ORIGINAL ARTICLE

Cardiac Magnetic Resonance Imaging Study on Cardiovascular Involvement >60 Days Post COVID-19 Recovery

Nihar Mehta[1*], Tanushree Somir Banerjee[2], Chandresh O Karnavat[3], Rajesh M Parikh[4], Ritu Kashikar[5], Shraddha Sinhasan[6], Shruti Tandan-Pardasani[7], Susheel Bindroo[8], Anand Bhabhor[9], Indraneil Raut[10], Nimish Shah[11], Shrinivas B Desai[12], Munira Kapadia[13], Maherra Desai[14], Aarshee Kapoor[15]

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Abstract

Context: This study evaluates individuals who have recovered from coronavirus disease 2019 (COVID-19) for cardiac manifestations at least 60 days postrecovery, using cardiac magnetic resonance (CMR) imaging. Native T1, native T2, and late gadolinium enhancement (LGE) were used as parameters to detect potential cardiac pathologies.

Aim: To study the incidence and nature of cardiac manifestations in long COVID using CMR.

Study design: Prospective observational study.

Methods: A total of 54 COVID-19-recovered patients and 50 healthy matched controls (age- and sex-based) underwent CMR using a 3 T scanner. Pathologies were assessed through native T1 and T2 mapping and LGE imaging. Data analysis was conducted using SPSS version 23.

Results: Only 1 patient (1.85%) showed midmyocardial LGE in the basal and midinferior segments, along with raised native T1 and T2 values. The remaining 53 recovered patients (98.15%) revealed no LGE or elevated T1/T2 values compared to controls. Native T1 and T2 values did not significantly differ between the post-COVID participants and healthy controls. Mild concentric hypertrophy was observed in six patients, which appeared incidental and unrelated to COVID-19. None of the patients exhibited elevated troponin T levels.

Conclusion: In this study, CMR imaging did not find a significant increase in cardiac manifestations that may be attributed to long COVID in patients who had recovered from COVID-19 compared with uninfected controls. We believe the incidence of cardiovascular comorbidities related to long COVID syndrome is much lower in the Indian population than reported in Western literature. These findings are reassuring and may help alleviate concerns among the Indian population regarding long-term cardiovascular involvement of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection.

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Introduction

Since the onset of the coronavirus disease 2019 (COVID-19) pandemic, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection has infected millions of people all over the world, leading to significant morbidity and mortality. It is well established that COVID-19 is a multisystem disease, having pulmonary and extrapulmonary manifestations and adverse effects on the cardiovascular system.[1] Several studies have shown that COVID-19 significantly impacts cardiac function during the acute phase, with approximately 20–30% of patients affected.[2,3] Multiple mechanisms have been proposed, including direct myocardial infection by SARS-CoV-2 *via* angiotensin-converting enzyme 2 (ACE2) receptors, hemodynamic stress secondary to respiratory failure, systemic cytokine activation, and stress-induced cardiomyopathy.[4–7]

Evidence regarding post-COVID-19 cardiac manifestations has been conflicting, with the prevalence of COVID-19-related cardiac pathologies varying between 1.4 and 58–78%.[8–10] Another aspect is the occurrence of "long COVID" syndrome. According to the Department of Health and Human Services (HHS), in association with the Centers for Disease Control and Prevention (CDC), "long COVID" refers to symptoms, signs, and conditions that persist for 4 weeks or more after SARS-CoV-2 infection. These symptoms may be multisystemic and can have a relapsing-remitting presentation. Some commonly reported cardiac problems of long COVID include chest pain, electrocardiogram (ECG) changes, postural tachycardia, new-onset arrhythmias, and conditions such as postinfectious perimyocarditis with subsequent ventricular failure, arterial wall inflammation, or microthrombosis.[11]

Cardiac magnetic resonance (CMR) is the benchmark for noninvasive cardiovascular imaging and is essential in characterizing myocardial tissue, quantifying volumes, and detailing the structural and dynamic functions of the heart.[9] Myocardial mapping, which includes T1 and T2 mapping, is valuable in visualizing and quantifying focal or diffuse pathologies of the myocardium.[10] T1 values can help identify fibrosis, lipid accumulation, amyloid deposits, and iron overload,[12] while T2 values assist in detecting myocarditis, edema due to infarction, and other inflammatory pathologies, as well as intramyocardial hemorrhage.[13,14] Late gadolinium enhancement (LGE) helps differentiate viable from nonviable myocardium and is used to detect myocardial infarction and scarring. LGE occurs approximately 10–20 minutes after administering gadolinium-based contrast and results from regional differences in myocardial uptake and washout within the extracellular space, seen in conditions like myocardial injury, edema, necrosis, and fibrosis.[15]

Several studies evaluating the cardiac manifestations of COVID-19 have utilized CMR imaging. Given the variations in the prevalence of cardiac manifestations and to assess the incidence of cardiac involvement in the Indian population, we conducted this study to understand the long-term cardiovascular involvement of COVID-19 beyond 60 days after recovery from the infection.

