Original Article

Long-term impact of COVID-19 on cardiovascular health of healthcare workers of a tertiary care hospital of Bangladesh

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ABSTRACT

Background: *The coronavirus disease (COVID-19) is a multisystem disease that sometimes affects the cardiovascular system (CVS) during acute illness and follows a protracted course in a proportion of patients causing 'post-COVID syndrome'. Exercise tolerance test (ETT) can assess functional capacity and imaging modalities like echocardiography and cardiac magnetic resonance (CMR) can assess structural and functional status of cardiovascular system (CVS) efficiently. However, the pattern of cardiovascular involvement and its effect on functional capacity in COVID-19 survivors long after the index illness are inadequately known. This study was carried out to detect the functional status and imaging findings in COVID-19 recovered healthcare workers.*

Methods: *This cohort study was carried out in the Department of Cardiology of the National Institute of Cardiovascular Diseases (NICVD), Dhaka, Bangladesh from July 2021 to December 2021. Thirty male healthcare workers, previously not known to have any cardiac disease, who suffered from reverse transcriptase polymerase chain reaction (RT-PCR)-confirmed COVID-19 of mild severity at least 3 months back were included. Besides clinical evaluation, ETT, echocardiography and CMR were done.*

Results: *The mean age was 37.2±4.7 years. The mean time interval from RT-PCR positivity was 317.4±85.7 days. The functional capacity was good in the study subjects; the achieved metabolic equivalents (METs) were 13.0±1.5. The mean left ventricular ejection fraction (LVEF) was 66.9% ± 6.2%; all were within the normal range. The mean LV global longitudinal strain (LVGLS) was -19.2% ± -1.9%; 6 patients had LVGLS <-18%. One patient had mildly reduced tricuspid annular plane systolic excursion (TAPSE) of 16 mm but 3 patients had reduced lateral tricuspid annulus peak systolic velocity (RVS'). CMR revealed subtle abnormalities in 14 (46.7%) patients. Eight patients (26.67%) had increased T2 signal indicating myocardial oedema, 2 (6.7%) had subepicardial late gadolinium enhancement (LGE).*

Conclusion: *In healthcare workers who recovered from mild COVID-19, despite preserved functional capacity, subclinical cardiac abnormalities detected by echocardiography and CMR may be present. Long-term follow-up is warranted to define the clinical significance of these findings.*

Key words: *post-acute COVID-19 syndrome, myocarditis, echocardiography, magnetic resonance imaging, Bangladesh.*

