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Long-term cardiac follow-up of athletes infected with SARS-CoV-2 after resumption of elite-level sports

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ABSTRACT

Objective Longitudinal consequences and potential interactions of COVID-19 and elite-level sports and exercise are unclear. Therefore, we determined the long-term detrimental cardiac effects of the interaction between SARS-CoV-2 infection and the highest level of sports and exercise.

Methods This prospective controlled study included elite athletes from the Evaluation of Lifetime participation in Intensive Top-level sports and Exercise cohort. Athletes infected with SARS-CoV-2were offered structured, additional cardiovascular screenings, including cardiovascular MRI (CMR). We compared ventricular volumes and function, late gadolinium enhancement (LGE) and T1 relaxation times, between infected and non-infected elite athletes, and collected follow-up data on cardiac adverse events, ventricular arrhythmia burden and the cessation of sports careers.

Results We included 259 elite athletes (mean age 26±5 years; 40% women), of whom 123 were infected (9% cardiovascular symptoms) and 136 were controls. We found no differences in function and volumetric CMR parameters. Four infected athletes (3%) demonstrated LGE (one reversible), compared with none of the controls. During the 26.7 (±5.8) months follow-up, all four athletes resumed elite-level sports, without an increase in ventricular arrhythmias or adverse cardiac remodelling. None of the infected athletes reported new cardiac symptoms or events. The majority (n=118; 96%) still participated in elite-level sports; no sports careers were terminated due to SARS-CoV-2.

Conclusions This prospective study demonstrates the safety of resuming elite-level sports after SARS-CoV-2 infection. The medium-term risks associated with SARS-CoV-2 infection and elite-level sports appear low, as the resumption of elite sports did not lead to detrimental cardiac effects or increases in clinical events, even in the four elite athletes with SARS-CoV-2 associated myocardial involvement.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Short-term follow-up and cross-sectional studies have shown a low prevalence (3%–5%) of myocardial abnormalities, including infrequent cardiac adverse events in competitive athletest infected with SARS-CoV-2.

WHAT THIS STUDY ADDS

⇒ This prospective, controlled study demonstrates the safety of resuming elite-level sports in athletes infected with SARS-CoV-2. During 2-year follow-up, elite-level sports did not lead to detrimental cardiac effects or an increase in cardiac events, even in four elite athletes with SARS-CoV-2 associated myocardial involvement. None of the athletes infected with SARS-CoV-2 ended their professional sports careers due to SARS-CoV-2.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Our study provides longer-term reassuring insights regarding cardiac safety in the context of elite-level sports and SARS-CoV-2 and suggests that a liberal but personalised approach may be warranted for athletes infected with SARS-CoV-2 who wish to return to (elite-level) sports.

INTRODUCTION

Active myocarditis is considered an absolute contraindication for participation in sports and exercise.[1] As SARS-CoV-2 has been associated with myocardial involvement in up to 5% of young competitive athletes,[2] the longer-term effects of sports after SARS-CoV-2 infection are of particular relevance and interest. While studies investigating the effects of exercise during active myocarditis in humans are lacking due to ethical reasons, mice models have shown strong deleterious cardiac effects such as adverse ventricular remodelling and an increase in ventricular tachyarrhythmias and mortality.[3] Consequently, athletes diagnosed with or suspected of active myocarditis are advised to refrain from sports for 3–6 months,[1] which can significantly impact an athlete's professional career.

Screening strategies for myocardial involvement following SARS-CoV-2 infection have been subject to intense scrutiny. The majority of these mainly cross-sectional and opportunistic studies have focused on the prevalence of cardiac abnormalities and the implementation of return-to-sports (RTS) screening protocols.[4–11] Prospective studies

Table 1 Population characteristics of athletes infected with SARS-CoV-2 compared with elite athlete controls

	Total n=259		
	Elite athletes infected with SARS-CoV-2	Elite athlete controls	
	n=123	n=136	P value
Age (years), mean (±SD)	25 (6)	27 (7)	**0.014**
Woman, n (%)	41 (33)	61 (45)	0.077
Body surface area (kg/m²), mean (±SD)	2.0 (0.2)	1.9 (0.2)	**0.006**
Ethnicity, n (%)			**<0.001**
Caucasian	98 (79.7)	135 (99.3)	
African/Afro-Caribbean	15 (12.2)	1 (0.7)	
West Asian (Arabic and Middle Eastern)	2 (1.6)	0 (0.0)	
East Asian and South Asian	2 (1.6)	0 (0.0)	
Latin America	6 (4.9)	0 (0.0)	
Athletic discipline, n (%)			**<0.001**
Field hockey	14 (11.4)	27 (19.9)	
Para-cycling (road)	2 (1.6)	8 (5.9)	
Road cycling	12 (9.8)	29 (21.3)	
Rowing	10 (8.1)	9 (6.6)	
Sailing	1 (0.8)	9 (6.6)	
Football	36 (29.3)	9 (6.6)	
Swimming	0 (0.0)	10 (7.4)	
Tennis	6 (4.9)	4 (2.9)	
Track cycling	9 (7.3)	9 (6.6)	
Water polo	11 (8.9)	7 (5.1)	
Miscellaneous*	22 (17.9)	15 (11.0)	
Professional athlete years	10.6 (6.0)	13.4 (6.7)	**0.001**
SARS-CoV-2 symptoms severity, n (%)			
Asymptomatic	24 (19.5)		
Cardiovascular symptoms	11 (8.9)		
No cardiovascular symptoms	88 (71.5)		

*Miscellaneous: artistic gymnastics (n=3), athletics (n=4), basketball (n=2), BMX racing (n=2), boxing (n=2), dressage (n=2), eventing (n=1), judo (n=3), lacrosse (n=1), long track speed skating (n=3), mountain biking (n=3), para-alpine skiing (n=2), para-dressage (n=1), skateboarding (n=2), table tennis (n=1), trampoline jumping (n=1) and wheelchair basketball (n=2).

investigating longer-term outcomes are lacking. Additionally, there is a knowledge gap concerning the potential association between SARS-CoV-2-associated cardiac abnormalities and sports- and exercise, particularly in relation to adverse cardiac remodelling and ventricular arrhythmias. Elite athletes, whose cardiac integrity is a prerequisite for maintaining excellence in performance and who are potentially prone to the most deleterious exercise-induced cardiac effects, constitute a group of special interest when investigating this.