[1]Consultant, Department of Cardiology; [2,3,5,6]Consultant, Department of Radiology; [4]Honorary Consultant Psychiatrist & Neuropsychiatrist, Director, Medical Research, Department of Psychiatry/ Medical Research; [7]Academic and Research Coordinator, Consultant, Department of Critical Care; [8]Head, Department of Interventional Pulmonology; [9,10]Additional Director, Critical Care, Department of Critical Care; [11]Consultant, Department of Respiratory Medicine; [12]Director of Imaging and Interventional Radiology, Department of Radiology; [13,14]Consultant, Department of Psychology; [15]Research Coordinator, Department of Cardiology, Jaslok Hospital & Research Centre, Mumbai, Maharashtra, India; *Corresponding Author

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Methods

Study Design and Participants

This prospective observational study included patients with SARS-CoV-2 infection confirmed by a positive reverse transcription–polymerase chain reaction (RT-PCR) or rapid antigen test at least 60 days before enrollment. The study was conducted during the first and second waves of COVID-19, which included infections with the Delta variant. All patients were asymptomatic postrecovery and reported no cardiovascular issues related to their illness.

Inclusion Criteria

- Confirmed diagnosis of SARS-CoV-2 infection.
- Recovered from COVID-19 with at least 60 days postpositive test result.
- Signed informed consent to participate in the study.

Exclusion Criteria

- Preexisting cardiac disease, including dilated cardiomyopathy, myocarditis, severe valvular heart disease, infective endocarditis, recent acute coronary syndrome (within the last 3 months), hypertrophic or restrictive cardiomyopathy.
- Current alcohol or substance abuse.
- History of cardiotoxic chemotherapy.
- Active malignancies.
- Chronic kidney disease/end-stage renal disease [creatinine >1.5 mg/dL or glomerular filtration rate (GFR) <50 mg/dL] or on chronic dialysis.
- Contraindications to magnetic resonance imaging (MRI) scanning.
- Pregnancy (negative pregnancy test required before enrollment for women of childbearing potential).
- Allergy to contrast agents used in MRI.
- Patients not confirmed as infected with SARS-CoV-2 by a positive rapid antigen test or RT-PCR, regardless of high-resolution computed tomography (HRCT) findings.
- Unwillingness to participate.

The study results were evaluated against age- and sex-matched adult controls not taking cardiac drugs and with normal cardiac function and volumes and no myocardial scarring (healthy controls; $n = 50$).

Cardiac Magnetic Resonance Scanning Protocol

Cardiac magnetic resonance was performed using a 3 T Philips Ingenia scanner with standardized protocols. For T1 mapping, apical, mid, and basal short-axis Modified Look-Locker (MOLLI) images were attained during breath-hold in expiration. T2 mapping was performed with mGRASE sequences in apical, basal, and mid short-axis slices. LGE images were acquired 10 minutes after administration of 0.2 mmol/kg body weight of gadobutrol (Gadovist; Bayer).

Cardiac function and volumes were quantified using artificial intelligence–based automated contour detection, with manual adjustments applied when necessary. T1 and T2 relaxation times were quantified in apical, mid, and basal slices using motion-corrected images. LGE interpretation was categorized as myocardial or pericardial.

Figure 1 shows representative images of native T1 and T2 mapping in apical, basal, and mid slices.