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INTRODUCTION

The coronavirus disease (COVID-19) is a multisystem disease. It may affect the cardiovascular system (CVS) during acute illness, the manifestation of which ranges from subclinical involvement to acute myocarditis and acute coronary syndrome.[1,2] In a subset of COVID-19 patients, cardiovascular (CV) involvement follows a protracted course.[3-5] The array of manifestations after the index COVID-19 illness has been recognized as a post-COVID syndrome, long COVID or post-COVID conditions.[6,7] According to the World Health Organization (WHO), post-COVID-19 condition occurs in individuals with a history of probable or confirmed severe acute respiratory syndrome-coronavirus type 2 (SARS-CoV-2) infection, usually 3 months from the onset of COVID-19 with symptoms and that last for at least 2 months and cannot be explained by an alternative diagnosis.[8] Recently, the American College of Cardiology (ACC) has issued a clinical guidance on cardiovascular consequences of COVID-19.[9] The ACC used the term "Post-acute Sequelae of SARS-CoV-2 Infection (PASC)" to encompass a constellation of symptoms that emerge or persist weeks to months after recovery from COVID-19. PASC has further been divided into PASC cardiovascular disease (PASC-CVD) and PASC cardiovascular syndrome (PASC-CVS). PASC-CVD refers to a broad group of CV conditions that include but are not limited to, myocarditis and other forms of myocardial involvement, pericarditis, new or worsening myocardial ischemia, microvascular dysfunction, non-ischemic cardiomyopathy, thromboembolism, cardiovascular sequelae of pulmonary disease and arrhythmia. PASC-CVS includes widely ranging CV symptoms, without objective evidence of CVD using standard diagnostic testing. A recently published study using the national healthcare databases from the US Department of Veterans Affairs involving 153,760 individuals with COVID-19, 5,637,647 contemporary controls and 5,859,411 historical controls evidenced that the risk and 1-year burden of CV disease in survivors of acute COVID-19 are substantially increased as compared with the contemporary and historical cohorts and the risks increased with severity of the index acute illness.[10] Ongoing cardiac involvement may occur even in those COVID-19 survivors who were not known to have cardiac manifestations during the acute illness. And this has been reported in patients suffering from critical to mild, or even asymptomatic patients.[5] The long-term prognosis of cardiac involvement in COVID-19 is unknown; however, myocardial dysfunction and cardiomyopathy may be a significant sequela.[5] Besides clinical skills, serological investigations for inflammation (e.g., high-sensitivity C-reactive protein, hs-CRP), myocardial injury (e.g., high-sensitivity cardiac troponin I, hs-cTnI) and heart failure [e.g., N-terminal (NT)-prohormone BNP (NT-proBNP)], as well as, cardiac imaging, including echocardiography and magnetic resonance imaging of heart or cardiac magnetic resonance (CMR), have been employed to detect and monitor the cardiac involvement in COVID-19 survivors.[4,11-14] For echocardiographic evaluation, in addition to conventional 2D, M-mode and Doppler modalities, strain imaging has been utilized to pick up the subtle functional abnormalities of the myocardium.[15] On the other hand, CMR, in addition to assess ventricular volumes and function more efficiently, can perform advanced tissue characterisation uniquely.[16] This has made CMR a critical tool for clinicians to understand cardiovascular involvement and for risk stratification of patients recovering from COVID-19.[16] However, the knowledge in this field is rapidly evolving but is still incomplete. Moreover, the published data are mainly from the developed world. The present study was carried out to evaluate the cardiac status of healthcare workers working in a tertiary care hospital who recovered from COVID-19.

METHODS

This cohort study was carried out in the Department of Cardiology of the National Institute of Cardiovascular Diseases (NICVD), Dhaka, Bangladesh from July 2021 to December 2021. Thirty male healthcare workers of 20-50 years, who suffered from reverse transcriptase polymerase chain reaction (RT-PCR)-confirmed COVID-19 of mild severity, at least 3 months back, were included. Patients with known cardiac disease, known clinical cardiac involvement during the COVID-19 illness and significant comorbidity like bronchial asthma and chronic obstructive pulmonary disease were excluded. Exercise tolerance test (ETT) was done following the Bruce protocol and was symptom-limited. The adult treadmill ergometer EC-12LT (Labtech Ltd., Vág street 4. H-4031 Debrecen, Hungary) was used and the protocol by the American College of Cardiology/American Heart Association (ACC/AHA) was followed.[17]

Echocardiographic examination was carried out by Vivid E95 (GE Healthcare, 9900 Innovation Drive, Wauwatosa, WI 53226, USA) following the recommendations of the American Society of Echocardiography (ASE).[18] CMR was done using 3T MRI MAGNETOM Vida (Siemens Healthcare GmbH, Henkestr. 127, 91052 Erlangen, Germany) and findings were reported.[19]

The study protocol was approved by the Ethical Review Committee of NICVD (NICVD/Research/2020-21/121). Informed written consent was taken from each patient. Data were collected in an approved data collection form.

RESULTS

The study involved 30 healthcare workers recovered from COVID-19 of mild severity. The mean age was 37.2 years, ranging from 31 to 50 years. All were male. The mean time interval from RT-PCR positivity to study was 317.4±85.7 (range, 181 to 438 days) days. Serological tests reports including CRP, cTnI and NT-proBNP were within normal limits (Table I).