We therefore aimed to prospectively investigate the clinical course of SARS-CoV-2 and RTS in elite athletes over time. Moreover, we determined the prevalence of SARS-CoV-2 associated myocardial sequelae. Lastly, in athletes with SARS-CoV-2 associated cardiac abnormalities who returned to sports, we assessed long-term cardiac remodelling and monitored ventricular arrhythmias.

METHODS

Study design

We performed a prospective analysis of elite athletes included in the Evaluation of Lifetime participation in Intensive Top-level sports and Exercise (ELITE) cohort (NL9328). The methods of ELITE have been described elsewhere.[12] In short, ELITE is a prospective, multicentre longitudinal cohort study that collects standardised periodic cardiovascular screening data of elite athletes in The Netherlands and is ongoing since April 2019. Athletes and the public are actively involved, as the Dutch National Olympic Committee & National Sports Federation is a coinitiator of this study. From March 2020, elite athletes with a confirmed SARS-CoV-2 infection underwent (additional) standardised cardiovascular screening after infection and were simultaneously included in a subcohort of the ELITE study titled 'COVID-19 Myocardial Manifestations in Intensive Top-level sports and exercise' (COMMIT). Athletes were approached by their personal or team/sports physician to participate in the ELITE study and/or COMMIT subcohort; all included athletes provided written informed consent. Athletes with a history of cardiovascular disease prior to SARS-CoV-2 infection were excluded.

Study population

All included elite athletes (ie, Olympians, Paralympians and/or professional athletes) are 16 years or older and exercise more than 10 hours per week with an emphasis on competition and performance.[1] Athletes included before the onset of the COVID-19 pandemic and athletes who self-reported not to have been infected with SARS-CoV-2, with additional confirmation by a negative serology test, were considered as non-infected controls. SARS-CoV-2 infection was confirmed by either a positive PCR or a positive serology test in unvaccinated individuals.

Table 2 Cross-sectional assessment of ECG, laboratory and CMR findings in elite athletes exposed to SARS-CoV-2 versus non-exposed elite athlete controls

	Total n=259		
	Elite athletes infected with SARS-CoV-2	Elite athlete controls	
	n=123	n=136	P value
ECG assessment			
Heart rate (bpm), median (IQR)	57 (51 to 65)	52 (46 to 57)	**<0.001**
ECG conclusion (according to 2017 international athlete ECG criteria), n (%)			0.357
Normal	101 (82)	118 (87)	
Borderline	11 (9)	14 (10)	
Abnormal	6 (5)	3 (2)	
Laboratory assessment, n (%)			
hsTnT >14 ng/L	9 (7)	6 (4)	0.456
NT-proBNP >130 ng/L	2 (2)	3 (2)	1.000
CKMB >5.2 ug/L	14 (11)	21 (15)	0.278
CRP >5 mg/L	2 (2)	4 (3)	0.743
Leucocytes >10.5×10^9	4 (3)	3 (2)	0.888
CMR assessment, mean (±SD)			
Left ventricle			
LVEDV/BSA (mL/m^2)	116 (18)	120 (19)	0.076
LVESV/BSA (mL/m^2)	51 (10)	53 (11)	0.216
LVSV (mL)	128 (27)	128 (27)	0.976
LVEF (%)	56 (5)	56 (5)	0.835
LVM/BSA (g/m^2)	58 (14)	58 (14)	0.848
Ratio of LVM/LVEDV	0.5 (0.1)	0.5 (0.1)	0.254
Right ventricle			
RVEDV/BSA (mL/m^2)	117 (18)	121 (20)	0.178
RVESV/BSA (mL/m^2)	54 (10)	55 (11)	0.377
RVSV (mL)	126 (27)	125 (26)	0.850
RVEF (%)	54 (5)	55 (5)	0.659
Ratio of LVEDV/RVEDV	1.0 (0.1)	1.0 (0.1)	0.348
Native T1 relaxation time* (ms)	962 (22)	961 (24)	0.635
LGE, n (%)			0.496
Non-pathological LGE	119 (97)	136 (100)	
No LGE	77 (63)	81 (60)	
Hinge point LGE	42 (34)	55 (40)	
Pathological LGE	4† (3)	0 (0)	

*Amsterdam UMC normal value of native T1 relaxation time=950–1050 ms.
†Basal to apical lateral myocardial and pericardial LGE of the LV (n=1), focal pericardial/epicardial LGE at the inferolateral wall of the LV with pericardial effusion (PE) at the lateral wall (n=1), basolateral to midventricular posterior epicardial LGE (n=1) and pericardial/epicardial LGE at the inferolateral wall (n=1).
CKMB, creatine phosphokinase-MB; CRM, cardiovascular MRI; hsTnT, high-sensitive troponin T; LGE, late gadolinium enhancement; LV, left ventricle; LVEDV, left ventricle BSA indexed end-diastolic volume; LVEF, left ventricle ejection fraction; LVESV, left ventricle BSA indexed end-systolic volume; LVM, left ventricle BSA indexed wall mass; LVSV, left ventricle stroke volume; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; RVEDV, right ventricle BSA indexed end-diastolic volume; RVEF, right ventricle ejection fraction; RVESVi, right ventricle BSA indexed end-systolic volume; RVSV, right ventricle stroke volume.

