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EDITED BY
Jiu-Chang Zhong,
Capital Medical University, China

REVIEWED BY
Emir Yonas,
YARSI University, Indonesia
Giulia Iannaccone,
Catholic University of the Sacred Heart, Italy

*CORRESPONDENCE
Roman Panovský
✉ panovsky@fnusa.cz

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Lower ventricular and atrial strain in patients who recovered from COVID-19 assessed by cardiovascular magnetic resonance feature tracking

Mary Luz Mojica-Pisciotti[1,2], Roman Panovský[1,2,3]*, Tomáš Holeček[1,4,5], Lukáš Opatřil[1,2,3] and Věra Feitová[1,4]

[1]International Clinical Research Center, St. Anne's University Hospital, Brno, Czech Republic, [2]1st Department of Internal Medicine/Cardioangiology, St. Anne's University Hospital, Brno, Czech Republic, [3]Faculty of Medicine, Masaryk University, Brno, Czech Republic, [4]Department of Medical Imaging, St. Anne's University Hospital, Brno, Czech Republic, [5]Department of Biomedical Engineering, Brno University of Technology, Brno, Czech Republic

Introduction: One of the most common complications of coronavirus disease 2019 (COVID-19) is myocardial injury, and although its cause is unclear, it can alter the heart's contractility. This study aimed to characterize the ventricular and atrial strain in patients who recovered from COVID-19 using cardiovascular magnetic resonance feature-tracking (CMR-FT).
Methods: In this single-center study, we assessed left ventricle (LV) and right ventricular (RV) global circumferential strain (GCS), global longitudinal strain (GLS), global radial strain (GRS), left atrial (LA) and right atrial (RA) longitudinal strain (LS) parameters by CMR-FT. The student's t-test and Wilcoxon rank-sum test were used to compare the variables.
Results: We compared seventy-two patients who recovered from COVID-19 (49 ± 16 years) to fifty-four controls (49 ± 12 years, $p = 0.752$). The patients received a CMR examination 48 (34 to 165) days after the COVID-19 diagnosis. 28% had LGE. Both groups had normal LV systolic function. Strain parameters were significantly lower in the COVID-19 survivors than in controls.
Discussion: Patients who recovered from COVID-19 exhibited significantly lower strain in the left ventricle (through LVGCS, LVGLS, LVGRS), right ventricle (through RVGLS and RVGRS), left atrium (through LALS), and right atrium (through RALS) than controls.

KEYWORDS

cardiovascular magnetic resonance, feature tracking, heart deformation, strain analysis, COVID-19

1. Introduction

Coronavirus disease 2019 (COVID-19) is an acute and highly contagious disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Although COVID-19 is primarily respiratory, it affects most organs (1) and leads to several cardiac problems (2, 3), including heart failure (4). One of the most common complications is myocardial injury, associated with a worsened prognosis and occurs in 20% to 30% (5, 6).

The cause of cardiac injury is not entirely clear. Current evidence suggests it may be due to excessive inflammatory responses, specifically cytokine release syndrome or "cytokine storm" (7). Other potential mechanisms could include viral myocarditis or pericarditis,

stress-induced cardiomyopathy, and microvascular thrombosis (8–11). Regardless of its cause, cardiac injury can alter the heart's contractility, and studying its impact might offer insight into improving post-COVID-19 care.

Cardiovascular magnetic resonance (CMR) has become crucial in diagnosing cardiovascular pathologies and characterizing myocardial tissue and damage (12). In particular, CMR feature tracking (CMR-FT) allows the assessment of the regional deformation of the heart by evaluating the myocardial strain (13, 14). CMR-FT can help to detect subtle systolic or diastolic dysfunction (15), making it an effective tool for assessing cardiovascular health, especially for COVID-19 survivors.

This study aims to characterize the myocardial strain in patients who recovered from COVID-19 with cardiovascular magnetic resonance feature tracking.