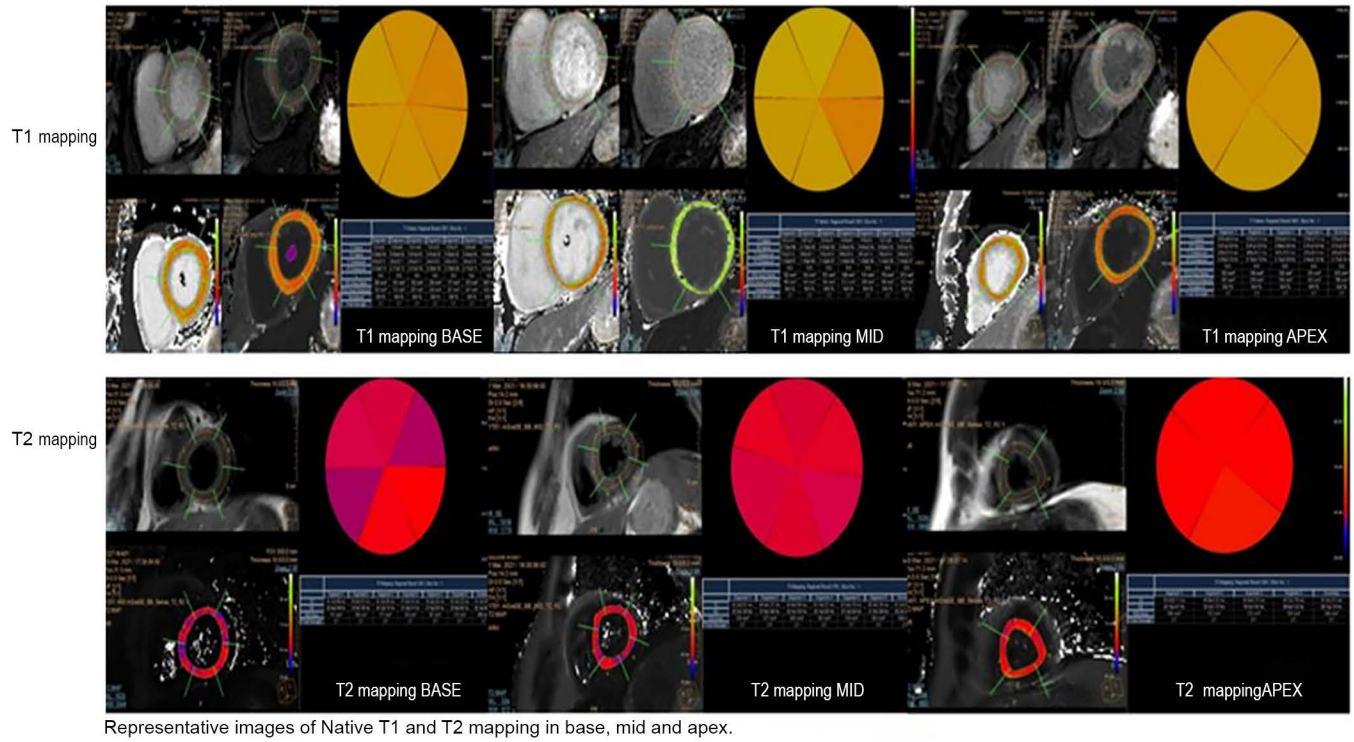
Statistical Analysis

Data were entered and organized in Microsoft Excel. All statistical analyses were conducted using SPSS software, version 23 (IBM Corp., Armonk, NY, USA). Continuous variables are summarized as mean ± standard deviation or as median and interquartile range (IQR), as appropriate, while categorical variables are reported as frequencies and percentages.

For two-group comparisons of continuous data, an independent-samples *t*-test was performed. Linear relationships between two continuous variables were evaluated using Pearson's correlation coefficient for normally distributed data and Spearman's rank correlation coefficient for nonnormally distributed data. A p-value < 0.05 was considered indicative of statistical significance.

Ethical Statement

The study protocol adhered to the Declaration of Helsinki. The protocol was approved by the



Representative images of Native T1 and T2 mapping in base, mid and apex.

Fig. 1: Representative T1 and T2 mapping in healthy controls

hospital's Institutional Ethics Committee before study initiation. Written informed consent was obtained from all participants. No harm was intended for the subjects. Prior to obtaining consent, the advantages and potential risks of participating in the study were discussed with the patients. Participants were not subjected to any additional costs due to the study.

Results

Population Characteristics

A total of 62 recovered COVID-19 patients who completed at least 60 days after the positive COVID-19 test were recruited for the study and underwent CMR. Of these, eight patients were excluded; seven were excluded due to poor breath-hold, and one was excluded due to extensive motion artifacts caused by patient movement. The study population consisted of 54 patients who had recovered from COVID-19 and 50 healthy control participants.