Data collection

We collected cardiovascular screening and clinical follow-up data of all included elite athletes, including demographic characteristics such as age, sex, ethnicity, body surface area (BSA), type of athletic discipline and time (years) participating on a professional athlete level. Cardiovascular screening consisted of electrocardiography, laboratory assessment (high-sensitive troponin T (HsTnT), N-terminal prohormone of brain natriuretic peptide (NT-proBNP), creatine phosphokinase-MB (CKMB), inflammatory markers (C reactive protein (CRP) and leucocytes) and cardiovascular MRI (CMR). Post-infection cardiovascular screening was performed according to the standardised ELITE screenings protocol and was performed at the Amsterdam University Medical Centres or the Erasmus University Medical Centre. SARS-CoV-2 symptom severity was recorded, classified as: (1) asymptomatic (no symptoms during infection); (2) cardiovascular symptoms (dyspnoea, chest pain, (near-)syncope and palpitations); and (3) symptomatic; no cardiovascular symptoms (eg, gastrointestinal, respiratory symptoms and fever). The date of SARS-CoV-2 onset was defined as the date of positive PCR, or in case of positive serology, the date of onset of symptoms. The interval between infection and cardiovascular assessment was calculated based on the date of SARS-CoV-2 infection onset.

Electrocardiogram

Twelve-lead resting supine ECGs were independently assessed by two investigators, of which one expert in (sports) cardiology, according to the international recommendations for electrocardiographic interpretation in athletes by the European Society of

Table 3 Prospective assessment of pre-SARS-CoV-2 and post-SARS-CoV-2 infection ECG, laboratory and CMR findings within the same elite athlete

	CMR pre-SARS-CoV-2	CMR post-SARS-CoV-2	
	n=23	n=23	P value
ECG assessment			
Heart rate (bpm), mean (±SD)	54 (10)	57 (12)	0.341
ECG conclusion (according to 2017 international athlete ECG criteria), n (%)			0.676
Normal	15 (65)	13 (57)	
Borderline	3 (13)	4 (17)	
Abnormal	1 (4)	2 (9)	
Laboratory assessment, n (%)			
hsTnT >14 ng/L	1 (5)	1 (5)	1.000
NT-proBNP >130 ng/L	0 (0)	1 (5)	1.000
CKMB >5.2 ug/L	1 (5)	2 (10)	0.143
CRP >5 mg/L	0 (0)	0 (0)	
Leucocytes >10.5×10^9	1 (5)	0 (0)	1.000
CMR assessment, mean (±SD)			
Left ventricle			
LVEDV/BSA (mL/m^2)	116 (16)	121 (15)	0.343
LVESV/BSA (mL/m^2)	51 (11)	54 (8)	0.353
LVSV (mL)	135 (25)	137 (26)	0.745
LVEF (%)	56 (5)	55 (5)	0.551
LVM/BSA (g/m^2)	66 (16)	55 (15)	**0.018**
Ratio of (LVM/LVEDV)	0.6 (0.1)	0.5 (0.9)	**0.001**
Right ventricle			
RVEDV/BSA (mL/m^2)	120 (18)	120 (15)	0.929
RVESV/BSA (mL/m^2)	56 (13)	54 (9)	0.636
RVSV (mL)	133 (25)	133 (24)	0.983
RVEF (%)	54 (6)	55 (5)	0.629
Ratio of LVEDV/RVEDV	1.0 (0.1)	1.0 (0.1)	0.199
Native T1 relaxation time* (ms)	938 (22)	967 (27)	0.440
LGE, n (%)			1.000
Non-pathological LGE	23 (100)	23 (100)	
No LGE	12 (52)	12 (52)	
Hinge point LGE	11 (48)	11 (48)	
Pathological LGE	0 (0)	0 (0)	

*Amsterdam UMC normal value of native T1 relaxation time=950–1050 ms.
CKMB, creatine phosphokinase-MB; hsTnT, high-sensitive troponin T; LGE, late gadolinium enhancement; LVEDV, left ventricle BSA indexed end-diastolic volume; LVEF, left ventricle ejection fraction; LVESV, left ventricle BSA indexed end-systolic volume; LVM, left ventricle BSA indexed wall mass; LVSV, left ventricle stroke volume; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; RVEDV, right ventricle BSA indexed end-diastolic volume; RVEF, right ventricle ejection fraction; RVESVi, right ventricle BSA indexed end-systolic volume; RVSV, right ventricle stroke volume.

Cardiology (ESC), and accordingly classified as normal, borderline or abnormal.[12 13]

Cardiovascular magnetic resonance

Contrast-enhanced CMR was performed on 1.5-Tesla and 3.0-Tesla MRI scanners (Siemens Avanto Fit 1.5T, Philips Elition 3.0T or GE Healthcare SGINA Artist 1.5T) with ECG gating and a dedicated body coil for cardiac measurements. The CMR protocol included cine imaging of long-axis and short-axis views, native T1 modified look locker inversion recovery sequence mapping short-axis slices and late gadolium enhancement (LGE) images.

CMR data analysis was performed in Circle Cardiovascular Imaging (cvi42 V.5.12.4, Calgary, Alberta, Canada) by a core lab consisting of ELITE investigators (SMV, JCvH, JJND and MAvD), with dedicated supervision of expert radiologists and imaging cardiologists (SMB, AvR, RNP, MG and AH) with extensive experience in quantitative analysis of athlete CMR data. Epicardial and endocardial end-systolic and diastolic contours of both ventricles were automatically determined using an artificial intelligence integrated tool and manually corrected if needed. Short-axis cine images were used to determine left and right ventricular (LV/RV) end-diastolic and end-systolic volumes (including papillary muscle and trabecularisation) (EDV/ESV), stroke volumes and ejection fractions and LV mass (LVM) (excluding papillary muscle and trabecularisation). All volumes were indexed for BSA according to the Mosteller formula. The remodelling index (LVM/LVEDV) and balanced remodelling ratio (LVEDV/RVEDV) were determined. The presence of LGE (including pericardial LGE) was determined on phase-sensitive inversion recovery images through visual identification and if visually present quantified using a threshold of 6 SD above normal myocardium signal intensity. Hinge-point LGE was not considered indicative of SARS-CoV-2 myocardial involvement. Native T1 relaxation times were measured in all acquired slices (basal, midventricular and apical) using a 15% margin of epicardial and endocardial contours. All CMRs were clinically

