurheartj/ehaa408
4. Pillai A, Lawson B. Coronavirus disease 2019 and cardiovascular diseases: collateral damage? Curr Opin Anaesthesiol. 2022;35(1):5–11. doi:10.1097/ACO.0000000000001076
5. 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. Erratum in: JAMA Cardiol. 2020 Nov 1;5(11):1308.
6. Friedrich MG, Sechtem U, Schulz-Menger J, et al. International Consensus Group on Cardiovascular Magnetic Resonance in Myocarditis. Cardiovascular magnetic resonance in myocarditis: A JACC White Paper. J Am Coll Cardiol. 2009;53(17):1475–87. doi:10.1016/j.jacc.2009.02.007
7. Huang L, Zhao P, Tang D, et al. Cardiac Involvement in Patients Recovered From COVID-2019 Identified Using Magnetic Resonance Imaging. JACC Cardiovasc Imaging. 2020;13(11):2330–2339. doi:10.1016/j.jcmg.2020.05.004
8. Rajpal S, Tong MS, Borchers J, et al. Cardiovascular Magnetic Resonance Findings in Competitive Athletes Recovering From COVID-19 Infection. JAMA Cardiol. 2021 Jan 1;6(1):116–118. doi:10.1001/jamacardio.2020.4916. Erratum in: JAMA Cardiol. 2021;6(1):123.
9. Tanacli R, Doeblin P, Götze C, et al. COVID-19 vs. Classical Myocarditis Associated Myocardial Injury Evaluated by Cardiac Magnetic Resonance and Endomyocardial Biopsy. Front Cardiovasc Med. 2021;8:737257. doi:10.3389/fcvm.2021.737257
10. Wiesmueller M, Wuest W, Heiss R, et al. Cardiac T2 mapping: robustness and homogeneity of standardized in-line analysis. J Cardiovasc Magn Reson. 2020;22(1):39. doi:10.1186/s12968-020-00619-x
11. Nakamura M, Kido T, Hirai K, et al. What is the mid-wall linear high intensity "lesion" on cardiovascular magnetic resonance late gadolinium enhancement? J Cardiovasc Magn Reson. 2020;22(1):66. doi:10.1186/s12968-020-00665-5
12. Mahrholdt H, Goedecke C, Wagner A, et al. Cardiovascular magnetic resonance assessment of human myocarditis: a comparison to histology and molecular pathology. Circulation. 2004;109(10):1250–8. doi:10.1161/01.CIR.0000118493.13323.81
13. Eiros R, Barreiro-Pérez M, Martín-García A, et al. Afección pericárdica y miocárdica tras infección por SARS-CoV-2: estudio descriptivo transversal en trabajadores sanitarios [Pericardial and myocardial involvement after SARS-CoV-2 infection: a cross-sectional descriptive study in healthcare workers]. Rev Esp Cardiol. 2022;75(9):735–747. doi:10.1016/j.recesp.2021.10.021
14. Inciardi RM, Lupi L, Zaccone G, et al. Cardiac Involvement in a Patient With Coronavirus Disease 2019 (COVID-19). JAMA Cardiol. 2020;5(7):819–824. doi:10.1001/jamacardio.2020.1096
15. Li H, Liu L, Zhang D, et al. SARS-CoV-2 and viral sepsis: observations and hypotheses. Lancet. 2020;395(10235):1517–1520. doi: 10.1016/S0140-6736(20)30920-X
16. Ferreira VM, Schulz-Menger J, Holmvang G, et al. Cardiovascular Magnetic Resonance in Nonischemic Myocardial Inflammation: Expert Recommendations. J Am Coll Cardiol. 2018;72(24):3158–3176. doi:10.1016/j.jacc.2018.09.072