Cardiology in the Young

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Echocardiographic findings in non-hospitalised children and adolescents following acute COVID-19

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Original Article

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Abstract

Background: Although COVID-19 is known to have cardiac effects in children, seen primarily in severe disease, more information is needed about the cardiac effects following COVID-19 in non-hospitalised children and adolescents during recovery. This study aims to compare echocardiographic markers of cardiac size and function of children following acute COVID-19 with those of healthy controls. *Methods:* This single-centre retrospective case–control study compared 71 cases seen in cardiology clinic following acute COVID-19 with 33 healthy controls. Apical left ventricle, apical right ventricle, and parasternal short axis at the level of the papillary muscles were analysed to measure ventricular size and systolic function. Strain was analysed on vendor-independent software. Statistical analysis was performed using t-test, chi-square, Wilcoxon rank sum, and regression modelling as appropriate ($p < 0.05$ significant). *Results:* Compared to controls, COVID-19 cases had slightly higher left ventricular volumes and lower left ventricular ejection fraction and right ventricular fractional area change that remained within normal range. There were no differences in right or left ventricular longitudinal strain between the two groups. Neither initial severity nor persistence of symptoms after diagnosis predicted these differences. *Conclusions:* Echocardiographic findings in children and adolescents 6 weeks to 3 months following acute COVID-19 not requiring hospitalisation were overall reassuring. Compared to healthy controls, the COVID-19 group demonstrated mildly larger left ventricular size and lower conventional measures of biventricular systolic function that remained within the normal range, with no differences in biventricular longitudinal strain. Future studies focusing on longitudinal echocardiographic assessment of patients following acute COVID-19 are needed to better understand these subtle differences in ventricular size and function.

COVID-19, caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has had a significant impact on health in both adults and children.[1–4] Up to 80% of children with severe multisystem inflammatory syndrome have evidence of cardiac involvement.[5–9] Although cardiac effects are known to be common in severe acute COVID-19 leading to hospitalisation,[1] less is known about these effects in children that do not require hospitalisation.

Initial studies from the fall of 2020 in cohorts of asymptomatic college-aged athletes with evidence of prior COVID-19 demonstrated cardiac MRI findings suggesting evidence of myocarditis.[2–9] More recent studies have suggested a low prevalence of myocardial involvement after mildly symptomatic infection in younger children,[10] and diagnostic evaluations in young adult athletes have largely been reassuring despite a high burden of cardiopulmonary symptoms following COVID-19.[11] Many children with persistent symptoms after COVID-19 are screened for myocardial involvement with electrocardiograms and echocardiograms. Additional diagnostic testing to rule out inflammatory heart disease, such as cardiac MRI or cardiopulmonary exercise testing, is considered based on the results of the echocardiogram or electrocardiogram.[12] However, the echocardiographic effects of mild acute COVID-19 during the recovery phase are less well described, particularly in children. The few published studies that have attempted to describe echocardiographic abnormalities following non-hospitalised COVID-19 in children have reported conflicting findings, although have been reassuring apart from the presence of subtle differences in cardiac size and function.[6,13,14] The burden and implications of echocardiographic changes in older children and adolescents after COVID-19 remain unclear. This study aims to compare cardiac size and function following acute non-hospitalised COVID-19 in previously healthy older children and adolescents referred to outpatient cardiology clinic for cardiac evaluation with those of healthy controls by using both conventional echocardiographic parameters and speckle-tracking echocardiography.

We hypothesise that following acute COVID-19 disease, patients will demonstrate differences in cardiac size and function compared to healthy controls.

Materials and methods

Study design

This retrospective single-centre study included patients less than 21 years of age seen in outpatient cardiology clinic between February 2020 and February 2021. Inclusion criteria were those with a preceding confirmed diagnosis of COVID-19 (positive COVID-19 polymerase chain reaction or antigen test) who had an echocardiogram performed as part of their diagnostic evaluation. Exclusion criteria were history of CHD, known pre-existing dysrhythmia, presumed COVID-19 without confirmatory testing, multisystem inflammatory syndrome in children, or hospitalisation following acute COVID-19. Referral indications varied, but included sports clearance, abnormal electrocardiogram findings, or cardiac symptomatology (chest pain, dyspnoea on exertion, fatigue, palpitations, syncope, or dizziness). Timing of referral was variable after COVID-19 diagnosis. Age-matched (within two years) and gender-matched controls with no known history or evidence of COVID-19 were retrospectively identified from patients referred to cardiology clinic between 2015 and 2021 for indications other than COVID-19 (referral reasons included family history, chest pain, abnormal EKG, syncope, palpitations, dizziness, or murmur) who had an echocardiogram performed as part of the visit and were subsequently determined to have no evidence of cardiac disease. The study was approved by the Children's Mercy Kansas City Institutional Review Board. Informed consent was waived due to the retrospective nature of the project.