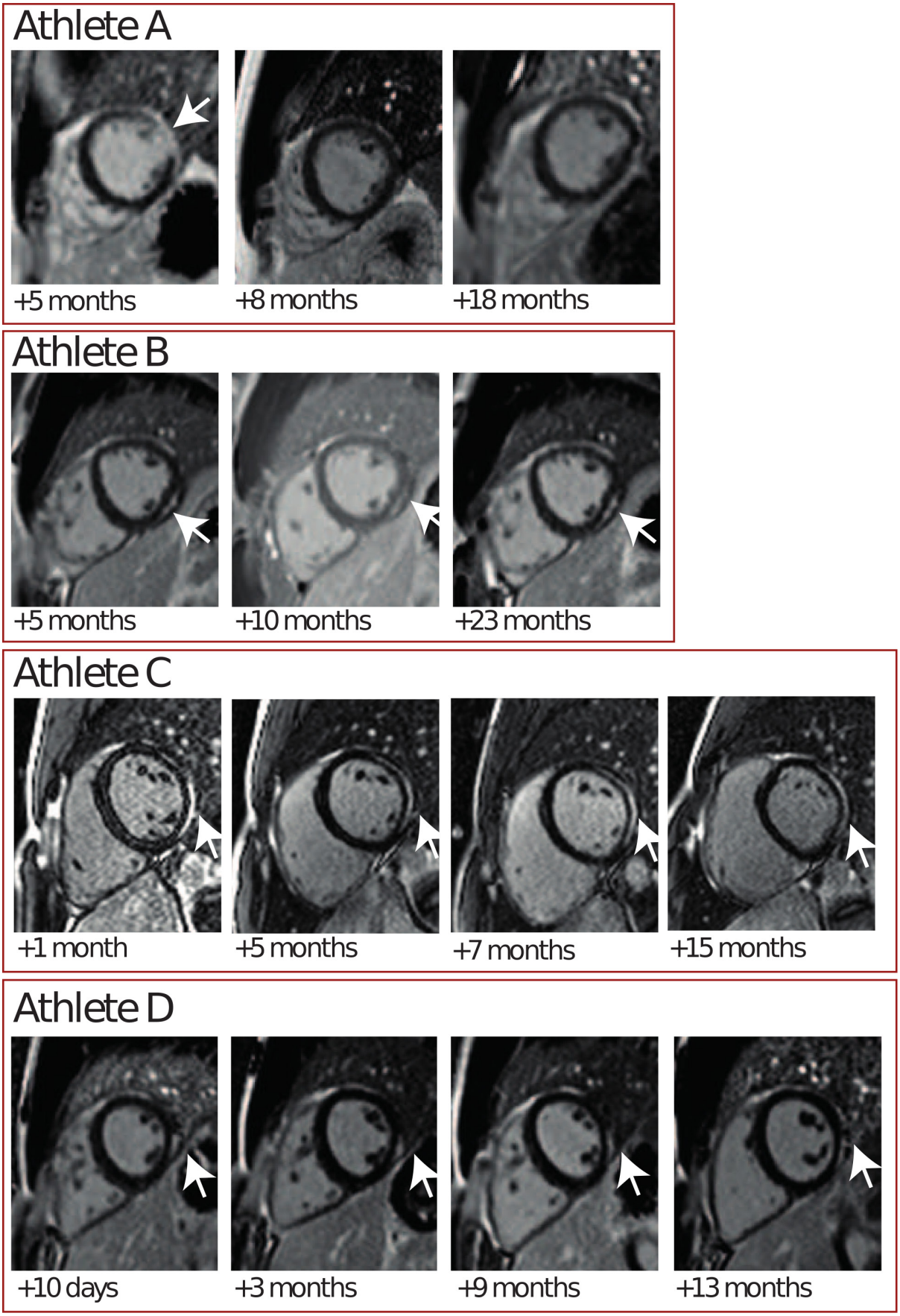

Figure 1 Short-axis late gadolinium enhancement (LGE) images of individual sequential CMRs performed in four athletes with SARS-CoV-2 associated pericardial/myocardial LGE demonstrated with an arrow including time interval between infection with SARS-CoV-2 and CMR. CMR, cardiovascular MRI.

Table 4 Individual case description over time of athletes with myocardial involvement after infection with SARS-CoV-2

	Athlete A	Athlete B	Athlete C*	Athlete D
Age	27	21	21	18
Sex	Male	Male	Male	Male
SARS-CoV-2 symptoms	Cough	Fever and cough	Nasal congestion, muscle ache and tiredness	Nasal congestion, headache, loss of smell and taste
Cardiac symptoms	None	None	Palpitations	None
Post-infection ECG abnormalities	None	None	Decrease of T wave amplitude in lead II and aVF, and T-wave inversion in lead III	New inferolateral T wave inversions
Infection to assessment interval	5 months	5 months	1 month	10 days
Initial cardiac MRI				
LGE	Basal to apical lateral myocardial and pericardial	Focal pericardial/epicardial at inferolateral midventricular wall with pericardial effusion (PE)	Basolateral to midventricular posterior epicardial	Pericardial/ epicardial LGE at the inferolateral wall
Native T1 relaxation time (ms)	939±63 ms	NA	1232±101 ms	NA
Sports restriction	No	No	9 months	1 month
LGE evolution	Complete resolution (8 and 18 months)	Persisting LGE without PE (10 and 17 months)	Persisting LGE without inflammation (15 months)	Persisting LGE (3 and 9 months)
Return to sports	No active myocardial inflammation	No active myocardial inflammation	After resolution of symptoms, ECG abnormalities, myocardial inflammation (assessed by PET-CT) and normalisation of cardiac biomarkers	After resolution of symptoms and ECG abnormalities and in the absence of elevated inflammatory markers

*The clinical course of this case, including LGE evolution, has been extensively described elsewhere.[24]
aVF, augmented vector foot; LGE, late gadolinium enhancement.

assessed according to local protocol and normal values. Due to interscanner differences in normal T1 relaxation times, only the T1 mapping results from the Amsterdam UMC performed on a Siemens Avanto Fit 1.5T MRI machine were included in the tables and analyses. T1 slices with poor quality (ie, due to movement or artefacts) were excluded from analyses.

