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RESEARCH LETTER

Athletes with mild COVID-19 illness demonstrate subtle imaging abnormalities without exercise impairment or arrhythmias

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Coronavirus disease-19 (COVID-19) has been associated with cardiac pathology raising the possibility of serious cardiac arrhythmias among athletes returning to competition post infection.[1–3] Several studies have assessed cardiac function and structure in athletes post-COVID-19 infection but have been challenged by selection bias, lack of an appropriate control group and none have included comprehensive imaging, exercise testing, and clinical electrophysiology.[2,4–7]

We recruited every player from a squad of professional basketballers involved in a 'super-spreader' event that lead to a majority being infected with COVID-19 following a single training session in Melbourne, Australia. We compared those athletes who tested positive to COVID-19 by polymerase chain reaction diagnostic testing (16 athletes) to athletes who (i) tested negative and (ii) had no symptoms suggestive of COVID-19 infection ($n = 8$).

Our hypothesis was that electrocardiogram, biochemical, and imaging variants are common in highly trained athletes and would not be more prevalent in athletes recovering from COVID-19 than non-infected teammates.

Comprehensive testing was performed between 10 and 21 days from the time of testing positive to COVID-19. Testing involved clinical assessment, biochemistry (including cardiac troponin I and B-type natriuretic peptide), 12-lead electrocardiography, 24-h Holter monitoring, cardiopulmonary exercise testing, echocardiography, studies of flow mediated dilation, and cardiac magnetic resonance imaging (CMR). Regional myocardial fibrosis was visually identified by delayed gadolinium enhancement (DGE), T1 mapping was analysed using a mid-wall region of the septum and three T2 measurements were analysed; mid septum, global left ventricle, and the highest T2 time at any site. A comprehensive description of all methods is included as supplementary content.

COVID-19 positive athletes had mild (75%) or moderate (25%) symptoms including ageusia/anosmia, fevers, myalgias, and cough persisting for a maximum of 6 days. One COVID-19 positive athlete had a known history of hypertrophic cardiomyopathy and one had been evaluated for ventricular ectopics several years prior.

COVID-19 positive athletes were similar in age, gender, biochemical markers of cardiac damage, exercise capacity, and ventilatory efficiency when compared with COVID-19 negative athletes. Similarly, there were no differences in electrocardiographic measures, arrhythmias, or ventricular pauses, nor any differences in cardiac or vascular imaging markers (see *Table 1*). Of note, one COVID-19 positive athlete with hypertrophic cardiomyopathy had a mildly elevated troponin, infero-lateral T-wave inversion, and minor DGE. One other COVID-19 positive athlete had evidence of DGE comprising a very small patch in the mid lateral wall (athlete 6 in *Figure 1*) and one COVID-19 negative athlete had very small patches of mid septal enhancement (athlete 3 *Figure 1*). In both cases the troponin, T1 and T2 mapping were normal and there were no arrhythmias on a 24-h Holter study. Native, post-contrast and T2 mapping were not different between groups. Pericardial enhancement and/or trivial pericardial effusions were frequently observed and were equally represented between groups (see *Figure 1*). Apart from the athlete

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Table 1 Demographics, biochemistry, electrophysiology, and imaging