COVID-19 data

Retrospective chart review was performed to collect demographic and clinical data. Patient demographics included gender, age, race, and body mass index. Clinical data included COVID-19 testing status, timing of evaluation after COVID-19 diagnosis, exercise stress test and MRI results when available, echocardiogram images and results, electrocardiograms, presence/absence and duration of acute COVID-19 symptoms, and any associated cardiovascular symptoms. Any additional testing performed, including cardiac MRI and exercise stress testing, was ordered at the discretion of the evaluating cardiologist at the time of the clinic visit. Acute COVID-19 symptoms were defined as fever, myalgia, chills, lethargy, anosmia, nasal congestion, and cough. Per American Academy of Paediatrics guidelines, patients were categorised as having either asymptomatic infection, mild infection (less than four days of symptoms), or moderate infection (at least four days of symptoms without hospitalisation or diagnosis of multisystem inflammatory syndrome in children). Long COVID-19-associated cardiovascular symptoms were defined as chest pain, palpitations, fatigue, shortness of breath at rest or exercise, dizziness, or syncope following acute COVID-19 illness persisting more than 60 days.[15,16]

Electrocardiograms

All subjects had a standard 12-lead electrocardiogram obtained after their COVID-19 diagnosis, either at the cardiology visit or at a prior appointment with their primary care physician or urgent care. Serial electrocardiograms preceding COVID-19 diagnosis or obtained after the initial electrocardiogram were analysed when available. All the electrocardiograms were reviewed by a board-certified paediatric electrophysiologist in either electronic (MUSE editor, GE Medical System Information Technologies, Inc.) or paper form. Rhythm assessment, atrial enlargement, ventricular hypertrophy, PR/QRS/QT interval duration, and ST-T wave abnormalities were reviewed. Abnormal electrocardiograms were defined as described in a previously published paper in an overlapping cohort, including abnormal rhythm, QTc>465ms, abnormal intervals for age, and repolarisation abnormalities.[17]

Echocardiography

Standard two-dimensional and Doppler transthoracic echocardiographic variables were either obtained from the written clinical report when available or measured directly from available images if not included in the report. Only the initial echocardiogram obtained at the time of the clinic visit was analysed. All echocardiograms were performed on Philips Epiq 7 or GE Vivid E95 cardiac ultrasound machines by American Registry for Diagnostic Medical Sonography–certified sonographers and reviewed by the ordering paediatric cardiologist. Standard two-dimensional guided M-mode, tissue Doppler, spectral Doppler, and colour flow mapping were performed to assess indices of systolic and diastolic function as per American Society of Echocardiography guidelines.[18] Z-scores were derived from the Boston method based on body surface area.[19] Left ventricular and right ventricular longitudinal strain were determined using 2D speckle-tracking echocardiography.[20] Left ventricular apical 4-chamber views were analysed manually using Tomtec software (CPA Version 2.31) to calculate single plane longitudinal strain. Interpretation of strain imaging was performed by three independent reviewers (NM, DA, DF) who were blinded to study data, including COVID-19 status.

Statistical analysis

Characteristics of the COVID-19 group and controls were summarised as percentages, means ± standard deviation, or as median, minimum, and maximum, as appropriate depending on the normalcy of the data. Categorical data were compared using chi-square test or Fisher's exact test for smaller sample groups ($n < 5$). The independent *t*-test and Wilcoxon rank sum test were used to compare normally and non-normally distributed continuous variables to examine differences between the COVID-19 group and the control group, with p-value < 0.05 defined as significant. Univariate linear regression models were used to determine the continuous relationship between echocardiographic parameters and clinical characteristics, including all variables with p-values < 0.2. Age, sex, race, body mass index, and blood pressure were included in the modelling regardless of their p-value, controlling for demographical differences between the COVID-19 group and the cases. All statistical analyses were performed using SPSS 24 (IBM SPSS Statistics for Windows, Version 24.0. IBM Corp., Armonk, NY, USA, 2016), with p-value of ≤ 0.05 considered statistically significant.