Follow-up

When the CMR of athletes infected with SARS-CoV-2 demonstrated cardiac abnormalities, follow-up CMR was performed according to clinical judgement, with a preference for repeat evaluations at 3, 6 and 9 months post-infection. Additionally, intensive rhythm monitoring was performed using multiple conventional 24-hour Holters and 8-day continuous 1-lead monitoring (Philips Biosensor) and cardiopulmonary exercise tests. Moreover, in all athletes exposed to SARS-CoV-2, we collected data on cardiac symptoms and/or events (dyspnoea, chest pain, (near-)syncope and palpitations), and return to sports, including current level of sports participation, through a combination of the ongoing data collection of ELITE, the athlete management system and by outcome data collection performed by their dedicated personal and team (sports) physicians.

Statistical methods

Categorical values are presented as numbers and percentages; continuous variables are presented as mean and SD or median with IQR, as appropriate. The normality of distribution was visually analysed with histograms and calculated using Shapiro-Wilk's test. For the cross-sectional analyses comparing athletes infected with SARS-CoV-2 with athletes who were not infected, we compared ECG data, cardiac biomarkers, inflammatory markers and CMR parameters using unpaired t-tests, Mann-Whitney U tests, χ^2 tests or Fisher's exact tests, as appropriate. For the subgroup eligible for analyses using pre- and post-infection data, we applied paired t-tests, Wilcoxon signed-rank tests or McNemar tests. The alpha level was set at 0.05. Athletes with missing data for a specific variable were only excluded from the analysis of that specific variable. However, they were included in other analyses conducted. Statistical analyses were performed using R (Rstudio V.2021.09.0).

RESULTS

A total of 259 elite athletes were included between May 2019 and November 2022. In total, 123 elite athletes recovered from a SARS-CoV-2 infection, and 136 were not exposed to SARS-CoV-2 (self-reported to be negative and confirmed with a negative serology test (n=107), or included in ELITE before the onset of the COVID-19 pandemic (n=29) (table 1). Athletes infected with SARS-CoV-2 were similar in sex distribution (33% vs 45% female athletes) compared with elite athlete controls. However, athletes infected with SARS-CoV-2 were younger (25±6 vs 27±7 years, p=0.014), had a higher BSA (2.0±0.2 vs 1.9±0.2 m^2, p=0.006) and fewer athletes were of Caucasian ethnicity (80% vs 99%, p=<0.001). A higher percentage of athletes infected with SARS-CoV-2 participated in football (30% vs 7%, p<0.001) and had fewer professional athlete years (11±6 vs 13±7, p=<0.001). Of all athletes infected with SARS-CoV-2, 19% were asymptomatic, 9% had cardiovascular symptoms (palpitations 6%, chest pain 9%) and 72% had respiratory symptoms (no cardiovascular symptoms). The mean symptom duration was 10±15 days. Follow-up data were collected up to a mean of 26.7 (±5.7) months in elite athletes who recovered from SARS-CoV-2 infection. No athletes were lost to follow-up; missing data are presented in the online supplemental material.

Cross-sectional cardiovascular assessment

We compared 123 elite athletes exposed to SARS-CoV-2 with 136 elite athlete controls. The mean time interval between the

onset of SARS-CoV-2 infection and cardiovascular assessment was 3.9 (±2.9) months. Results of ECG, laboratory and CMR findings in elite athletes exposed to SARS-CoV-2 compared with elite athlete controls are presented in table 2 and the online supplemental material.

Electrocardiography and laboratory assessment

Elite athletes exposed to SARS-CoV-2 infection showed a higher resting heart rate (57 (51–66) vs 52 (46–57) bpm, p<0.001) compared with controls. There were no differences in ECG categories (normal 82% vs 87%, borderline 9% vs 10%, and abnormal 5% vs 2%). Detailed ECG assessments can be found in the online supplemental material. Furthermore, there was no difference in the amount of athletes with elevated cardiac biomarkers (hsTnT, NT-proBNP and CK-MB) and inflammatory markers (CRP and leucocytes), compared with controls.

Cardiovascular magnetic resonance imaging

The CMR findings in elite athletes exposed to SARS-CoV-2 revealed no significant differences in mean LVEDV (116±18 vs 120±19 mL/m^2), LVESV (51±10 vs 53±11 mL/m^2), LVEF (56±5 vs 56±5%), RVEDV (117±18 vs 121±20 mL/m^2), RVESV (54±10 vs 55±11 mL/m^2) and RVEF (54±5 vs 55±5%) when compared to non-exposed elite athlete controls. Similarly, there were no differences in cardiac remodelling ratios: LVM/LVEDV (0.5±0.1 vs 0.5±0.1) and LVEDV/RVEDV (1.0±0.1 vs 1.0±0.1). Moreover, infected athletes did not show higher native T1 relaxation times (962±22 vs 961±24 ms). Four (3%) athletes exposed to SARS-CoV-2 demonstrated pathological non-ischaemic patterns of myocardial LGE (≤20% of total LV mass); none of the non-exposed athletes demonstrated pathological LGE patterns. All four athletes with a pathological LGE pattern had normal cardiac function and volumetric parameters according to current athlete CMR reference ranges.[14] No pre-infection CMRs were available for the athletes with pathological LGE.