2. Materials and methods

2.1. Study population

A total of 126 subjects, divided into two groups labeled as patients who recovered from COVID-19 (CoV) and a control group (CG), were included in this study. The CoV group comprised 72 patients who had recovered from COVID-19 and underwent a CMR examination after recovery between November 2020 and October 2022. No specific COVID-19 variant was studied. The CG comprised 54 patients of similar sex and age to those in the CoV group, with no history, symptoms, or signs of any pre-existing cardiac disease and negative findings. They were referred to CMR examination for atypical thoracic pain or suspected hypertrophy in those with insufficient echocardiography images. All participants were over 18 years old, had no pre-existence of cardiac disease, chronic inflammatory or autoimmune disease, and no contraindications for CMR examination.

The CoV patients were further divided into two subgroups, group A and group B. Group A consisted of 47 subjects who had recovered from COVID-19 and had a clinical indication for CMR examination. These patients were examined between November 2020 and October 2022 and retrospectively included. Their clinical indication for the CMR examination was not specific to any severity level of COVID-19. Group B consisted of 25 patients prospectively recruited and examined approximately one month after being released from the hospital for being treated for moderate to critical COVID-19 as defined by The National Institute of Health Coronavirus Disease 2019 treatment guidelines (16). These patients exhibited at least one of the following criteria: desaturation with $SpO2 \leq 94\%$, respiratory frequency ≥ 30 per minute, lung infiltrates according to x-rays, signs of respiratory failure, and septic shock.

The study is part of a grant project approved by the Ethics Committee of St Anne's University Hospital Brno (Reference Number 6G/2022). It was conducted following the Declaration of Helsinki (2000) of the World Medical Association. All prospectively included participants in the project signed informed consent.

2.2. CMR data acquisition

All CMR studies were performed on a 1.5T scanner (Ingenia, Philips Medical Systems) following our standard protocol (17). Cine images were obtained with balanced turbo field echo steady-state free precession (SSFP) sequences (typical parameters: FOV 300×300 mm, acquisition voxel size $1.67 \times 1.67 \times 8.00$ mm, reconstruction matrix 256, slice thickness 8 mm, SENSE factor 1.7, 30 to 50 frames per cardiac cycle) in long-axis (two-chamber, four-chamber, three-chamber) and short-axis views. Those were used for functional and strain assessment. Late gadolinium enhancement (LGE) images were acquired approximately 10 min after a contrast bolus injection [0.2 mmol/kg, gadobutrol (Gadovist, Bayer)].

2.3. Clinical assessment

An expert radiologist (VF) assessed the left ventricle (LV) and right ventricle (RV) function with the IntelliSpace Portal (ISP) workspace (version 11, Philips Healthcare) according to the established clinical protocols (12). The reported variables were the left ventricle ejection fraction (LVEF), LV end-diastole volume (LVEDV), LV end-systole volume (LVESV), LV stroke volume (LVSV), LV cardiac output (LVCO), LV mass (LVM), right ventricle ejection fraction (RVEF), RV end-diastole volume (RVEDV), RV end-systole volume (RVESV), and RV stroke volume (RVSV). All LV and RV volumes were indexed to the body surface area (BSA), which we indicated by adding the letter I at the end of the abbreviations. Two clinical experts (VF and RP) evaluated LGE and pericardial effusion.

2.4. CMR-FT strain assessment

Two experienced readers (MLMP and TH) assessed the LV deformation by 2D strain analysis using the commercial software cvi42 (release 5.13.9, Circle Cardiovascular Imaging). Each reader contoured the endocardial and epicardial LV and RV walls in both the end-diastole (ED) and end-systole (ES) frames in long-axis (two-chamber, four-chamber, three-chamber) and short-axis cine images. They excluded the papillary muscles, epicardial fat, and trabeculae, visually verified each contour, and adjusted if necessary. Only images from the short-axis stack free from the left ventricular outflow tract were considered in the analysis. The software automatically propagated the contours and determined the global longitudinal strain (GLS), global circumferential strain (GCS), and global radial strain from both SAX (GRS_{SAX}) and LAX images (GRS_{LAX}). GRS was the average of GRS_{SAX} and GRS_{LAX}.

Likewise, the readers analyzed the left atrium (LA) and right atrium (RA) with the same software. They traced the LA and RA

contours in the ED and ES frames in two- and four-chamber long-axis images. The software determined the atrial strain by averaging the measurements from these contours. The results included the following LA and RA parameters: minimum volume (LAVmin, RAVmin), maximum volume (LAVmax, RAVmax), ejection fraction (LAEF, RAEF), and longitudinal strain (LALS, RALS). LA and RA volumes were indexed to the BSA.