Table 1: Distribution of the cases in terms of age (years) and gender

	Age (years)
Mean (SD)	46.35
Median (IQR)	45.5
Range	23–81
Gender	
Male	38 (70.4%)
Female	16 (29.6%)

The demographic and clinical features of the patients who had recovered from COVID-19 are summarized in Table 1 and Figure 2. The COVID-19-recovered participants had a mean age of 46.35 ± 12.30 years, and 38 of them (70.4%) were male. None of the patients had documented elevated troponin T levels.

Native T1 Values

As summarized in Table 2, the mean native T1 values at the base, mid, and apex in COVID-19-recovered patients were 1258.81 ± 18.00, 1262.95 ± 21.03, and 1285.07 ± 30.54 ms, respectively. Among the control group patients, the mean T1 values were 1255.08 ± 39.90, 1260.91 ± 47.97, and 1293.88 ± 49.95 ms at the base, mid, and apex, respectively. Among the recovered and control individuals, the average native T1 values were 1267.99 ± 19.07 and 1269.96 ± 37.90 ms, respectively. In myocardial T1 relaxation times at the base, mid, apex, or in the average values, no statistical difference was found between the study patients and controls (W = 1425.000, $p = 0.628$).

Figure 3 shows the association between cases and controls with respect to native T1 values at the base, mid, apex, and average values. Elevated T1 values were noted in one patient, specifically in the midinferior segment (as compared to the highest values seen among controls).

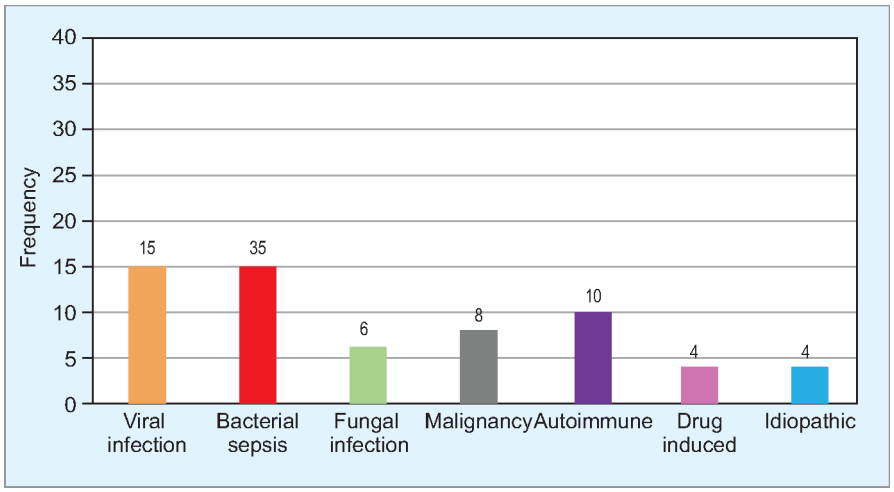


Fig. 2: Age and gender demographics of study participants

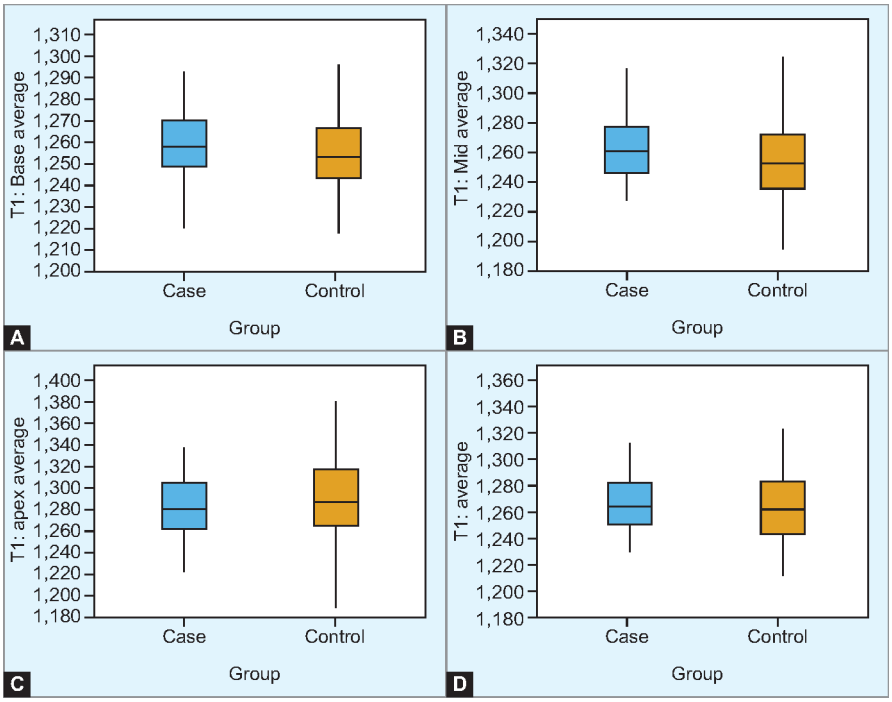
Table 2: CMR imaging T1 and T2 values amongst post COVID-19-infected patients and controls