Prospective CMR assessment

CMR assessment before and after SARS-CoV-2 infection was available in 23 (19%) elite athletes (mean age 26.8±5, 44% women) (table 3 and online supplemental table 2). The mean time between pre-infection and post-infection CMR was 16±7 months and between SARS-CoV-2 onset and post-infection CMR 2±2 months. Comparing pre-infection to post-infection CMR, we only observed a difference in BSA-indexed LVM (66±16 vs 55±15 g/m^2, p=0.018) and LVM/LVEDV cardiac remodelling ratio (0.6±0.1 vs 0.5±0.9, p=0.001). The post-SARS-CoV-2 infection CMR demonstrated no change in volumetric (LVEDV (116±16 vs 121±15 mL/m^2), LVESV (51±11 vs 54±8 mL/m^2), RVEDV (120±18 vs 120±15 mL/m^2), RVESV (56±13 vs 54±9 mL/m^2)) and functional (LVEF (56±5 vs 55±5%), RVEF (54±6 vs 55±5%)) CMR parameters. Moreover, there were no changes in native T1 relaxation times and/or the presence of pathological LGE patterns over time.

Return to sports and follow-up

SARS-CoV-2 exposed athletes

Of the 123 elite athletes infected with SARS-CoV-2, four (3%) had distinct pathological LGE patterns with variable evolution over time (figure 1), without complex ventricular tachycardias ((non-) sustained ventricular tachycardia) and no increase in PVC burden. Their clinical course is described in table 4 and in the online supplemental material. The remaining 119 athletes (97%) demonstrated CMR function and volumetric parameters within normal ranges for athletes,[14] normal T1 relaxation times and no pathological LGE. None of the infected athletes reported cardiovascular symptoms or events after a mean follow-up of 26.7 (±5.8) months. In total, 96% (118) made a complete return to elite-level sports. Five (4%) athletes terminated their professional sports careers due to non-SARS-CoV-2 related considerations.

DISCUSSION

This elite athlete, extreme phenotyping study demonstrates that infection with SARS-CoV-2 is not associated with detrimental effects on cardiac function and ventricular volumes after return to elite sports, both when investigated cross-sectionally with controls (non-infected athletes) and prospectively, comparing pre-infection and post-infection CMR in individual athletes. There was a low prevalence (3%) of perimyocardial involvement, with a variable clinical presentation and clinical course over time, ranging from complete resolution of LGE to persistent LGE without other signs of inflammation. We found no signs of detrimental morphological changes or increases in ventricular arrhythmias in elite athletes with SARS-CoV-2 cardiac sequelae, even after the resumption of elite competitive sports. Moreover, during more than 2 years of follow-up, there were no new SARS-CoV-2 related (de novo) cardiac complaints or adverse cardiac events in athletes with and without clear SARS-CoV-2 associated abnormalities at the initial post-infection examination.

While elite athletes might be protected from COVID-19 complications due to their exceptional fitness, they also constitute a unique extreme phenotyping model to investigate the role of exercise as a second hit or trigger for adverse cardiac remodelling after SARS-CoV-2 infection.[15–17] Our findings therefore add to previous studies, which have mainly been cross-sectional, survey-based and performed on collegiate athletes. Our rate of post-SARS-CoV-2 myocardial injury was low compared with non-athletic populations and in line with previous findings.[2] Reassuringly, we observed only a decrease in the left-ventricular remodelling index in individuals with pre- and post-SARS-CoV-2 CMR measurements, which potentially illustrates the impact of detraining during the COVID-19 pandemic, and no other detrimental cardiac changes in volumes or function.

Short-term follow-up studies have shown an absence or a low prevalence of cardiac adverse events in athletes with and without cardiac involvement after infection with SARS-CoV-2.[9 18 19] Our study extends on these findings with long-term follow-up during a period of more than 2 years. Although the group of athletes with SARS-CoV-2 related damage was small, and caution is warranted when deriving conclusions from this data, it is reassuring that we found no increase in ventricular arrhythmias or adverse cardiac remodelling, even after resumption of elite-level sports. Most importantly, the cessation of professional athletic careers, irrespective of the presence or absence of cardiac sequelae, was not associated with a prior SARS-CoV-2 infection, during more than 2 years of follow-up.

Clinical implications

Standardised CMR-based screening following infection with SARS-CoV-2 provides valuable insights into myocardial damage, with a low but non-negligible detection rate, consistent with previous studies. Importantly, successful RTS can be achievable for elite athletes, even in the presence of myocardial abnormalities. Moreover, counselling during return-to-sports trajectories,[20 21] in addition to consensus return-to-play protocols[22 23]

is important in the RTS process. These findings contribute to the development of evidence-based protocols for safe RTS after SARS-CoV-2 infection, which is of benefit for all athletes, regardless of level.

Strengths and limitations

This study has several strengths. First, earlier studies were mainly performed on collegiate athletes and were cross-sectional in nature. While not the first study to report follow-up in athletes, this is the first study to prospectively investigate a rigorously defined and phenotyped cohort of elite athletes with homogeneous high-level training regimens. Second, the study included an appropriately selected control group including athletes enrolled prior to the pandemic. The absence of an appropriate control group has been an important limitation in numerous COVID-19 studies. Third, we conducted both cross-sectional controlled comparisons and prospective analyses using pre-SARS-CoV-2 and post-SARS-CoV-2 infection CMR measurements. Fourth, consecutive CMR studies were performed in cases with SARS-CoV-2 cardiac abnormalities to investigate potential adverse cardiac remodelling and/or recovery of the abnormalities on resumption of elite-level sport. Finally, this study performed intensive arrhythmia monitoring in elite athletes with SARS-CoV-2 associated cardiac sequelae, not only during exercise testing but also during general team trainings on the field.

Some aspects of our study warrant consideration. First, it was not possible to determine the exact date of SARS-CoV-2 infection in unvaccinated asymptomatic athletes who tested positive with an antibody test. Therefore, the causality of SARS-CoV-2 associated abnormalities cannot be ascertained, potentially leading to attribution bias. However, extensive history-taking revealed no other possible explanations. Second, as only four athletes were found with SARS-CoV-2 related cardiac abnormalities, clinical decision making based on this modest number should adhere to current international guidelines. Follow-up studies and case series in such athletes are urgently needed. Finally, we performed no arrhythmia monitoring in the athletes infected with SARS-CoV-2 without cardiac sequelae. These athletes remained however under the supervision of experienced sports physicians, trainers and other professionals, and commonly use photo-plethysmography, with no reports of new abnormalities during follow-up. Although an increase in subclinical ventricular arrhythmias cannot be excluded, the presence thereof remains unlikely.