2.5. Statistical analysis

Descriptive statistics are reported as the mean (standard deviation, SD) or median (interquartile range, IQR) for normally and non-normally distributed continuous variables and as numbers (percentages) for categorical ones. The normality of the data was checked by the Shapiro-Wilk test and visual inspection of the histograms. Proportions of categorical variables were analyzed using the Chi-square test of independence. The student's t-test and Wilcoxon rank-sum test were used to compare normally and non-normally distributed variables. The adjusted *P*-value was obtained using a false discovery rate correction to account for multiple comparisons. A P-value < 0.05 was considered statistically significant. The intraobserver and interobserver agreement was assessed with the intraclass correlation coefficient (ICC). The ICC (two-way mixed-effects model) was determined from twenty randomly selected cases analyzed by two readers (MLMP, TH), one of whom repeated them one month apart. The repeatability was classified as poor (<0.5), fair (0.50 to 0.75), good (0.75 to 0.90), and excellent (0.90 to 1) (18). All statistical analyses were performed with R-4.2.2 and RStudio IDE (2022.12.0 + 353, RStudio, PBC).

3. Results

3.1. Study group

The study flowchart is shown in **Figure 1**. General characteristics were similar in both groups (see **Table 1**). The median time between the COVID-19 diagnosis and the CMR examination was 48 (34 to 165) days. Although both groups had normal LVEF, significantly lower LVEF, RVEF, and LAEF were found in patients who recovered from COVID-19 compared to CG. On the contrary, significantly higher LAVImin, RAVImin, and LVESVI were found in the CoV group than in CG. Additionally, 28% of CoV ($n = 20$) had LGE, and 3% ($n = 2$) had pericardial effusion (≥10 mm). The LGE patterns were non-ischemic, in most cases, subepicardial ($n = 12$), mid-myocardial ($n = 4$), transmural ($n = 2$), and subendocardial ($n = 2$) mainly found in the basal segment of anterolateral or inferolateral walls.

In the patients treated for moderate to critical COVID-19 (Group B), we found anemia, renal insufficiency, and appetite