Variable	*Group*	*Mean (SD)*	*Median (IQR)*	*Min–Max*	*Test statistic (t/W)*	*p-value*
T1: Base average	Case	1258.81 (18.00)	1257.58 (1248.67–1270.33)	1219.17–1313.67	$t = 0.606$	0.546
	Control	1255.08 (39.90)	1253.2 (1242.82–1266.53)	1162.3–1281		
T1: Mid average	Case	1262.95 (21.03)	1260.5 (1245.83–1277.17)	1226.83–1334	W = 1592.000	0.116
	Control	1260.91 (47.97)	1252.5 (1235.6–1271.58)	1141.5–1314		
T1: Apex average	Case	1285.07 (30.54)	1280.75 (1260.69–1305.62)	1222.5–1337.5	W = 1248.000	0.509
	Control	1293.88 (49.95)	1287.5 (1264.08–1316.28)	1189.5–1413		
T1: Average	Case	1267.99 (19.07)	1264.81 (1252.14–1282.91)	1230.81–1312	W = 1425.000	0.628
	Control	1269.96 (37.90)	1262.9 (1243.53–1283.19)	1182.83–1379.97		
T2: Base average	Case	45.81 (1.29)	46.11 (44.81–46.81)	43.12–58.28	$t = -0.551$	0.584
	Control	46.05 (2.81)	45.8 (44.4–47.1)	38.8–54.1		
T2: Mid average	Case	45.97 (1.21)	45.9 (45.36–46.59)	42.74–56.1	$t = -0.189$	0.851
	Control	46.04 (2.24)	46.3 (44.52–47.7)	40.4–49.4		
T2: Apex average	Case	47.86 (2.17)	48.35 (46.04–49.53)	42.58–51.42	$t = -4.571$	<0.001*
	Control	49.98 (2.53)	50.2 (48.65–51.58)	43.3–55.4		
T2: Average	Case	46.43 (0.98)	46.54 (45.66–47.14)	44.15–48.18	$t = -3.137$	0.002*
	Control	47.21 (1.47)	47.37 (46.1–48.25)	44.07–50.5		

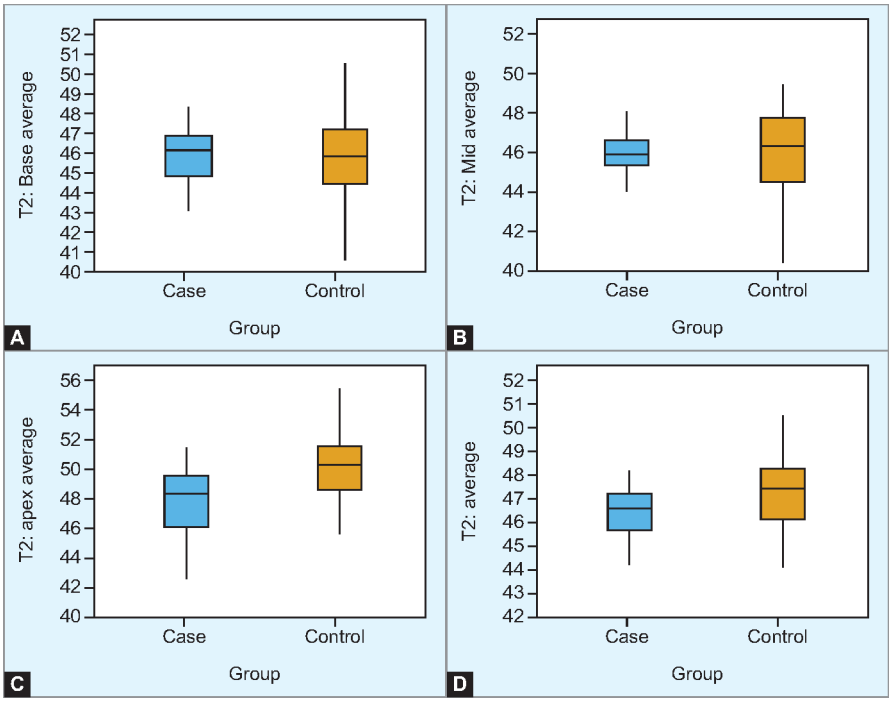
*The T2 values among the controls were found to be higher than those in the cases; however, this finding is clinically irrelevant; *t*, *t*-test; W, Wilcoxon–Mann–Whitney *U* test