Results

Demographics

A total of 85 patients between February 2020 and February 2021 were referred to cardiology clinic following confirmed COVID-19.

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Table 1. Demographic and clinical characteristics of children and adolescents after COVID-19 and healthy controls.

	COVID-19 (n = 71)	Controls (n = 33)	p-value
Age	14.96 ± 2.8	13.7 ± 3.26	0.04*
Male sex	40 (56.3%)	17 (51.5%)	0.6
Race			
Caucasian Non-Caucasian	56 (78.9%) 15 (21.1%)	17 (51.5%) 16 (48.5%)	0.005*
BMI (≥95%)	8 (11.3%) (8)	12 (36.4%)	0.003*
BP percentile			
Normal Elevated Stage I Stage II	38 (53.5%) 23 (32.4%) 9 (12.7%) 1 (1.4%)	23 (69.7%) 4 (12.12%) 1 (3%) 5 (15.2%)	0.6
Persistent symptoms>60 days	13 (18.3%)		
COVID-19 Severity			
Asymptomatic Mild Moderate Cannot determine	1 (1.4%) 32 (45.1%) 36 (50.7%) 2 (2.8%)		
Time since onset of symptoms to cardiology visit (days)	44.42 ± 37.73		
Time since onset of symptoms to echocardiogram (days)	44.21 ± 37.93		
Abnormal EKG	14 (21.1%)		

BMI = body mass index; BP = blood pressure; EKG = electrocardiogram.

Table 2. Baseline left and right heart echocardiographic parameters between children and adolescents after COVID-19 and healthy controls.

	COVID-19 (n = 71)	Controls (n = 33)	p-value
Left heart parameters:			
IVSd (cm)	0.82 ± 0.14	0.81 ± 0.18	0.71
PWTd (cm)	0.79 ± 0.12	0.76 ± 0.16	0.33
LVMI (g/m2)	127.58 ± 38.9	116.22 ± 46.78	0.2
LVIDd index (cm/m2)	4.75 ± 0.51	4.48 ± 0.55	0.02*
LVIDd Z score	−0.26 ± 0.94	−0.62 ± 0.98	0.09
LVEDVi (ml/m2)	144.78 ± 42.76	119.88 ± 44.44	0.01*
LVEDV Z score	0.66 ± 1.2	−0.01 ± 1.44	0.02*
LVESVi (ml/m2)	57.02 ± 17.85	45.82 ± 19.76	0.01*
LVESV Z score	0.48 ± 0.87	−0.2 ± 1.46	0.01*
LVEF %	60.57 ± 4.13	62.47 ± 4.68	0.04*
LV longitudinal strain	18.84 ± 2.49	19.38 ± 3.47	0.38
LV strain rate	0.91 ± 0.14	0.95 ± 0.24	0.36
Right heart parameters:			
RV Area Diastole	23.17 ± 5.84	21.4 ± 4.96	0.16
RV Area Systole	14.16 ± 4	12.5 ± 3.67	0.06
Fractional area change (%)	39.05 ± 4.63 (60)	42.16 ± 7.1	0.02*
Eccentricity Index	1.03 ± 0.08	1 ± 0.01	0.03*
RV longitudinal strain	21.84 ± 3	21.93 ± 2.94	0.90

CI = confidence interval; IVSd = interventricular septal thickness in diastole; PWTd = left ventricular posterior wall thickness in diastole; LVMI = left ventricular mass index; LVIDd = left ventricular internal dimension in diastole; LVEDVi = left ventricular end-diastolic volume indexed; LVESVi = left ventricular end-systolic volume indexed; LVEF = left ventricular ejection fraction; LV = left ventricle; RV = right ventricle.

71 (84%) patients had echocardiograms performed as part of the clinic visit and met inclusion criteria. 33 healthy controls were analysed. Patient characteristics of the two groups are shown in Table 1. Controls were slightly younger, more likely to be non-white, obese, and with a higher incidence of Stage II elevated blood pressure. Echocardiograms were obtained an average of 6 weeks (range 1 week–3 months) after COVID-19. Only one patient had more than one echocardiogram performed as part of their evaluation.