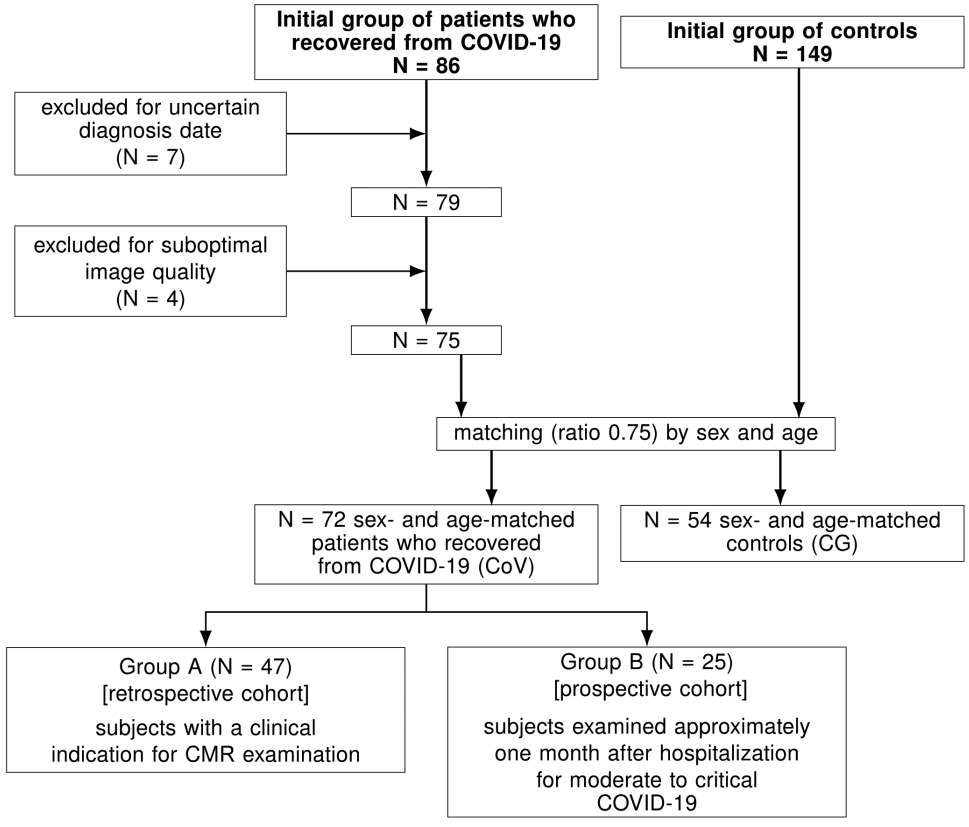

FIGURE 1
Study flowchart. CMR, cardiovascular magnetic resonance.

TABLE 1 Demographics and clinical parameters in patients who recovered from COVID-19 (CoV) and a control group (CG).

Variable	CG, $N = 54$	CoV, $N = 72$	*P*-value
Age (y)	49 (12)	49 (16)	0.752
Sex (female/male)	26 / 28	33 / 39	0.938
BMI (kg/m^2)	26.7 (23.6, 28.9)	27.3 (24.2, 30.5)	0.171
BSA (m^2)	1.98 (0.22)	1.98 (0.20)	0.998
HR (bpm)	64 (57, 69)	72 (63, 78)	**0.005**
LGE+ (n, %)		20 (28%)	
Pericardial effusion (≥10 mm) (*n*, %)		2 (3%)	
Left ventricle			
LVEF (%)	66 (63, 73)	63 (58, 70)	**<0.001**
LVEDVI (ml/m^2)	60.4 (55.3, 66.0)	64.8 (55.4, 74.2)	0.080
LVESVI (ml/m^2)	19.7 (14.9, 24.2)	23.3 (15.8, 31.5)	**0.002**
LVSVI (ml/m^2)	41.5 (6.5)	39.5 (8.5)	0.139
CI (l/min/m^2)	2,618.4 (2,318.9, 2,877.9)	2,878.7 (2,376.8, 3,170.8)	0.423
LVMI (g/m^2)	44.7 (38.0, 54.0)	49.3 (41.2, 60.5)	0.135
Right ventricle			
RVEF (%)	64 (8)	60 (10)	**0.034**
RVEDVI (ml/m^2)	66.2 (13.7)	67.0 (17.2)	0.751
RVESVI (ml/m^2)	22.8 (18.0, 30.1)	26.5 (17.6, 36.9)	0.123
RVSVI (ml/m^2)	41.5 (7.1)	39.3 (8.1)	0.120
Left atrium			
LAVImin (ml/m^2)	12.2 (9.6, 16.0)	12.6 (10.8, 19.2)	**0.008**
LAVImax (ml/m^2)	30.9 (25.0, 36.4)	31.7 (24.3, 38.2)	0.469
LAEF (%)	62 (56, 66)	57 (48, 63)	**0.001**
Right atrium			
RAVImin (ml/m^2)	18.1 (13.4, 23.5)	16.4 (11.1, 22.5)	0.700
RAVImax (ml/m^2)	39.9 (11.0)	35.2 (12.3)	**0.025**
RAEF (%)	54 (46, 58)	53 (42, 60)	0.076

Variables are expressed as numbers/total (percentages), mean (standard deviation), or median (interquartile range) for categorical, normally distributed, and non-normally distributed continuous variables.
BMI, body mass index; BSA, body surface area; CG, control group; CI, cardiac index; CoV, patients who recovered from COVID-19; EF, ejection fraction; EDV, end-diastole volume; ESV, end-systole volume; HR, heart rate; I, indexed; LA, left atrium; LGE, late gadolinium enhancement; LV, left ventricle; LVCO, left ventricular cardiac output; LVM, left ventricular mass; max, maximum; min, minimum; n, number of subjects; N, total number of subjects; RA, right atrium; RV, right ventricle; SV, stroke volume.
p-values <0.05 are presented in bold.

loss in 4% (1/25), diabetes mellitus in 8% (2/25), hypertension in 20% (5/25), and dyslipidemia in 12% (3/25). Also, cardiac biomarkers during hospitalization for this group were measured: NTproBNP 124 (55, 341) ng/L (20 patients), troponin 7 (5, 10) ng/L (17 patients), highest CPR 120 (57, 185) mg/L, highest leucocytes 9.5 (6.6, 11.2) cells/μl, glomerular filtration 1.17 ± 0.47 ml/s/1.73 m^2, and creatinin 87 (79, 120) μmol/L.