	COVID +ve athletes ($n = 16$)	COVID −ve athletes ($n = 8$)	*P*-value
Age (years)	25 ± 6	27 ± 5	0.45
Sex, male, *n* (%)	12 (75)	6 (75)	1.0
Height (m)	1.92 ± 0.14	1.87 ± 0.11	0.35
Weight (kg)	84 ± 13	84 ± 16	0.99
Body mass index (kg/m^2)	22.76 ± 2.40	23.80 ± 2.05	0.29
Systolic blood pressure (mmHg)	124 ± 12	119 ± 8	0.17
Diastolic blood pressure (mmHg)	64 ± 8	59 ± 5	0.070
Biochemistry			
Haemoglobin (g/L)	145 ± 12	146 ± 12	0.89
B-type natriuretic peptide (pg/mL)	18 ± 23	8 ± 4	0.089
Cardiac Troponin I (ng/L)	25 ± 54	21 ± 51	0.85
Elevated troponin (>14ng/L)	3	1	0.70
Cardiopulmonary exercise test			
Peak exercise heart rate (b.p.m.)	185 ± 9	187 ± 15	0.83
Peak respiratory exchange ratio	1.3 ± 0.9	1.3 ± 0.4	0.18
Peak VO_2 (L/min)	3.60 ± 0.71	3.91 ± 0.92	0.42
Peak VO_2 (mL/kg/min)	41.5 ± 5.0	47.2 ± 10.6	0.18
Ventilatory efficiency (VE/VCO_2)	22.6 ± 2.9	22.1 ± 3.2	0.72
Electrocardiogram			
Heart rate (b.p.m.)	56 ± 9	54 ± 11	0.69
QTc-interval (ms)	386 ± 61	398 ± 16	0.46
Abnormal T-wave inversion	1	0	0.67
Left bundle branch block	0	0	1.0
24-h Holter monitor			
Pauses >2 s	2.3 ± 6.8	2.9 ± 5.5	0.81
Ventricular ectopics over 24 h	92 ± 344	20 ± 55	0.43
Non-sustained VT	0	0	1.0
Number supraventricular beats	11 ± 14	31 ± 37	0.18
Number of AT or AF episodes	0	0	1.0
Cardiac magnetic resonance imaging			
LVEDV/BSA (mL/m^2)	100 ± 16	110 ± 11	0.10
LVESV/BSA (mL/m^2)	43 ± 11	45 ± 7	0.71
LVEF (%)	58 ± 3	59 ± 5	0.66
LV mass/BSA (g/m^2)	76 ± 9	83 ± 5	0.020
RVEDV/BSA (mL/m^2)	111 ± 15	124 ± 10	0.27
RVEF (%)	50 ± 3	51 ± 7	0.72
LAVI (mL/m^2)	35 ± 11	35 ± 7	0.95
RA area (cm^2)	24 ± 5	26 ± 4	0.51
Native T1 (ShMOLLI, ms)	1134 ± 38	1142 ± 32	0.59
ECV (%)	25 ± 3	24 ± 3	0.96
T2 (ms)—septum	37.6 ± 1.8	37.5 ± 2.0	0.87
T2 (ms)—mid SAX	38.4 ± 1.7	38.5 ± 1.9	0.92
T2 (ms)—max segment	42.6 ± 4.7	42.2 ± 2.7	0.79
Pericardial enhancement ± effusion	4	6	0.44
DGE	2	1	0.72
Echocardiography			
Global longitudinal strain (%)	−17.4 ± 2.6	−19.1 ± 2.0	0.094
Mechanical dispersion (ms)	39 ± 5	37 ± 6	0.53
Mitral valve *E/A*	2.2 ± 0.9	2.2 ± 0.8	0.86
TDI *E′* septal (cm/s)	11.7 ± 2.6	12.1 ± 2.3	0.68
E/e′ septal	6 ± 2	6 ± 1	0.93
TR max pressure (mmHg)	19 ± 1	18 ± 2	0.80

Continued

Table 1 *Continued*

	COVID +ve athletes ($n = 16$)	COVID −ve athletes ($n = 8$)	*P*-value
Flow-mediated dilation			
Resting diameter (cm)	0.44 ± 0.6	0.44 ± 0.5	0.82
Peak diameter (cm)	0.47 ± 0.06	0.48 ± 0.05	0.57
Flow-mediated dilation (%)	6.5 ± 2.8	8.4 ± 2.8	0.14
Shear stimulus SR (auc)	13428 ± 7377	9195 ± 5907	0.15

AF, atrial fibrillation; AT, atrial tachycardia; DGE, delayed gadolinium enhancement; ECV, extracellular matrix; EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; LAVI, left atrial volume indexed; LV, left ventricle; LVH, left ventricular hypertrophy; RA, right atrium; RV, right ventricle; STIR, short tau inversion recovery; TDI, tissue Doppler Imaging, DT; TR, tricuspid regurgitation; VCO_2, volume of carbon dioxide produced; VE, ventilation volume; VO_2, volume of oxygen uptake; VT, ventricular tachycardia.