CONCLUSION

Resumption of elite-level competitive sports after a SARS-CoV-2 infection is safe and feasible, even in the small group (3%) of athletes with SARS-CoV-2 related myocardial sequelae. Our study shows that, in elite athletes, cessation of professional sports careers was not associated with SARS-CoV-2. We observed no interaction between high-intensity exercise, cardiac remodelling and SARS-CoV-2 during cross-sectional controlled or prospective analysis. During more than 2 years of follow-up, no cardiac symptoms or adverse cardiac events were reported by the included athletes after resuming elite-level sports.

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Contributors HTJ conceived the main conceptual idea and acted as guarantor. JCvH, JJND, LVW and AH contributed to the design of the manuscript. JCvH wrote the manuscript with input from all authors in consultation with JJND, SMV, LVW, MAvD, MG, SMB, RNP, AvR, AH, MHM and HTJ. All authors discussed the findings and commented on the manuscript. YMP and AAMW supervised the project.

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Competing interests None declared.

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Patient consent for publication Not applicable.

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Supplemental material

Detailed CMR acquisition protocol

Cardiac magnetic resonance (CMR) was performed using preferentially 1.5 Tesla- scanners, a dedicated body coil for cardiac measurements, and electrocardiographic gating.

The standardised protocol consisted of:
(1) standard cardiac localizers
(2) modified Dixon sequence (lumbar spine)
(3) balanced turbo field echo cine imaging of long-axis views (two-, three-, and four-chamber left ventricle views, right ventricle outflow tract, pulmonary artery; field of view (FOV): 400 mm x 400 mm; voxel size: 2,1 x 2,1 x 6 mm; echo time (TE): 1,19 ms; repetition time (TR): 40,65 ms; flip angle: 70°)
(4) native T1 shortened modified look locker inversion recovery sequence (ShMOLLI) mapping on short-axis view in three slices (FOV: 400 mm x 400 mm; voxel size: 1,6 x 1,6 x 8 mm; 12 mm gap; TE: 1,09 ms; TR: 298,6ms; flip angle: 35°). Same position as slices of 2D balanced turbo field echo cine imaging SA.
(5) intravenous gadolinium (Dotarem®,Guerbet, Roissy, France) body weight dependent bolus injection (0.2 ml/Kg)
(6) 2D balanced turbo field echo cine imaging stack of 9-17 contiguous short-axis slices, covering both ventricles from apex to base, using balanced turbo field echo cine imaging (FOV: 400 mm x 400 mm; voxel size: 1,6 x 1,6 x 8 mm; 8 mm gap; TE: 1,16 ms; TR: 41,4 ms; flip angle: 70°)
(7) late gadolinium enhancement (LGE) 'overview' images (phase-sensitive inversion recovery sequence; PSIR) in two-, three-, four-chamber and short-axis views (identical to cine imaging localization and voxel size; TE: 1,03 ms; TR: 700 ms; flip angle: 40°), after a minimum of 8 minutes of intravenous Gadolinium injection. Same position as balanced turbo field echo cine imaging SA, 2,3 and 4 CH.
(8) LGE 'high-resolution' PSIR images (identical to LGE overview location; voxel size 1,6 x 1,6 x 8 mm; TE: 1,55 ms; TR: 750; flip angle: 20°)
(9) Post-contrast (15 minutes after Gadolinium injection) T1 ShMOLLI mapping (identical to native settings positions not parameters)
(10) three-dimensional whole heart dataset using respiratory navigator gating and electrocardiograph triggered isotropic 1.5 mm in diastole.

Cross-sectional cardiovascular assessment

Supplemental Table 1 presents the missing data from cross-sectional cardiovascular assessments, including laboratory measurements and ECGs. In the cross-sectional analyses, we were unable to conduct functional and volumetric CMR analyses of the right ventricle in one athlete due to the unavailability of contrast-enhanced images. Additionally, late gadolinium enhancement analysis was not performed in one athlete for the same reason. Furthermore, a total of 21 athletes did not undergo CMR at the Amsterdam UMC, and 26 athletes had their CMR conducted using a 3 Tesla MRI scanner.

Supplemental Table 1. Missing data of cross-sectional assessment of ECG, and laboratory findings in elite athletes exposed to SARS-CoV-2 versus non-exposed elite athletes.

		Total n=259			P-value
	SARS-CoV-2 infected elite athletes n=123	**Missing, n (%)**	**Elite athlete controls n=136**	**Missing, n (%)**	
Elevated Troponin T (>14 ng/L) , n (%)	9 (7.3)	7 (6)	6 (4.4)	7 (5)	0.456
Elevated NT-proBNP (>130 ng/L)	2 (1.6)	9 (7)	3 (2.2)	4 (3)	1.000
Elevated CKMB (>5.2 ug/L)	14 (11.4)	25 (20)	21 (15.4)	37 (27)	0.278
Elevated CRP (>5 mg/L)	2 (1.6)	27 (22)	4 (2.9)	33 (24)	0.743
Elevated leukocytes (>10.5 x10^9/L)	4 (3.3)	5 (4)	3 (2.2)	5 (4)	0.888
Heart rate (bpm), median (IQR)	57.0 (51.0 to 65.8)	5 (4)	52.0 (46.0 to 57.0)	1 (1)	**<0.001**
ECG assessment (according to 2017 international athlete ECG criteria)		5 (4)		1 (1)	0.357
Normal	101 (82.1)		118 (86.8)		
Borderline	11 (8.9)		14 (10.3)		
Abnormal	6 (4.9)		3 (2.2)		

Borderline findings of ECG assessment

In elite athletes exposed to SARS-CoV-2, according to consensus document certain borderline ECG findings did not warrant additional examinations. This included left atrial enlargement (LAE) in five athletes, right atrial enlargement (RAE) in two athletes, right axis deviation (RAD) in one athlete, and complete right bundle branch block (RBBB) in one athlete. However, according to consensus document two athletes exposed to SARS-CoV-2 warrant additional examinations based on their borderline ECG findings, which included both LAE and RAE in one athlete, and RAD combined with RBBB in another athlete.