3.2. Strain parameters

Ventricular and atrial strain parameters were significantly lower in the CoV group than in CG, except for the RVGCS and $RVGRS_{SAX}$ (see Table 2). A comparison of the strain assessment is shown in Figure 2.

3.3. Comparison between two groups of patients who recovered from COVID-19

General parameters were similar between the two subgroups of patients who recovered from COVID-19 (see Table 3). We compared the groups considering three age groups (<50, 50 to 70, and >70 years) and found no significant differences in the cardiac strain (see Supplementary Table S1).

3.4. Reproducibility

The intraobserver and interobserver reproducibility was good or excellent for most strain parameters and fair for the interobserver $RVGRS_{SAX}$ (see Table 4).

4. Discussion

We assessed CMR-derived ventricular and atrial strain parameters in patients who recovered from COVID-19 and found significantly lower cardiac strain values compared to a control group. As far as we know, this study is the first to report lower atrial longitudinal strain values in this population.

Some studies reported lower values of GLS and GCS in patients who recovered from COVID-19 than in healthy controls (19–23). However, this difference was only significant in a handful

TABLE 2 Left ventricular, right ventricular, left atrial and right atrial strain in patients who recovered from COVID-19 (CoV) and a control group (CG).

Variable	CG, $N = 54$	CoV, $N = 72$	*P*-value
Left ventricle			
LVGCS (%)	−17.9 (−19.6, −16.9)	−17.0 (−18.5, −14.9)	**<0.001**
LVGLS (%)	−17.8 (−19.3, −16.8)	−17.4 (−18.6, −15.4)	**0.002**
$LVGRS_{LAX}$ (%)	32.1 (29.7, 36.1)	29.4 (25.3, 33.1)	**<0.001**
$LVGRS_{SAX}$ (%)	29.0 (26.9, 33.6)	27.1 (22.5, 30.8)	**<0.001**
LVGRS (%)	31.2 (28.0, 33.9)	28.5 (24.1, 31.8)	**<0.001**
Right ventricle			
RVGCS (%)	−14.8 (4.2)	−13.7 (4.5)	0.157
RVGLS (%)	−25.9 (−28.3, −23.0)	−24.4 (−26.5, −22.0)	**0.048**
$RVGRS_{LAX}$ (%)	59.5 (47.3, 74.2)	53.5 (46.8, 61.2)	**0.027**
$RVGRS_{SAX}$ (%)	23.7 (19.0, 32.6)	21.9 (17.0, 26.3)	0.124
RVGRS (%)	42.6 (35.8, 48.60)	38.3 (32.7, 44.2)	**0.017**
Left atrium			
LALS (%)	36.6 (29.5, 47.9)	28.8 (19.5, 41.0)	**0.003**
Right atrium			
RALS (%)	43.9 (14.4)	37.9 (17.8)	**0.037**

Variables are expressed as mean (standard deviation) or median (interquartile range) for normally distributed and non-normally distributed continuous variables.
CG, control group; CoV, patients who recovered from COVID-19; GCS, global circumferential strain; GLS, global longitudinal strain; GRS, global radial strain; LA, left atrium; LAX, long axis; LS, longitudinal strain; LV, left ventricle; RA, right atrium; RV, right ventricle; SAX, short axis.
p-values <0.05 are presented in bold.