Resting 12-lead electrocardiogram (ECG) findings were normal in all patients. The functional capacity was good in the study subjects; the achieved metabolic equivalents (METs) were 12.98±1.51, ranging from 10.20 to 17.10. The chronotropic and haemodynamic response were also normal. None had evidence of provocable myocardial ischaemia. Echocardiography constituted M-mode, 2-D, Doppler and strain imaging and the findings are depicted in Table II and Figures 2 and 3). The cavity dimensions were normal. The mean LV ejection fraction (LVEF) was 66.70%±6.90%, ranging from 53% to 80%. The measures of right ventricular systolic function were also normal: the mean tricuspid annular plane systolic excursion (TAPSE) was 21.63±3.10 mm, and RVS' was 11.50±1.14 cm/s. None of the 30 study subjects had LV diastolic dysfunction at the time of inclusion. LV global longitudinal strain (LVGLS) was also assessed. The mean GLS was -19.41%±1.87%, ranging from -14.80% to 27.10%. Three out of 30 study subjects had reduced LVGLS (i.e., <-18%):.-14.8%, -15.2%, and -15.9%. CMR was done in all of 30 patients and the findings are displayed Tables III and IV. The findings revealed subtle abnormalities in 14 (46.67%). Among those with positive CMR findings, 8 patients (8 of 14, 57.1%) had increased T2 signal indicating myocardial edema. Two cases (2 of 14, 14.29%) showed subepicardial late gadolinium enhancement (LGE), involving the mid inferior and mid infero-lateral segments. When compared with the negative counterparts, the patients with positive CMR findings had increased global native T1, T2 and extracellular volume (ECV) values and the differences were statistically significant ($p < 0.001$, 0.011 and <0.001, respectively).

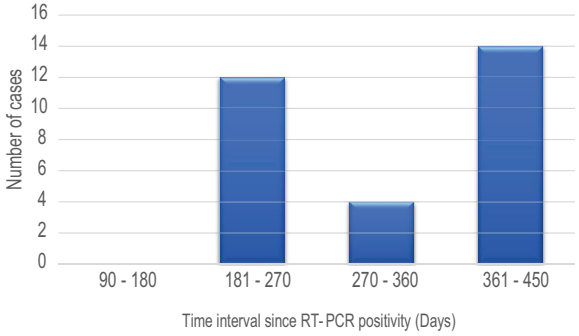

Figure 1. Time interval since RT-PCR positivity to study (N=30)

Table I. Serological tests of the study subjects (N=30)

Tests	N	Minimum	Maximum	Mean	SD
CRP (mg/L)	30	3.00	14.00	3.9667	2.37056
NT-proBNP (pg/mL)	30	5.00	71.05	22.0703	17.21955
Troponin I (ng/ml)	30	.0010	.0210	.005973	.0060072

Table II. Echocardiographic parameters of the study subjects (N=30)

	N	Minimum	Maximum	Mean	Std. Deviation
LVIDD (mm)	30	38	58	47.93	4.008
LVIDS (mm)	30	23	40	29.73	4.102
LVEF (%)	30	53	80	66.70	6.904
LAVI (ml/m^2)	29	20.79	37.50	28.4369	4.31005
TAPSE (mm)	30	16	28	21.63	3.102
RVS‘ (cm/sec)	29	9	14	11.52	1.430
LVGLS (%)	30	-21.70	-14.80	-19.4033	-1.84381
E/A	30	.60	2.00	1.2733	.29117
DT (millisecond)	30	120.00	198.00	160.3667	20.74390
E/e’	30	1.50	11.10	6.8500	1.75946

Here, LVIDD = left ventricular internal diameter in diastole, LVIDS = left ventricular internal diameter in systole, LVEF = left ventricular ejection fraction, LAVI = left atrial volume index, TAPSE = tricuspid annular plane systolic excursion, RVS‘ = lateral tricuspid annulus peak systolic velocity, LVGLS = left ventricular global longitudinal strain, E/A = mitral peak E and A wave velocity ratio, DT = deceleration time, E/e’ = mitral peak E and e’ eave velocity ratio.