Echocardiographic data

Table 2 demonstrates echocardiographic data for the right and left heart between the two groups. Volumetric assessment of ventricles was performed in all COVID-19 patients and in 97% of the controls, as left ventricular end-diastolic volume and left ventricular end-systolic volume could not be accurately measured in one control due to suboptimal imaging quality. Similarly, left ventricular strain analysis was able to be performed in 97% of the COVID-19 group and controls; right ventricular strain analysis was performed in 86% of the COVID-19 group and 85% of the control group. Compared to the control group, the COVID-19 group had statistically significantly higher indexed ventricular end-diastolic volume and left ventricular end-systolic volume with associated z-scores, and slightly lower left ventricular ejection fraction(Fig 1). Although left ventricular longitudinal strain and strain rate were slightly lower in the COVID-19 group compared to controls, this difference did not reach statistical significance. For right heart parameters, our COVID-19 group showed a slight but statistically significant reduction in right ventricular fractional area change (Fig 1), with no significant difference in right ventricular longitudinal strain between the two groups.

Relationship between clinical and electrocardiographic characteristics with echocardiographic findings

Overall, there were no statistically significant associations between concerning clinical characteristics, and electrocardiographic and echocardiographic findings, as shown in Table 3. The timing of the echocardiograms after initial illness showed a weak negative correlation with cardiac volumes; −0.25 for left ventricular end-systolic volume and −0.23 for left ventricular end-diastolic volume.

Table 4 summarises the clinical characteristics and electrocardiographic findings in patients who had abnormal echocardiograms. 10/71 (14%) had abnormalities in right heart function, either right ventricular longitudinal strain or right ventricular fractional area change. 5/71 (7%) had abnormalities in left heart function, either depressed LV ejection fraction or LV longitudinal strain. Only two of these patients had electrocardiographic anomalies, and two had serial echocardiograms which normalised within 1–3 months. One of the patients with initial low LVEF had a cardiac MRI and was subsequently diagnosed with COVID-19

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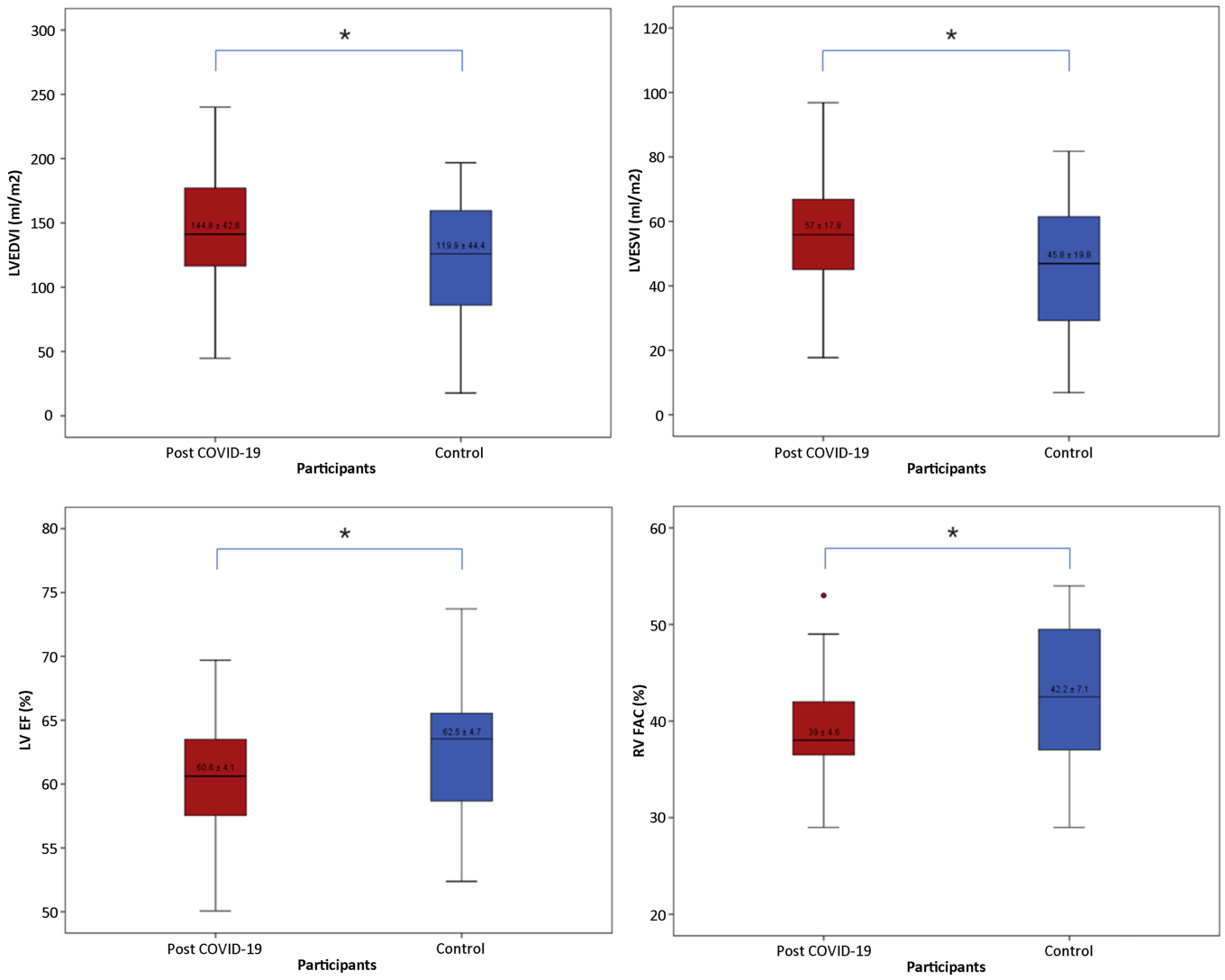