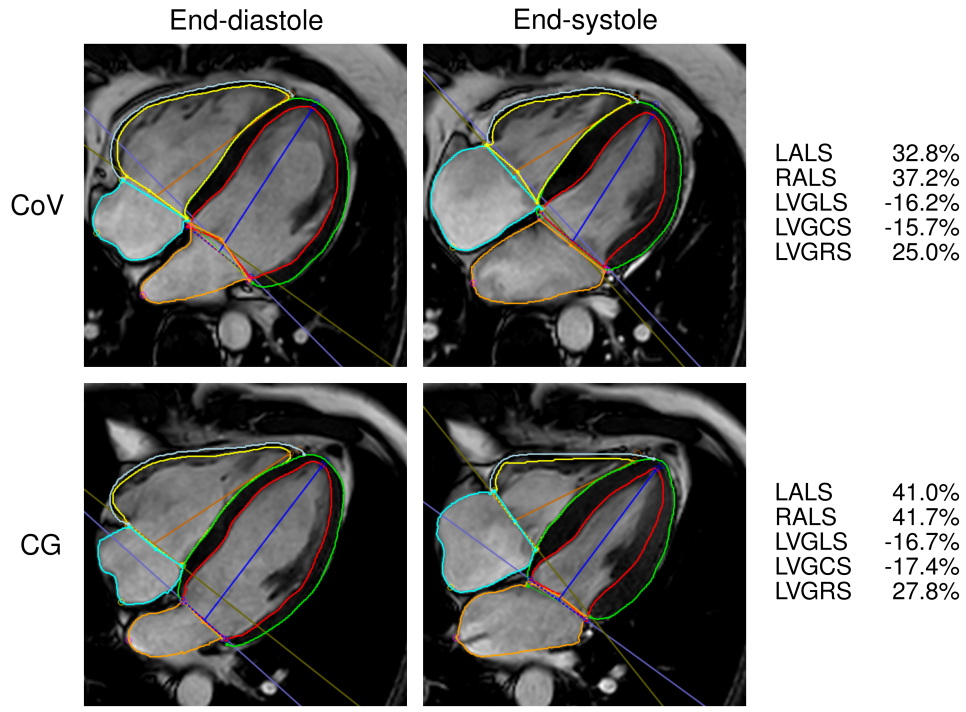

FIGURE 2
Contouring for the ventricular and atrial strain calculation in one patient who recovered from COVID-19 (CoV) and one control (CG). GCS, global circumferential strain; GLS, global longitudinal strain; GRS, global radial strain; LA, left atrium; LS, longitudinal strain; LV, left ventricle; RA, right atrium; RV, right ventricle.

(21–23). Both parameters were lower in patients who recovered from the delta variant of COVID-19 (23). Lower GLS was found in patients who recovered from severe or moderate cases (21) and those with LGE (22). No significant alterations in GRS values have been reported so far in these patients (21–24). In our study, we included patients regardless of their COVID-19 variant.

The improvement of cardiac function in patients who recovered from COVID-19 seems time-dependent. A study

TABLE 3 Demographics and clinical parameters in patients who recovered from COVID-19: group A (with a clinical CMR indication after recovery) and group B (treated for moderate to critical COVID-19).