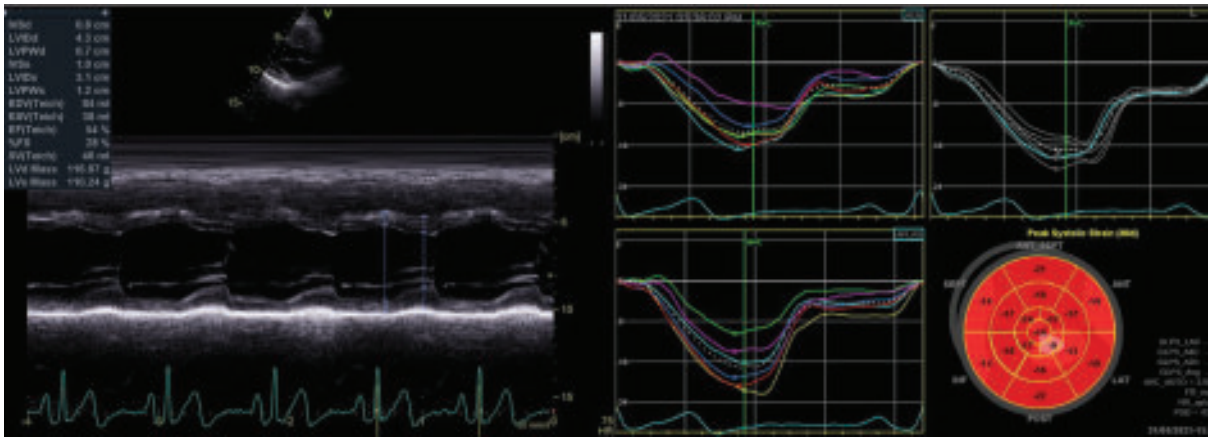

Figure 2. Echocardiography of AS. LVEF was 54% and LVGLS was reduced to -15.9%

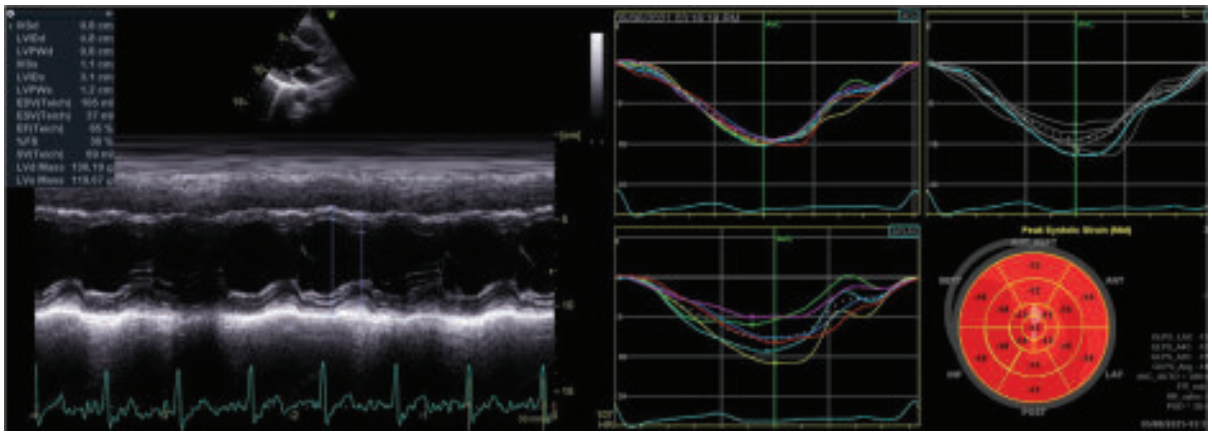

Figure 3. Echocardiography of JI. LVEF was 65% and LVGLS was reduced to -14.3%

Table III. Left ventricular CMR parameters of patients recovered from COVID-19 (N=30)