Table 3: CMR imaging LGE

LGE	*Post COVID-19 patients*	*Healthy controls*
Myocardial enhancement	1	0
Pericardial enhancement	0	0



Figs 3A to D: Distribution of T1 values in the base, mid, apex, and T1 average in cases and controls



Figs 4A to D: Distribution of T2 values in the base, mid, apex, and T2 average in cases and controls

Native T2 Values

As shown in Table 2 the mean native T2 values at the base, mid, and apex in the COVID-19-recovered patients were 45.81 ± 1.29, 45.97 ± 1.21, and 47.86 ± 2.17 ms, respectively. Among controls, the mean T2 values were 46.05 ± 2.81, 46.04 ± 2.24, and 49.98 ± 2.53 ms at the base, mid, and apex, respectively. Among the recovered and control individuals, the average native T2 values were 46.43 ± 0.98 and 47.21 ± 1.47 ms, respectively. No statistically significant difference was identified between the recovered patients and controls in myocardial T2 relaxation times at the base, mid, apex, or average values. Additionally, T2 values were not elevated in cases compared to controls.

Figure 4 illustrates the association between cases and controls with respect to native T2 values at the base, mid, apex, and average values. Elevated T2 values were observed in one patient, specifically in the midinferior segment (as shown in Figure 5). No patient had myocardial hemorrhage on the scan.

Late Gadolinium Enhancement

LGE was noted in 1 patient as midmyocardial enhancement (as shown in Table 3 and Figure 5). In the midinferior segment, midmyocardial enhancement and raised native T1 and T2 values were noted. The patient with midmyocardial enhancement, however, did not show decreased left ventricular ejection fraction (LVEF), right ventricular ejection fraction (RVEF), or raised troponin levels.

Left Ventricular Ejection Fraction Analysis

Mean (SD) of LVEF (%) was 67.41 (4.17). The median (IQR) of LVEF (%) was 67.00 (64–70.75). The LVEF (%) ranged from 60–78 (Table 4).

The distribution showed minimal skewness (0.12), indicating that it was normally distributed. The kurtosis value of −0.68 was consistent with a normal distribution. A nonsignificant Shapiro–Wilk test ($p = 0.396$) also suggested normally distributed data. There appeared to be only one mode/peak in the data, confirming that the data were unimodal, as represented in Figure 6.

Right Ventricular Ejection Fraction Analysis

The mean (SD) of RVEF (%) was 52.28 (4.23). The median (IQR) of RVEF (%) was 52.00 (49–55). The RVEF (%) ranged from 45–62 (Table 5).

The distribution showed minimal skewness (0.27), indicating that it was normally distributed. The kurtosis value of −0.89 was consistent with a normal distribution. A nonsignificant Shapiro–Wilk test ($p = 0.141$) also suggested normally distributed data. There appeared to be only one mode/peak in the data, confirming that the data were unimodal, as represented in Figure 7.

Morphological Analysis

Six patients were found to have mild concentric hypertrophy. This appears to be an incidental finding with no obvious relation to COVID-19.