Figure 1. Comparison of left and right heart parameters in children and adolescents following COVID-19 and healthy controls. LVEDVi = left ventricular end-diastolic volume indexed; LVESVi = left ventricular end-systolic volume indexed; LV EF = left ventricular ejection fraction; RV FAC = right ventricular fractional area change; * = p-value < 0.05.

myocarditis. No additional cardiac MRI testing was performed in the cohort.

Discussion

This single-centre retrospective study was conducted to compare echocardiographic findings in previously healthy older children and adolescents recovering from acute COVID-19 not requiring hospitalisation with those of healthy controls. We found that overall, clinically significant echocardiographic abnormalities following acute COVID-19 were rare, suggesting a low burden of acute cardiac inflammation in the recovery phase. In comparison to healthy controls, there were slightly increased biventricular volumes and trivially decreased biventricular function in the COVID-19 cohort as assessed by conventional two-dimensional measures, although all values remained within the normal range. We detected no differences in right or left longitudinal strain or strain rate between the two groups. Additionally, clinical characteristics such as severity of initial illness, persistence of symptoms past the initial illness, and abnormal electrocardiograms did not predict these echocardiographic changes.

Echocardiographic changes after COVID-19

When evaluating traditional measures of cardiac size and function, we found increased left ventricular volumes compared to controls, as measured by left ventricular end-diastolic and systolic volume, although these remained within the normal range. In addition, both left and right ventricular systolic function were reduced compared to controls as measured by left ventricular ejection fraction and right ventricular fractional area change, although they remained within the normal range.

We found no differences between the groups for either left or right longitudinal strain or strain rate. Strain analysis is thought to be less load-dependent than two-dimensional volume/area-based measures of systolic function, and this suggests that children and adolescents following COVID-19 have no differences in cardiac function compared to healthy controls. It is possible that the initial illness resulted in ventricular dilation as a response to the acute inflammatory response that explains the small differences in

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Table 3. Univariate linear regression to assess the relationship of clinical characteristics to left and right heart functional parameters.

	LVEF	LVEDVi	LVESVi	LVLS	RVFAC	RVLS
Long COVID>60days	0.5 (−0.8)	0.07 (−24.3)	0.1 (−8.8)	0.96 (0.05)	0.3 (0.02)	0.2 (1.4)
Abnormal EKG*	0.3 (−1.4)	0.9 (−1.8)	0.8 (1)	0.8 (0.2)	0.5 (0.01)	0.8 (0.2)
Moderate COVID severity	0.97 (0.04)	0.2 (−12.5)	0.2 (−5.2)	0.4 (−0.5)	0.2 (0.02)	0.7 (0.3)

EKG = electrocardiogram; LVEDVi = left ventricular end-diastolic volume indexed; LVEF = left ventricular ejection fraction; LVESVi = left ventricular end-systolic volume indexed; LVLS = left ventricular longitudinal strain; RVFAC = right ventricular fractional area change; RVLS = right ventricular longitudinal strain. Backward stepwise regression analysis to estimate p-value with regression coefficient in parenthesis.