LV parameter	CMR Findings		p value
	Negative (n=16)	Positive (n=14)	
EF (%)#	62.0 ± 7.7	61.7 ± 5.5	0.898
EDV (ml)#	102.8(91.5-112.75)	111(101.25-136.5)	0.037
ESV (ml)#	40.2 ± 12.2	15.1 ± 13.4	0.298
SV (ml)#	60.2 ± 11.2	68.9 ± 12.9	0.058
Myo mass/BSA (g/m^2)#	55.3 ± 10.3	60.9 ± 9.2	0.124
EDVI (ml/m^2)	55.9 ± 9.9	66.4 ± 12.1	0.014
ESVI (ml/m^2)	21.2 ± 6.6	25.4 ± 7.5	0.116
SVI (ml/m^2)	34.1 ± 7.2	40.6 ± 7.1	0.020
Global T1 (ms)#	1155.5(1137.25-1172.75)	1231(1218.25-1257.5)	<0.001
<1200*	16(100.0)	2(14.3)	
>1200*	0(0.0)	12(85.7)	
Global T2 (ms)#	35.5(34-37)	40(34.5-43.25)	0.011
<40*	16(100.0)	6(42.9)	
>40*	0(0.0)	8(57.1)	
Global ECV (%)#	23.5(21.25-24.0)	31(29.75-33.25)	<0.001
<25*	16(100.0)	3(21.4)	
>25*	0(0.0)	11(78.6)	
Myocardial oedema#	1.3 ±0.3	1.8 ±0.4	<0.001
<2*	16(100.0)	6(42.9)	
>2*	0(0.0)	8(57.1)	
LGE#	2.0 ± 0.0	1.8 ± 0.4	<0.001

Figures in the parentheses indicate corresponding %
* Chi square test (χ^2) was done to analyzed the data.
# Data were analyzed using unpaired t-test and were presented as mean ± SD.

Here, LV = left ventricle, EF = ejection fraction, EDV = end diastolic volume, ESV = end systolic volume, SV = stroke volume, EDVI = end diastolic volume index, ESVI = end systolic volume index, SVI = stroke volume index, Myo mass = myocardial mass, ECV = extra-cellular volume, LGE = late gadolinium enhancement.

Table IV. Right ventricular CMR parameters of patients recovered from COVID-19 (N=30)

RV Parameter	CMR Finding		p values
	Negative (n=16)	Positive (n=14)	
EF (%)#	54.5(52.0-57.75)	32.05(25.25-39.0)	<0.001
>50*	16(100.0)	0(0.0)	
<50*	0(0.0)	14(100.0)	
EDV (ml)#	95(71.75-99.75)	117.5(102.0-134.25)	<0.001
<103*	16(100.0)	3(63.3)	
>103*	0(0.0)	11(78.6)	
ESV (ml)#	43.3 ± 14.3	76.6 ± 22.2	<0.001
SV (ml)#	43.9 ± 11.3	38.7 ± 12.7	0.248
EDVI (ml/m^2)	47.5 ± 9.9	66.8 ± 12.8	<0.001
ESVI (ml/m^2)	27.5 ± 10.7	43.4 ± 12.8	0.001
SVI (ml/m^2)	23.5 ± 6.3	23.2 ± 10.6	0.938

Figures in the parentheses indicate corresponding %
* Chi-square test (χ^2) was done to analyzed the data.
# Data were analyzed using unpaired t-test and were presented as mean ± SD.

Here, RV = right ventricle, EF = ejection fraction, EDV = end diastolic volume, ESV = end systolic volume, SV = stroke volume, EDVI = end diastolic volume index, ESVI = end systolic volume index, SVI = stroke volume index.

DISCUSSION

In this study, CRP, a marker of inflammation, was found to be within normal range in all the participants. High sensitivity CRP (hs-CRP) differed significantly between patients recently recovered from COVID-19 and the non-COVID controls.[14] Cardiac troponin I (Tn I) was assayed and was found within normal range in all. Huang et al. carried out a CMR-based study involving 26 patients recovered from COVID-19 to see sustained cardiac involvement after recovery. The high sensitivity troponin I (hs-TnI) was in the normal range for all recovered patients at the time of CMR.[13] In their CMR-based study involving COVID-19 recovered patients (n = 100), age-matched and sex-matched healthy volunteers (n = 50) and risk factor–matched patients (n = 57), Puntmann et al. found significantly elevated hs-TnI more commonly in COVID-19 recovered patients than the counterparts, but the differences were not statistically significant.[14] Out of 79 COVID-19 survivors to see adverse ventricular remodeling by echo-cardiography, none had hs-TnI above the 99% percentile at 3 months in another study by Moody et al.[20] So, the findings of the present study correlate well with the studies carried out elsewhere.