Variable	Overall, *N* = 72	Group A, *N* = 47	Group B, *N* = 25	*P*-value
Age (y)	49 (16)	47 (16)	55 (15)	**0.039**
Sex (female/male)	33 / 39	18 / 29	15 / 10	0.131
BMI (kg/m^2)	27.3 (24.2, 30.5)	27.1 (24.1, 30.4)	29.0 (24.8, 30.6)	0.567
BSA (m^2)	1.98 (0.20)	1.99 (0.22)	1.96 (0.17)	0.538
HR (bpm)	72 (14)	72 (14)	70 (12)	0.526
LGE+ (*n*, %)	20 (28%)	17 (36%)	3 (12%)	**0.029**
Time between diagnosis and CMR (days)	48 (34, 165)	76 (40, 222)	37 (34, 46)	**<0.001**
Left ventricle				
LVEF (%)	63 (58, 70)	62 (55, 68)	66 (61, 70)	**0.011**
LVEDVI (ml/m^2)	65.8 (17.1)	69.4 (16.9)	59.1 (15.8)	**0.012**
LVESVI (ml/m^2)	23.3 (15.8, 31.5)	26.0 (18.8, 32.6)	18.7 (13.7, 25.7)	**0.004**
LVSVI (ml/m^2)	39.5 (8.5)	39.9 (8.1)	38.7 (9.3)	0.605
CI (l/min/m^2)	2,798.2 (552.2)	2,867.4 (565.5)	2,668.0 (512.1)	0.135
LVMI (g/m^2)	51.3 (14.3)	52.6 (15.2)	48.9 (12.1)	0.267
Right ventricle				
RVEF (%)	60 (10)	59 (10)	63 (9)	0.093
RVEDVI (ml/m^2)	67.0 (17.2)	69.6 (17.0)	62.3 (16.9)	0.089
RVESVI (ml/m^2)	26.5 (17.6, 36.9)	28.2 (17.7, 37.8)	23.8 (15.7, 31.6)	0.055
RVSVI (ml/m^2)	39.3 (8.1)	39.9 (7.6)	38.2 (9.0)	0.423
Left atrium				
LAVImin (ml/m^2)	12.6 (10.8, 19.2)	12.6 (10.9, 20.0)	12.3 (7.8, 17.5)	**0.048**
LAVImax (ml/m^2)	31.7 (24.3, 38.2)	31.2 (25.5, 38.3)	32.2 (20.3, 37.9)	0.270
LAEF (%)	57 (48, 63)	56 (42, 61)	59 (53, 63)	**0.017**
Right atrium				
RAVImin (ml/m^2)	16.4 (11.1, 22.5)	17.8 (11.5, 23.3)	13.4 (10.4, 20.3)	0.101
RAVImax (ml/m^2)	35.2 (12.3)	35.8 (12.8)	34.0 (11.4)	0.542
RAEF (%)	53 (42, 60)	50 (36, 60)	59 (49, 63)	**0.006**

Variables are expressed as numbers/total (percentages), mean (standard deviation), or median (interquartile range) for categorical, normally distributed, and non-normally distributed continuous variables.
BMI, body mass index; BSA, body surface area; CI, cardiac index; EF, ejection fraction; EDV, end-diastole volume; ESV, end-systole volume; HR, heart rate; I, indexed; LA, left atrium; LGE, late gadolinium enhancement; LV, left ventricle; LVCO, left ventricular cardiac output; LVM, left ventricular mass; max, maximum; min, minimum; n, number of subjects; N, total number of subjects; RA, right atrium; RV, right ventricle; SV, stroke volume.
p-values <0.05 are presented in bold.

TABLE 4 Intraobserver and interobserver reproducibility (ICC, two-way mixed-effects model) of left ventricular, right ventricular, left atrial, and right atrial strain.

Variable	Intraobserver	Interobserver
LVGCS (%)	0.995 (0.986–0.998)	0.998 (0.994–0.999)
LVGLS (%)	0.996 (0.989–0.998)	0.996 (0.986–0.999)
$LVGRS_{LAX}$ (%)	0.995 (0.989–0.998)	0.994 (0.980–0.998)
$LVGRS_{SAX}$ (%)	0.993 (0.982–0.997)	0.998 (0.994–0.999)
RVGCS (%)	0.837 (0.634–0.932)	0.808 (0.461–0.941)
RVGLS (%)	0.959 (0.900–0.984)	0.940 (0.807–0.982)
$RVGRS_{LAX}$ (%)	0.948 (0.874–0.979)	0.925 (0.762–0.978)
$RVGRS_{SAX}$ (%)	0.825 (0.609–0.927)	0.675 (0.195–0.894)
LALS (%)	0.972 (0.930–0.989)	0.960 (0.867–0.988)
RALS (%)	0.831 (0.622–0.930)	0.973 (0.909–0.992)

Values are expressed as ICC (95% CI).
CI, confidence interval; GCS, global circumferential strain; GLS, global longitudinal strain; GRS, global radial strain; ICC, intraclass correlation coefficient; LA, left atrium; LAX, long axis; LS, longitudinal strain; LV, left ventricle; RA, right atrium; RV, right ventricle; SAX, short axis.

reported that 30 days after the initial COVID-19 diagnosis, the GLS could detect subclinical LV dysfunction in patients with low cardiac risk (25). Another one, in patients during the acute phase of COVID-19 infection, showed abnormal myocardial mechanics within 3 to 8 days after the diagnosis, revealing significantly lower peak GLS than controls and a low rate of positive LGE (4%) (26).