Table 4. Clinical, electrocardiographic, and echocardiographic abnormalities in children and adolescents following COVID-19.

Age (years)	Sex	COVID-19 severity	Persistent cardiac symptoms	EKG abnormalities	Echo abnormalities [LV EF%; LVLS; LV LS Strain rate; RVFAC; RVLS]	Resolution of echo abnormalities
12.6	F	Moderate	CP, DOE, F	None	LV LS–13.7%	No follow-up echo
18	M	Mild	None	None	LVEF 50%	Normalisation of LVEF on follow-up echocardiogram 3 months later
17.5	M	Moderate	CP, DOE	None	LVEF 50%	Normalisation of LVEF on follow-up echocardiogram 1 month later
14.5	M	Moderate	None	Diffuse ST-T wave abnormality mimicking RBBB with ST doming in anterior precordial leads	LVEF 51%	Patient lost to follow-up
14.9	M	Mild	CP, P	None	LV EF 54%	No follow-up echo
15.2	M	Mild	CP, DOE, S	None	RV LS –17.0%	No follow-up echo
12.3	M	Moderate	F	None	RV LS –17.7%, RV FAC 30%	No follow-up echo
17.9	M	Mild	None	None	RV LS –17.7%	No follow-up echo
15.1	M	Mild	None	None	RV FAC 29%	No follow-up echo
14.6	M	Mild	CP, DOE, *F*, P	None	RV FAC 33%	No follow-up echo
14.4	M	Mild	None	None	RV FAC 33%	No follow-up echo
16.7	M	Mild	CP, DOE, *F*, P, S	PR prolongation	RV FAC 32%	No follow-up echo
17	F	Mild	CP	None	RV FAC 34%	No follow-up echo
17.9	F	Moderate	None	None	RV FAC 34%	No follow-up echo
17.3	M	Mild	None	None	RV FAC 34%	No follow-up echo

CP = chest pain; DOE = dyspnoea on exertion; EKG = electrocardiogram; F = fatigue; P = palpitations; S = syncope/dizziness; LVEF = left ventricular ejection fraction; LVLS = left ventricular longitudinal strain; RVFAC = right ventricular fractional area change; RVLS = right ventricular longitudinal strain.

ventricular volumes between the COVID-19 group and controls and may have contributed to the small measured differences in cardiac function as measured by left ventricular ejection fraction and right ventricular fractional area change. As our echocardiograms were obtained an average of 6 weeks to 3 months after initial illness, any initial alterations in cardiac function as measured by strain analysis may have resolved by the time of evaluation. As serial and baseline evaluations were not performed, it is unknown whether there was a change from baseline prior to COVID-19. We did see a weak negative correlation between time to echo after initial illness and left ventricular volumes, which may be explained by the time course of the ventricular dilation following the initial inflammation.

Overall, our findings shared similarities to those in limited previous literature that described echocardiograms in children following non-hospitalised acute COVID-19.[11,13] These studies showed that clinically significant echocardiographic changes were not present but that subtle subclinical differences in cardiac size and function may exist compared to healthy controls.[11,13] Examining a younger cohort of patients (mean age 7.5 years), Sirico et al.[13] found that, compared to healthy controls, patients had a lower left ventricular ejection fraction (62.4% versus 65.2%). However, they found no difference in cardiac size or right ventricular function as measured by tricuspid annular plane systolic excursion. In contrast, we found higher biventricular volumes and slightly lower right ventricular function as measured by right ventricular fractional area change in this group of COVID-19 patients compared to controls. We chose right ventricular fractional area change over tricuspid annular plane systolic excursion as a marker of right ventricular systolic function in our cohort to better approximate right ventricular function.[21] This may be a more sensitive two-dimensional measure for evaluating

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right ventricular function in this population. In line with our findings, Sirico et al did not find a difference in left ventricular longitudinal strain after COVID-19, although they did report a reduction in regional peak systolic strain in at least two myocardial segments among COVID-19 cases compared to controls.[13] Regional peak systolic strain was not assessed in our study due to known pitfalls in reproducibility of this method.[22] Compared to Sirico et al's cohort, our cohort was significantly older (mean age 15 years versus 7.5 years), which better represents the age that is most likely to participate in competitive sports and thus be evaluated by general practitioners or cardiologists for athletic clearance following COVID-19.