Plasma NT-proBNP was measured and was found within normal range in all of the patients. NT-proBNP was in the normal range for all recovered patients and no statistically significant difference was found in plasma NT-proBNP level between the COVID-19 recovered patients with and without positive CMR findings at the time of CMR in the study by Huang et al.[13] Plasma NT-proBNP level did not differ significantly among COVID-19 recovered patients and the controls in the study by Puntmann et al.[14] In the study by Moody et al. the median NT-proBNP level was 76 (20-246) ng/L, however, in 8 out of 79 studied patients, the level was >450 ng/L.[20] Resting 12-lead ECG was normal in all patients. The functional status assessed by ETT was not reported previously.

Echocardiographic evaluation included 2D, M-mode, Doppler and strain imaging. 2D and M-mode findings were within normal range. None had left atrial dilatation or right ventricular enlargement. LVEF was also normal in all. A recent report from Hong Kong studied CV

sequalae in 97 uncomplicated COVID-19 survivors 1- 4 weeks after hospital discharge; the mean LVEF was 66.4 ± 6.8% and 1 patient had LVEF 44%.[4] Assessment of LV global longitudinal strain by speckle-tracking echocardiography has been employed to look for the more subtle deficit in LV systolic function. In the present study, LVGLS was obtained in 29 patients. For the normal cut-off of ≥-18%, 3 patients had reduced LVGLS despite having normal LVEF. Özer et al. evaluated LVGLS by 2D speckle tracking echocardiography (2D-STE) within 29.5 ± 4.5 days after COVID-19 in 74 patients with or without troponin I elevation during index illness. With the cut-off of -18%, LVGLS was found abnormal in 28 (37.8%) patients. The average LVGLS values of the group with myocardial injury tended to be higher than the group without myocardial injury (-17.7 ± 2.6 vs. -18.9 ± 1.8, p = 0.051).[21] In our study, all were apparently healthy before COVID-19, all had mild disease and the time lapse since recovery was longer. Another echocardiography-based study from Argentina reported abnormal LVGLS more commonly in patients recovered from COVID-19 compared to the healthy controls.[22] CMR has been employed to look for the ongoing subclinical involvement of the myocardium in COVID-19 recovered patients. The observed abnormalities include elevated T1 values (a marker of fibrosis or inflammation), increased T2 values (a marker of oedema), and myocarditis-like LGE patterns.[5]

In the present study, CMR revealed subtle abnormalities in 14 (46.67%) patients, including increased T2 signal indicating myocardial edema in 8 patients and subepicardial LGE in 2 patients indicating fibrosis. The CMR findings in post-COVID patients vary widely in available literature. Initial CMR-based studies concerning COVID-19 survivors reported high incidence of cardiac involvement. In the study by Puntmann et al., out of 100 patients recently recovered from COVID-19, 78 (78%) had abnormal CMR findings, including raised myocardial native T1 (n = 73), raised myocardial native T2 (n = 60), myocardial LGE (n = 32) or pericardial enhancement (n = 22).[14] Myocarditis-like LGE pattern on cardiac MRI was reported in 15% of college athletes by Rajpal et al.[11] None of the 26 participants required hospitalization, and most (73%) were asymptomatic. Cardiac MRI revealed ongoing myocarditis or prior evidence of myocardial injury in 12 subjects (46%) despite largely normal ejection fraction.[11] In a similar study, Malek et al. evaluated the cardiac MRI of 26 consecutive elite athletes with a median age of 24 years. Participants were asymptomatic or had mild disease (77%). ECG had no abnormalities and troponin levels were not elevated; however, cardiac MRI was abnormal in 5 of 26 (19%) athletes, including 4 with features of isolated myocardial edema and 1 manifesting non-ischemic late gadolinium enhancement with pleural and pericardial effusion.[23] Subsequent CMR-based studies did find cardiac involvement in COVID-19 recovered patients, but at a lower rate. A large study of 1,597 college athletes with COVID-19 found only 2.3% with myocarditis, most of whom were asymptomatic.[24] Abnormal CMR findings were rather uncommon, e.g., LGE myocardial scarring in 4% of 74 COVID-19 recovered health- care workers only. In fact, CMR findings and other end-points did not significantly between the survivors of COVID-19 and matched healthy controls.[25] Also, no correlations were found between abnormal cardiac MRI findings and biomarker in COVID-19 recovered patients in general. At present, the long-term clinical significance of the abnormal CMR findings is mostly unknown.