In our study, the group of patients examined around 30 days after recovering from COVID-19 had similar cardiac strain to those examined later. In this group, we did not study the severity of the COVID-19 symptoms during the active phase of the disease or its connection to cardiac outcomes. It has been shown that mild cases of COVID-19 experience fewer complications and take less time to recover than more severe cases (27–29). There is also compelling evidence of cardiovascular sequelae among survivors beyond the first month of the illness (30).

So far, only one published study reported similar CMR-derived LA and RA strains between patients with persistent symptoms (such as arrhythmia, fatigue, weakness, and lack of taste or smell) examined three months after a positive test for COVID-19 infection and healthy volunteers (20). Contrary to our study, our patients had no similar persistent disease symptoms and were examined earlier than three months after the diagnosis. We also identified lower global LV strains in our

cohort, which may account for these two studies' differences. The severity of the COVID-19 symptoms in the participants of both studies was not compared, which could also influence cardiac function recovery (21).

We found LGE in 28% of the patients who recovered from COVID-19, which agrees with other studies (22, 24, 31, 32). However, we did not observe as many mid-myocardial or patchy patterns as other authors (22), but instead, mainly subepicardial involvement.

Cardiac strain is assessed through Speckle-Tracking echocardiography (STE) or CMR-FT (14, 33). Although STE is more available and portable than CMR, it is frequently affected by motion artifacts and has a lower spatial resolution than CMR-FT (14). Nonetheless, there is evidence of a good agreement between the assessment of GLS and GCS using STE and CMR-FT (14). On the other hand, there has been a growing interest in assessing atrial function using STE. Although such a possibility dramatically depends on high-quality images and is challenging because of the narrow walls of the atria, some studies support its feasibility and comparability with CMR-FT (34–36).

Regarding patients who recovered from COVID-19, echocardiography-based studies reported lower GLS in those examined within two months of the diagnosis (25, 37) and a slight improvement after three months of the hospital discharge (38). The GLS reaches similar levels to those in controls around six months after the diagnosis (39–41). One study reported similar LA and RA peak systolic strain values between COVID-19 survivors and healthy controls (39).

5. Limitations

This single-center study has some limitations. Firstly, our study population was relatively small, primarily due to the unprecedented demand for healthcare during the first waves of the COVID-19 pandemic. Such conditions translated into prioritizing other imaging modalities over CMR to treat affected individuals. The hospital strain posed a lower availability of CMR equipment. Secondly, most sufferers in our cohort did not exhibit severe enough symptoms, had previous pathologies, or had low compliance to participate in research. Therefore, our results may not apply to the complete range of patients who recovered from COVID-19. Thirdly, we lack the clinical biomarkers assessment for patients with a clinical CMR indication after recovery (Group A), as they were retrospectively recruited. Also, we could not ascertain the specific COVID-19 variant for each patient. Fourthly, we did not study the severity of the COVID-19 symptoms during the active phase of the disease, its connection to cardiac outcomes, or the influence of other cardiovascular conditions, such as heart failure (4). Also, we had no baseline CMR examination for the participants, meaning we cannot directly link the decreased strain values to the COVID-19 infection. Finally, we did not perform phasic atrial strain assessment as the cvi42 software is not optimized for this task.

6. Conclusions

We conclude that patients who recovered from COVID-19 exhibited significantly lower strain in the left ventricle (through GCS, GLS, GRS), right ventricle (through RVGLS and RVGRS), left atrium (through LALS), and right atrium (through RALS) than controls.

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Ethics statement

The studies involving humans were approved by Ethics Committee of St Anne's University Hospital Brno. The studies were conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study.

Author contributions

MM-P: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Software, Validation, Visualization, Project administration, Supervision, Writing – original draft, Writing – review & editing. RP: Conceptualization, Funding acquisition, Investigation, Methodology, Project administration, Resources, Supervision, Validation, Writing – review & editing. TH: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Validation, Writing – review & editing. LO: Conceptualization, Resources, Validation, Writing – review & editing. VF: Conceptualization, Investigation, Project administration, Supervision, Writing – review & editing.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Supplementary material

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fcvm.2023.1293105/full#supplementary-material

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