For the athlete controls who were not exposed to SARS-CoV-2, there were also borderline ECG findings that did not warrant additional examinations according to consensus document. These findings included LAE in six athletes, RAE in three athletes, and complete RBBB in two athletes. However, three athlete controls required further evaluation based on their borderline ECG findings, which included both LAE and RAE in two athletes, and prolonged QTc with biphasic T waves in lead V3 and V4 in one athlete.

Abnormal findings of ECG assessment

Abnormal ECG findings in elite athletes exposed to SARS-CoV-2 included prolonged QRS duration of 160ms combined with T-wave inversion (TWI) in lead I and aVL (n=1), left axis deviation (LAD) combined with 2 premature ventricular beats (PVBs) exhibiting right bundle branch block (RBBB) morphology (n=1), presence of 3 PVBs with left bundle branch block (LBBB) morphology (n=1), TWI in the inferior leads (lead II, III, and AVF) (n=1), TWI in lead V2 and V3 in a mature white athlete (n=1), and TWI in lead III and biphasic T-wave in lead aVF (n=1).

Among athlete controls not exposed to SARS-CoV-2, abnormal ECG findings included sinus rhythm combined with atrial tachycardia and a single premature atrial complex (n=1), J-point depression in lead III and aVF (n=1), and TWI in lead II, III, and V5 (n=1).

Prospective cardiovascular assessment

Population characteristics are presented in Supplemental Table 2. Within our prospective analyses, two athletes could not be included in the analysis of right ventricle functional and volumetric due to multiple CMR artefacts. In addition, one athlete had no T1-mapping images for native T1

assessment. In total 4 athletes were excluded from the ECG sub-analyses (no pre-SARS-CoV-2 ECG available). Additionally, from laboratory sub-analyses; HsTnT (n=2), NT-ProBNP (n=1), CKMB (n=13), and CRP (n=13) athletes were excluded as no pre-SARS-CoV-2 data was available.

Supplemental Table 2. Population characteristics of elite athletes with both pre- and post-SARS-CoV-2 cardiovascular assessment (prospective cardiovascular assessment).

	SARS-CoV-2 infected elite athletes n = 23
Age (years), mean (±SD)	26.8 (5.14)
Woman, n (%)	10 (43.5)
Ethnicity, n (%)	
Caucasian	21 (91.3)
African/Afro-Caribbean	1 (4.3)
West Asian (Arabic and Middle Eastern)	0 (0)
East Asian and South Asian	0 (0)
Latin America	1 (4.3)
Athletic discipline, n (%)	
Field hockey	1 (4.3)
Road cycling	3 (13.0)
Rowing	3 (13.0)
Football	2 (8.7)
Tennis	1 (4.3)
Track cycling	2 (8.7)
Water polo	6 (26.1)
Miscellaneous[a]	5 (21.7)
Professional athlete years, mean (±SD)	13.0 (5.0)
SARS-CoV-2 symptoms severity, n (%)	
Asymptomatic	0 (0)
Cardiovascular symptoms[b]	2 (9)
No cardiovascular symptoms	21 (91)

[a] *Miscellaneous: basketball (n=1), skateboarding (n=1), table tennis (n=1), and wheelchair basketball (n=2)*
[b] *Cardiovascular symptoms: chest pain (n=1, and both chest pain and palpitations (n=1)*

Case description of SARS-CoV-2 infected athletes with cardiac sequelae

Four (3%) male elite athletes, who engaged in mixed, high-intensity, competitive, acceleration-deceleration sports demonstrated pathological non-ischemic LGE patterns after confirmed SARS-CoV-2 infection. The LGE patterns showed distinct patterns of evolution over time and are shown in Figure 1.

Athlete A, with as main symptom cough (March 2020), initially demonstrated (CMR 5 months after infection) LV LGE of the basal to apical lateral myo- and pericardium, with normal native T1 relaxation time. However, CMR repeated at 8 and 18 months after infection demonstrated complete LGE resolution.

Athlete B had a fever and cough (March 2020), and initial CMR (5 months after infection) showed focal peri-/epicardial increased signal intensity at the inferolateral midventricular wall of the LV with pericardial effusion (PE) at the lateral wall; the LGE persisted without PE on repeated CMR 10 and 17 months after infection.

Athlete C had palpitations during a resting period after training 15 days after PCR- confirmed SARS-CoV-2 infection. The clinical course of this case has been extensively described elsewhere (1). Initial CMR demonstrated basolateral to midventricular posterior epicardial LGE with locally increased native T1 relaxation time (1232 ± 101ms). The athlete was diagnosed with focal COVID-19 myocarditis and was given a negative sports advice, which he adhered to. Repeated CMR nine months after infection showed resolution of all signs of ongoing myocardial inflammation but with persistent LGE.

Athlete D was referred for cardiovascular evaluation after demonstrating new inferolateral T wave inversions on ECG after a recent SARS-CoV-2 infection. Initial CMR 10 days after infection showed peri-/epicardial LGE at the inferolateral wall which persisted on CMR at 3 and 9 months. One month after the infection, there was a resolution of ECG abnormalities.

After a comprehensive multi-disciplinary evaluation (2), all four athletes made a complete return to elite competitive sports. Athletes A and B were not restricted from elite sports during follow-up (as there were no absolute contra-indications for sports in these athletes). Athletes C and D adhered to an extensive graduated return-to-play protocol before making a complete return to elite, competitive sports. All four athletes underwent intensive rhythm monitoring, including Holter monitoring during team-training sessions, which demonstrated no increases of PVCs over time and no complex ventricular tachycardias ((non-) sustained ventricular tachycardia).

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