Clinical characteristics and echocardiographic changes

We found no association between clinical symptoms and echocardiographic differences. This result is in line with previous literature that suggests that persistent symptoms, time from diagnosis, and severity of initial symptoms do not predict cardiac involvement[11,13,23] and indicates that the presence of cardiopulmonary symptoms including chest pain, shortness of breath, and exercise intolerance in nearly all patients is unlikely to be secondary to acute cardiac inflammation or ongoing cardiac derangements.

Echocardiographic and electrocardiographic abnormalities

Electrocardiographic abnormalities, including changes in PR interval, ventricular voltages, ST-T-wave pattern and QTc interval, have been noted to occur with COVID-19 secondary to myopericarditis, hypoxia, or inflammatory changes. However, as demonstrated in our previous study of the same cohort, there were few with concerning findings and nearly all these changes resolve over time without evidence of significant myocarditis.[17] In addition, we did not find any significant associations between electrocardiographic abnormalities and echocardiographic differences.

Limitations

There are multiple limitations to this study, including a retrospective single-centre study design and variable timing of the echocardiograms post-COVID-19. Serial echocardiograms were not performed in most patients, making it difficult to draw conclusions regarding the timing of development and resolution of the echocardiographic changes. As conventional echocardiographic measurements were performed and reviewed by multiple ordering paediatric cardiologists, errors due to inter-observer variability may exist. The small sample size of our cohort likely contributed to decreased statistical significance of the findings, and all differences between groups may be within the range of variability of the echocardiographic measurements. As this was a retrospective study, no gold standard data in the form of cardiac MRIs were available for confirmation of our functional and volumetric results in most patients and thus we were also unable to assess for the presence of myocarditis by MRI criteria. Thus, it is possible that the presence of cardiac abnormalities as detected by cardiac MRI was underdiagnosed in this cohort by echocardiographic screening only. In addition, because the cases and controls were not matched by body mass index, race, and presence of hypertension, this may have partially contributed to the higher percentage of echocardiographic abnormalities seen in the control group and thus may have affected our results.

Conclusions

Echocardiographic findings in this cohort of children and adolescents 6 weeks to 3 months following acute COVID-19 not requiring hospitalisation were overall reassuring. Compared to healthy controls, the COVID-19 group demonstrated mildly larger left ventricular size and lower conventional measures of biventricular systolic function that remained within the normal range. There were no differences in biventricular longitudinal strain between the two groups. Future studies focusing on longitudinal echocardiographic assessment of children and adolescent following acute COVID-19 are needed to better understand these subtle differences in ventricular size and function and establish whether these are associated with clinically significant outcomes.

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Competing interests. None.

Ethical standard. No human or animal experimentation was utilised in this retrospective study

References

1. Cantarutti N, Battista V, Adorisio R, et al. Cardiac manifestations in children with SARS-COV-2 infection: 1-year pediatric multicenter experience. Children 2021; 8: 717. DOI: 10.3390/CHILDREN8080717.
2. Baggish A, Drezner JA, Kim J, Martinez M, Prutkin JM. Resurgence of sport in the wake of COVID-19: cardiac considerations in competitive athletes. Br J Sports Med 2020; 54: 1130–1131. DOI: 10.1136/bjsports-2020-102516.
3. Kim JH, Levine BD, Phelan D, et al. Coronavirus disease 2019 and the athletic heart: emerging perspectives on pathology, risks, and return to play. JAMA Cardiol 2020; 6: 219–227. DOI: 10.1001/jamacardio.2020.5890.
4. Phelan D, Kim JH, Elliott MD, et al. Screening of potential cardiac involvement in competitive athletes recovering from COVID-19: an expert consensus statement. JACC Cardiovasc Imaging 2020; 13: 2635–2652. DOI: 10.1016/j.jcmg.2020.10.005.
5. Martinez MW, Tucker AM, Bloom OJ, et al. Prevalence of inflammatory heart disease among professional athletes with prior COVID-19 infection who received systematic return-to-play cardiac screening. JAMA Cardiol 2021; 6: 745–752. DOI: 10.1001/JAMACARDIO.2021.0565.
6. Moulson N, Petek BJ, Drezner JA, et al. SARS-CoV-2 cardiac involvement in young competitive athletes. Circulation 2021; 144: 256–266. DOI: 10.1161/CIRCULATIONAHA.121.054824.
7. Cavigli L, Frascaro F, Turchini F, et al. A prospective study on the consequences of SARS-CoV-2 infection on the heart of young adult competitive athletes: implications for a safe return-to-play. Int J Cardiol 2021; 336: 130–136. DOI: 10.1016/J.IJCARD.2021.05.042.
8. Chowdhury D, Fremed MA, Dean P, et al. Return to activity after SARS-coV-2 infection: cardiac clearance for children and adolescents. Sports Health 2021; XX: 1–6. DOI: 10.1177/19417381211039746.
9. Brito D, Meester S, Yanamala N, et al. High prevalence of pericardial involvement in college student athletes recovering from COVID-19. JACC Cardiovasc Imaging. 2020; 14: 541–555. DOI: 10.1016/j.jcmg.2020.10.023.
10. Seidel F, Kuehne T, Kelle S, et al. Cardiovascular magnetic resonance findings in non-hospitalized paediatric patients after recovery from COVID-19. ESC Heart Fail 2021; 8: 5583–5588. DOI: 10.1002/EHF2.13678.
11. Moulson N, Gustus SK, Scirica C, et al. Diagnostic evaluation and cardiopulmonary exercise test findings in young athletes with persistent symptoms following COVID-19. Br J Sports Med 2022; 56: 927–932. DOI: 10.1136/BJSPORTS-2021-105157.