Limitations

The study has got some critical limitations. The sample size was small, and no controls were included. Only mild cases were considered. Investigation findings at baseline and at the time of COVID-19 were not available. RV and LA strain were not assessed. No correlation was done among serological, echo, and CMR findings.

Conclusion

In healthcare workers who recovered from mild COVID-19, subclinical cardiac involvement detected by echocardiography and CMR may be present in a proportion in absence of clinical and serological abnormalities. Abnormal LVGLS may be more commonly encountered than the conventional measures of systolic function by echocardiography. CMR may detect even subtle structural and functional abnormalities of heart. The clinical significance of these findings is at present uncertain but may warrant long-term monitoring.

Authors' contribution: MJU was involved in generation of the concept, AKMMI contributed to generation of concept, review of literature and writing of manuscript, KFA reviewed literature, collected data and wrote manuscript, NG took part in literature review,

data collection and review of results, SR was involved in data collection, data analysis and review of results, and SS, MRM, MH and AKSZMK critically reviewed manuscript.

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Conflicts of interest: Nothing to declare.

REFERENCES

1. Toloui A, Moshrefiaraghi D, Neishaboori AM, Yousefifard M, Haji Aghajani M. Cardiac complications and pertaining mortality rate in COVID-19 Patients; a systematic review and meta-analysis. Arch Acad Emerg Med 2021 Feb 13;9(1):e18.
2. Dy LF, Lintao RCV, Cordero CP, Cabaluna ITG, Dans LF. Prevalence and prognostic associations of cardiac abnormalities among hospitalized patients with COVID-19: a systematic review and meta-analysis. Sci Rep 2021 Apr 19;11(1):8449.
3. Akbarialiabad H, Taghrir MH, Abdollahi A, Ghahramani N, Kumar M, Paydar S, et al. Long COVID, a comprehensive systematic scoping review. Infection 2021 Dec;49(6):1163-86.
4. Zhou M, Wong CK, Un KC, Lau YM, Lee JC, Tam FC, et al. Cardiovascular sequalae in uncomplicated COVID-19 survivors. PLoS One 2021 Feb 11;16(2):e0246732.
5. Satterfield BA, Bhatt DL, Gersh BJ. Cardiac involvement in the long-term implications of COVID-19. Nat Rev Cardiol 2022 May;19(5):332-41.
6. Yong SJ. Long COVID or post-COVID-19 syndrome: putative pathophysiology, risk factors, and treatments. Infect Dis (Lond) 2021 Oct;53(10):737-54.
7. Centers for Disease Control and Prevention. Post-COVID conditions: Information for healthcare providers, updated July 9, 2021. Available at: https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-care/post-covid-conditions.html, accessed 30 June, 2022.
8. World Health Organization. Post COVID-19 condition (Long COVID), 16 October, 2021. Available at: https://www.who.int/srilanka/news/detail/16-10-2021-post-covid-19-condition, accessed 30 June, 2022.
9. Writing Committee, Gluckman TJ, Bhave NM, Allen LA, Chung EH, Spatz ES, et al. 2022 ACC Expert Consensus Decision Pathway on Cardiovascular Sequelae of COVID-19 in Adults: Myocarditis and Other Myocardial Involvement, Post-Acute Sequelae of SARS-CoV-2 Infection, and Return to Play: A Report of the American College of Cardiology Solution Set Oversight Committee. J Am Coll Cardiol 2022 May 3;79(17):1717-56.
10. Xie Y, Xu E, Bowe B, Al-Aly Z. Long-term cardiovascular outcomes of COVID-19. Nat Med 2022 Mar;28(3):583-90.
11. Rajpal S, Tong MS, Borchers J, Zareba KM, Obarski TP, Simonetti OP, et al. Cardiovascular magnetic resonance findings in competitive athletes recovering from COVID-19 infection. JAMA Cardiol 2021 Jan 1;6(1):116-8.
12. Wang H, Li R, Zhou Z, Jiang H, Yan Z, Tao X, et al. Cardiac involvement in COVID-19 patients: mid-term follow up by cardiovascular magnetic resonance. J Cardiovasc Magn Reson 2021 Feb 25;23(1):14.
13. Huang L, Zhao P, Tang D, Zhu T, Han R, Zhan C, et al. Cardiac involvement in patients recovered from COVID-2019 identified using magnetic resonance imaging. JACC Cardiovasc Imaging 2020 Nov;13(11):2330-9.
14. Puntmann VO, Carerj ML, Wieters I, Fahim M, Arendt C, Hoffmann J, et al. Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from coronavirus disease 2019 (COVID-19). JAMA Cardiol 2020 Nov 1;5(11):1265-73.
15. Croft LB, Krishnamoorthy P, Ro R, Anastasius M, Zhao W, Buckley S, et al. Abnormal left ventricular global longitudinal strain by speckle tracking echocardiography in COVID-19 patients. Future Cardiol 2021 Jul;17(4):655-61.
16. Clark DE, Aggarwal SK, Phillips NJ, Soslow JH, Dendy JM, Hughes SG. Cardiac Magnetic Resonance in the Evaluation of COVID-19. Card Fail Rev 2022 Mar 23;8:e09.
17. Gibbons RJ, Balady GJ, Bricker JT, Chaitman BR, Fletcher GF, Froelicher VF, et al.; American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Committee to Update the 1997 Exercise Testing Guidelines. ACC/AHA 2002 guideline update for exercise testing: summary article. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1997 Exercise Testing Guidelines). J Am Coll Cardiol 2002 Oct 16;40(8):1531-40.
18. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2015 Jan;28(1):1-39.e14.
19. Ghafoor N, Islam MM, Shakil SN, Deepa KP, Dutta AK, Medha KA, et al. Observation of Myocardial Involvement in Patients Recovered From COVID-19 by Using Cardiac