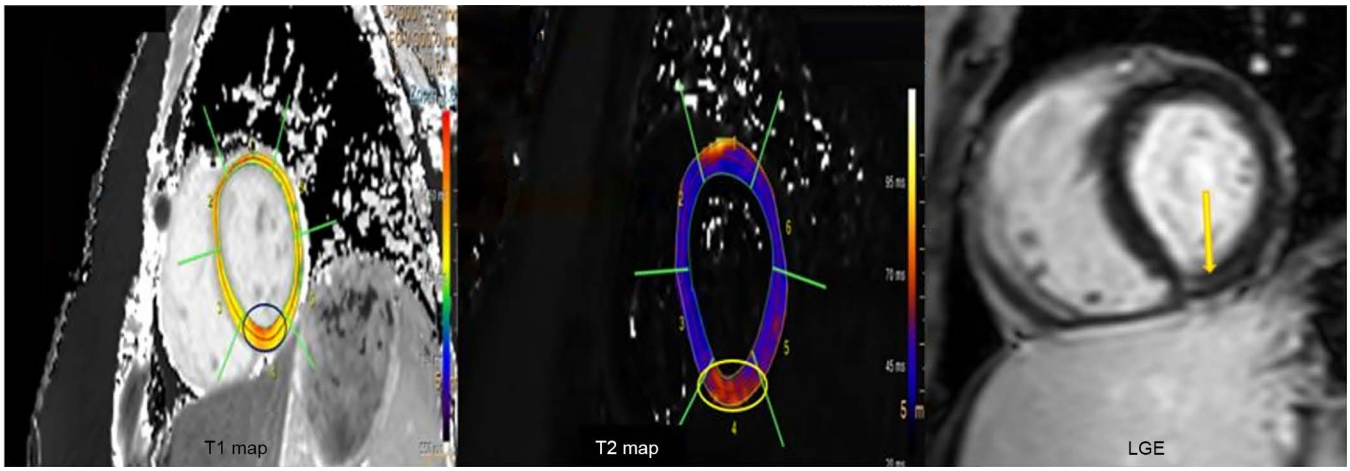


Fig. 5: Elevated midmyocardial T1 and T2 values and LGE in the basal inferior and mid inferior locations

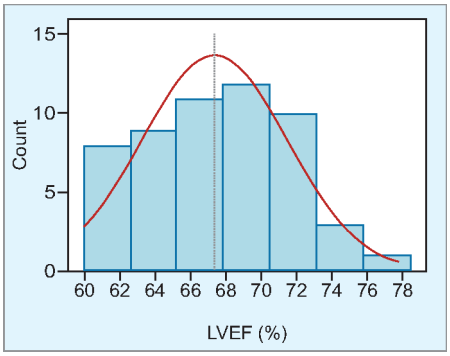


Fig. 6: Distribution of left ventricular ejection fraction (LVEF)

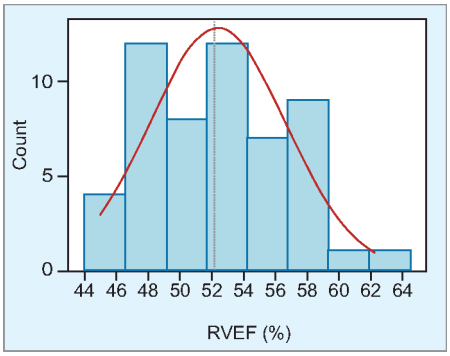


Fig. 7: Distribution of right ventricular ejection fraction (RVEF)

Table 4: Distribution of the participants in terms of LVEF (%)

	LVEF (%)
Mean (SD)	67.41 (4.17)
Median (IQR)	67 (64–70.75)
Range	60–78

Table 5: Distribution of the participants in terms of RVEF (%)

	RVEF (%)
Mean (SD)	52.28 (4.23)
Median (IQR)	52 (49–55)
Range	45–62

Discussion

Cardiovascular involvement due to COVID-19 infection has been reported worldwide, with prevalence rates ranging from 1.4 to 58–78%.[1–3] Most studies have been dedicated to findings during the acute phase of the illness, with less research on the cardiac consequences of "long COVID." This study aimed to assess potential cardiac pathologies after 60 days of recovery from COVID-19 in individuals without preexisting cardiovascular comorbidities. Patient scans included CMR imaging, with parameters such as native T1 and T2 values, LGE, left ventricular ejection fraction (LVEF), and right ventricular ejection fraction (RVEF) being studied. The primary objective was to identify myocarditis, pericarditis, and heart failure due to long COVID syndrome. In our study, myocarditis and pericarditis were rare, with only one patient (1.85%) demonstrating myocarditis (raised native T1 and T2 values and LGE) among 54 patients. Six patients had mild concentric hypertrophy, but this appeared to be incidental, with no clear clinical relationship to COVID-19.

None of the patients exhibited abnormal left or right ventricular ejection fractions suggestive of heart failure.