12. Committee W, Gluckman TJ, Bhave NM, et al. 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; 79: 1717–1756. DOI: 10.1016/J.JACC.2022.02.003.
13. Sirico D, di Chiara C, Costenaro P, et al. Left ventricular longitudinal strain alterations in asymptomatic or mildly symptomatic paediatric patients with SARS-coV-2 infection. Eur Heart J Cardiovasc Imaging 2021; 23: 1083–1089. DOI: 10.1093/EHJCI/JEAB127.
14. Erol N, Alpinar A, Erol C, Sari E, Alkan K. Intriguing new faces of covid-19: persisting clinical symptoms and cardiac effects in children. Cardiol Young 2021; 32: 1–1091. DOI: 10.1017/S1047951121003693.
15. v. Raveendran A, Jayadevan R, Sashidharan S.Long COVID. An overview. Diabetes Metab Syndr 2021; 15: 869–875. DOI: 10.1016/J.DSX.2021.04.007.
16. Fernández-de-las-Peñas C. Long COVID: current definition. Infection 2022; 50: 285–286. DOI: 10.1007/S15010-021-01696-5.
17. Heching HJ, Goyal A, Harvey B, et al. Electrocardiographic changes in non-hospitalised children with COVID-19. Cardiol Young 2022; 32: 1–7. DOI: 10.1017/S1047951121005138.
18. Lai WW, Geva T, Shirali GS, et al. Guidelines and standards for performance of a pediatric echocardiogram: a report from the task force of the pediatric council of the American society of echocardiography. J Am Soc Echocardiogr 2006; 19: 1413–1430. DOI: 10.1016/j.echo.2006.09.001.
19. Colan SD. Normal echocardiographic values for cardiovascular structures. Echocardiograph Pediatr Congenit Heart Dis 2016: 883–901. DOI: 10.1002/9781118742440.app1.
20. Voigt JU, Pedrizzetti G, Lysyansky P, et al. Definitions for a common standard for 2D speckle tracking echocardiography: consensus document of the EACVI/ASE/Industry task force to standardize deformation imaging. Eur Heart J Cardiovasc Imaging 2015; 16: 1–11. DOI: 10.1093/EHJCI/JEU184.
21. Anavekar NS, Gerson D, Skali H, Kwong RY, Kent Yucel E, Solomon SD. Two-dimensional assessment of right ventricular function: an echocardiographic-MRI correlative study. Echocardiography 2007; 24: 452–456. DOI: 10.1111/J.1540-8175.2007.00424.X.
22. Halliday BP, Senior R, Pennell DJ. Assessing left ventricular systolic function: from ejection fraction to strain analysis. Eur Heart J 2021; 42: 789–797. DOI: 10.1093/eurheartj/ehaa587.
23. McEachern WA, Coburn Jr. GW, Jonathan SH, Parra DA. Echocardiographic findings in adolescents presenting for sports clearance following COVID-19. Pediatrics 2022; 149: 326.

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