Magnetic Resonance Imaging, In a Tertiary Care Hospital, Bangladesh. Mymensingh Med J 2022 Oct; 31(4): 1108-14.

20. Moody WE, Liu B, Mahmoud-Elsayed HM, Senior J, Lalla SS, Khan-Kheil AM, et al. Persisting adverse ventricular remodeling in COVID-19 survivors: A longitudinal echocardiographic study. J Am Soc Echocardiogr 2021 May;34(5):562-6.

21. Özer S, Candan L, Özyýldýz AG, Turan OE. Evaluation of left ventricular global functions with speckle tracking echocardiography in patients recovered from COVID-19. Int J Cardiovasc Imaging 2021 Jul;37(7):2227-33.

22. Bianqui CT, Cueto A, Soto R, Urbano P, Amenabar A, Carnevalini M, et al. Cardiovascular findings in echocardiography in post-Covid-19 mild infection population in Argentina. Available at: https://www.authorea.com/users/408004/articles/518156-cardiovascular-findings-in-echocardiography-in-post-covid-19-mild-infection-population-in-argentina, accessed 30 June 2022.

23. Ma³ek £A, Marczak M, Mi³osz-Wieczorek B, Konopka M, Braksator W, Drygas W, et al. Cardiac involvement in consecutive elite athletes recovered from Covid-19: A magnetic resonance study. J Magn Reson Imaging 2021 Jun;53(6):1723-9.

24. Daniels CJ, Rajpal S, Greenshields JT, Rosenthal GL, Chung EH, Terrin M, et al.; Big Ten COVID-19 Cardiac Registry Investigators. Prevalence of clinical and subclinical myocarditis in competitive athletes with recent SARS-CoV-2 infection: Results from the Big Ten COVID-19 Cardiac Registry. JAMA Cardiol 2021 Sep 1;6(9): 1078-87.

25. Joy G, Artico J, Kurdi H, Seraphim A, Lau C, Thornton GD, et al. COVIDsortium Investigators. Prospective case-control study of cardiovascular abnormalities 6 months following mild COVID-19 in healthcare workers. JACC Cardiovasc Imaging 2021 Nov;14(11):2155-66.