As mentioned previously, several studies have reported varied results pertaining to the cardiovascular effects of SARS-CoV-2 infection. Puntmann et al.[10] studied 100 individuals who recovered from COVID-19, with a gap of 64–92 days between COVID-19 diagnosis and CMR analysis, and found that 78 patients (78%) had abnormal CMR findings. They reported that 73% had raised native T1 values, 60% had raised T2 values, 32% had myocardial LGE, and 22% had pericardial enhancement. They concluded that a substantial proportion of people with recent SARS-CoV-2 infection exhibited cardiac involvement, highlighting the need for investigation into long COVID cardiac consequences. Similarly, Huang et al.[9] found that 15 patients (58%) out of a cohort of 26 patients recovered from SARS-CoV-2 had abnormal findings on CMR, including myocardial edema, cardiac fibrosis, as well as impairment of right ventricular function.

Xie et al.[16] used national healthcare databases from the US Department of Veterans Affairs to study 1,53,760 individuals with COVID-19, along with control cohorts, to assess the risks and burden of cardiovascular outcomes at 1 year. They demonstrated that after the first 30 days of infection, individuals had an increased risk of cardiovascular complications, including ischemic and nonischemic heart disease, heart failure, cerebrovascular disorders, arrhythmias, pericarditis, myocarditis, as well as thromboembolic disease. Another retrospective double-cohort study consisting of symptomatic and asymptomatic COVID-19-infected patients and uninfected people concluded that both symptomatic and asymptomatic patients were at an increased risk of late cardiovascular outcomes.[17]

A pathological study during autopsy on COVID-19 patients' hearts identified myocyte necrosis as being caused most commonly by microthrombi, suggesting antithrombotic strategies may mitigate COVID-19's cardiac effects.[18] Another literature review summarized several possible mechanisms of myocardial injury following SARS-CoV-2 infection.[19]

Similar to our study, several others have reported that COVID-19 is not associated with an increase in the incidence of cardiovascular involvement. Breitbart et al.[20] found that only one patient (2%) had myocarditis and eight patients (14%)

had suspicious CMR findings out of 56 post-COVID-19 infection patients. They hypothesized that myocarditis in post-COVID-19 patients was infrequent and that, among the eight patients with suspicious CMR findings, myocarditis was unlikely because left ventricular function was preserved. Tuvali et al.[21] analyzed data from 1,96,992 patients, with findings of myocarditis in 9 cases and pericarditis in 11 cases among COVID-19-recovered patients, with no significant difference in incidence compared to controls. Starekova et al.[8] reported similar results, finding myocarditis in only two (1.4%) out of 145 athletes recovering from COVID-19. Kotecha et al.[22] found no significant difference in the proportion of LGE or T1 and T2 abnormalities between case and control subjects. Another study found no differences in cardiac function, structure, tissue characterization, or biomarkers between COVID-19-seropositive and seronegative healthcare workers.[23]

Large variability in the incidence of cardiac manifestations post-COVID-19 infection is observed. Possible reasons for this variability include:

- Inconsistent use of the 2018 Lake Louise criteria for interpreting CMR findings (as noted by Breitbart et al.[20]).
- Preexisting cardiac disease confounding CMR findings, which our study sought to exclude.
- Raised native T2 values, which are not specific to myocarditis and may occur in other cardiac pathologies.
- Lack of controls with other viral infections.

Limitations

This investigation was carried out at one center, with a relatively small cohort, and did not include control groups with other viral infections.

Conclusion

In our study of 54 patients who had COVID-19 infection and were at least 60 days from the infection, LGE (myocarditis) was noted in only 1 patient (1.85%), who also had raised native T1 and T2 values. The remaining 53 patients (98.15%) showed no LGE or increase in native T1 and T2 values compared to controls. Overall, our study suggests that there is no statistically significant increase in cardiac manifestations in patients who have recovered from COVID-19 compared to uninfected controls. We believe the frequency of cardiovascular comorbidities related to long COVID syndrome is much lower in the Indian population than reported in Western literature. These findings are reassuring and may help alleviate concerns among the Indian population regarding the long-term cardiovascular effects of COVID-19 infection.

Data Availability

Available on reasonable request.

Funding

The research was funded by Jaslok Hospital and Research Centre, Mumbai, where the research was carried out, and an approximate fund of Rupees 18,78,240 was contributed by the institute (62 patients underwent the study, of which eight were excluded, and 50 healthy controls were included).

Orcid

Tanushree Somir Banerjee https://orcid.org/0009-0009-2692-3464

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