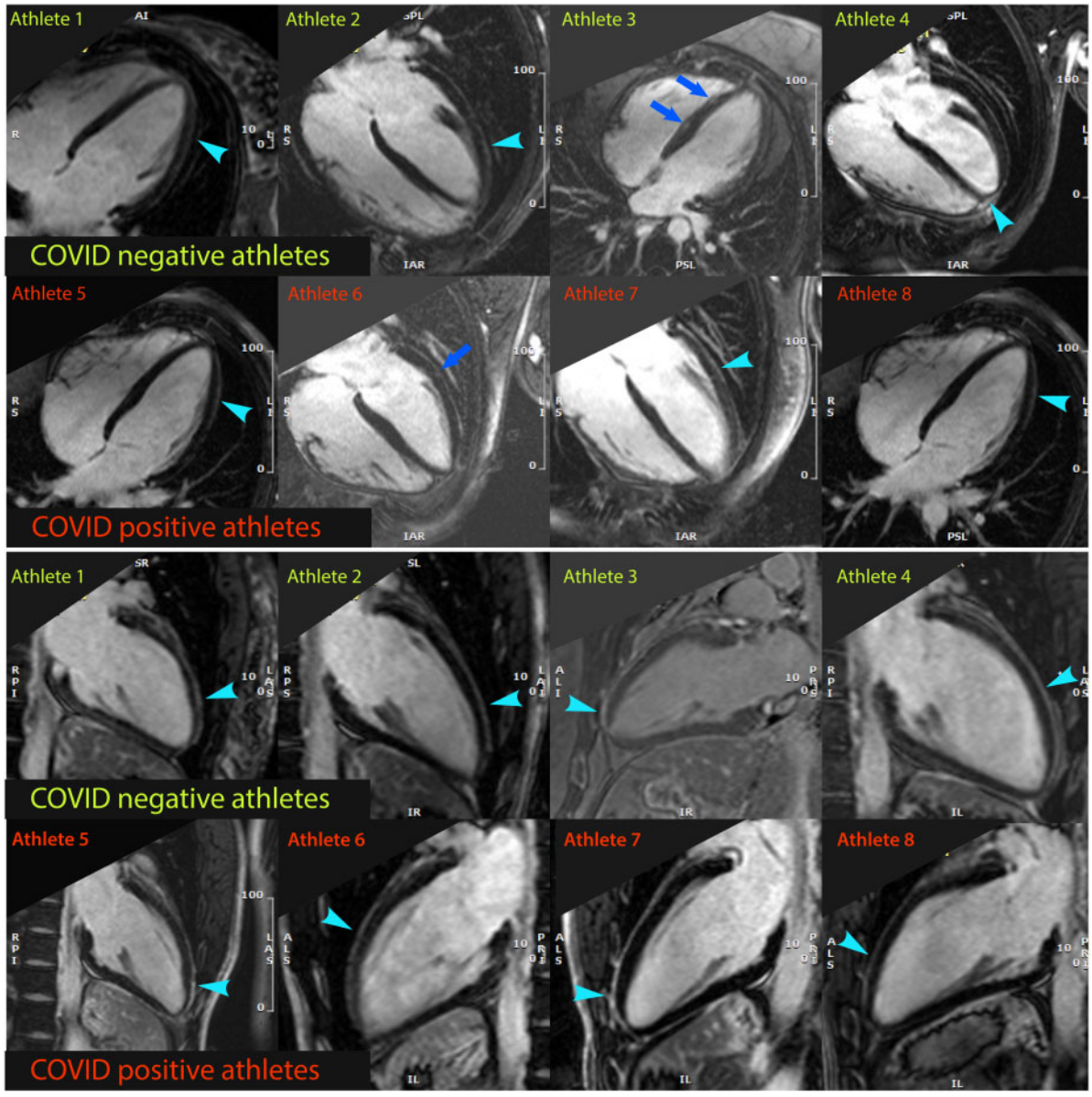

Figure 1 Delayed enhancement of the myocardium and pericardium in athletes with and without prior COVID-19 infection. Long-axis phase sensitive inversion recovery (PSIR) images after gadolinium demonstrating very small patches of myocardial enhancement (royal blue arrows) and pericardial enhancement (light blue arrowheads) in four athletes with and without COVID.

with hypertrophic cardiomyopathy, very minor elevations in troponin were observed in three additional athletes (two COVID-19 positive and one COVID-19 negative, $P = 0.70$). In these athletes, there were no other abnormalities on imaging or exercise testing. At 6-month post-assessment, all athletes continue training and competition without any symptoms or clinical events.

With the goal of assessing the health outcomes in professional athletes returning to sport after COVID-19 infection, this cohort study is unique in the completeness of recruitment and the comprehensiveness of the measures. By enrolling all athletes from a single team at the start of a pandemic outbreak (with extremely low chance of prior infection), it was possible to eliminate selection bias. Prior studies may have been compromised by a tendency to recruit athletes with symptoms sufficient to warrant testing. Furthermore, the comparison with appropriate control athletes provides important qualification of the assumption that all abnormalities in COVID-19 athletes are significant. For example, troponin elevations are considered pathological in most settings, but are prevalent amongst training athletes, including basketballers.[8] Similarly, the finding of ventricular ectopics, increases in CMR mapping values or pericardial abnormalities may have been considered significant if it were not for the comparison with an appropriate control group.

Studies to date have tended to focus on cardiac imaging abnormalities without the context of impact on symptoms, exercise capacity, or electrophysiological abnormalities.[2,4,5,7,9] In comparing athletes with mild COVID-19 illness to a group of uninfected athletes, we observed no differences in imaging parameters and a lack of association with abnormalities on biochemistry, exercise testing or electrophysiology. The findings from these detailed examinations add to the reassurance provided from larger but less comprehensive studies of athletes recovering from COVID-19 illness.[6,7]

In conclusion, abnormalities can be observed in athletes' biomarkers, imaging, and electrophysiology, regardless of COVID-19 infection. These are not associated with persisting symptoms, exercise intolerance, or clinical sequalae.

Conflict of interest: none declared.

Data availability statement

The data underlying this article will be shared on reasonable request to the corresponding author.

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