



SARS-CoV-2 Cardiac Involvement in Young Competitive Athletes

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BACKGROUND: Cardiac involvement among hospitalized patients with severe coronavirus disease 2019 (COVID-19) is common and associated with adverse outcomes. This study aimed to determine the prevalence and clinical implications of COVID-19 cardiac involvement in young competitive athletes.

METHODS: In this prospective, multicenter, observational cohort study with data from 42 colleges and universities, we assessed the prevalence, clinical characteristics, and outcomes of COVID-19 cardiac involvement among collegiate athletes in the United States. Data were collected from September 1, 2020, to December 31, 2020. The primary outcome was the prevalence of definite, probable, or possible COVID-19 cardiac involvement based on imaging definitions adapted from the Updated Lake Louise Imaging Criteria. Secondary outcomes included the diagnostic yield of cardiac testing, predictors for cardiac involvement, and adverse cardiovascular events or hospitalizations.

RESULTS: Among 19 378 athletes tested for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, 3018 (mean age, 20 years [SD, 1 year]; 32% female) tested positive and underwent cardiac evaluation. A total of 2820 athletes underwent at least 1 element of cardiac triad testing (12-lead ECG, troponin, transthoracic echocardiography) followed by cardiac magnetic resonance imaging (CMR) if clinically indicated. In contrast, primary screening CMR was performed in 198 athletes. Abnormal findings suggestive of SARS-CoV-2 cardiac involvement were detected by ECG (21 of 2999 [0.7%]), cardiac troponin (24 of 2719 [0.9%]), and transthoracic echocardiography (24 of 2556 [0.9%]). Definite, probable, or possible SARS-CoV-2 cardiac involvement was identified in 21 of 3018 (0.7%) athletes, including 15 of 2820 (0.5%) who underwent clinically indicated CMR (n=119) and 6 of 198 (3.0%) who underwent primary screening CMR. Accordingly, the diagnostic yield of CMR for SARS-CoV-2 cardiac involvement was 4.2 times higher for a clinically indicated CMR (15 of 119 [12.6%]) versus a primary screening CMR (6 of 198 [3.0%]). After adjustment for race and sex, predictors of SARS-CoV-2 cardiac involvement included cardiopulmonary symptoms (odds ratio, 3.1 [95% CI, 1.2, 7.7]) or at least 1 abnormal triad test result (odds ratio, 37.4 [95% CI, 13.3, 105.3]). Five (0.2%) athletes required hospitalization for noncardiac complications of COVID-19. During clinical surveillance (median follow-up, 113 days [interquartile range=90–146]), there was 1 (0.03%) adverse cardiac event, likely unrelated to SARS-CoV-2 infection.

CONCLUSIONS: SARS-CoV-2 infection among young competitive athletes is associated with a low prevalence of cardiac involvement and a low risk of clinical events in short-term follow-up.

Key Words: athletes ■ COVID-19 ■ myocarditis ■ return to sport ■ SARS-CoV-2

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Cardiac involvement associated with adverse outcomes is common among hospitalized patients with severe coronavirus disease 2019 (COVID-

19).¹ Limited data exist on the prevalence and clinical relevance of cardiac involvement in nonhospitalized—and otherwise healthy—populations including young

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Clinical Perspective

What Is New?

- The Outcomes Registry for Cardiac Conditions in Athletes was created to investigate the prevalence and clinical outcomes of cardiovascular disease, including cardiac involvement after severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, among young competitive athletes.
- Our data indicate a low prevalence of cardiac involvement (0.5% to 3.0%) after SARS-CoV-2 infection among young competitive athletes undergoing return-to-play cardiac testing.
- No adverse cardiac events related to SARS-CoV-2 infection have been observed among >3000 collegiate athletes during short-term clinical surveillance.

What Are the Clinical Implications?

- The findings suggest that asymptomatic or mildly symptomatic athletes who have fully recovered from SARS-CoV-2 infection may return to sport without cardiac testing.
- Cardiac evaluation inclusive of 12-lead ECG, troponin, and transthoracic echocardiography should be considered in athletes with moderate or cardiopulmonary symptoms during initial illness or on return to exercise.
- Cardiac magnetic resonance imaging is most useful in athletes with high pretest probability for SARS-CoV-2 cardiac involvement as defined by the presence of cardiopulmonary symptoms or abnormalities on cardiac testing (ECG, troponin, transthoracic echocardiography); however, the significance of cardiac magnetic resonance imaging findings in the absence of symptoms remains unknown.

Nonstandard Abbreviations and Acronyms

CMR	cardiac magnetic resonance imaging
COVID-19	coronavirus disease 2019
OR	odds ratio
RTP	return to play
SARS-CoV-2	severe acute respiratory syndrome coronavirus 2
TTE	transthoracic echocardiography

competitive athletes. Postviral myocarditis is a well-documented cause of sudden cardiac death during exercise.²⁻⁴ Concern for COVID-19 myocarditis contributed to the cancellation of organized sports and led to the development of expert consensus recommendations for the cardiac evaluation of athletes after severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection.⁵⁻⁸

Observational data among middle-aged people recovering from SARS-CoV-2 infection document high rates of potential cardiac involvement (78%) as evidenced by elevated cardiac biomarkers and cardiac magnetic resonance imaging (CMR) abnormalities.⁹ Single-center observational cohort studies of collegiate athletes undergoing return-to-play (RTP) cardiac testing inclusive of CMR report highly variable rates of SARS-CoV-2 cardiac involvement (1.4% to 56%) using heterogeneous, non-standardized definitions of disease.¹⁰⁻¹³ A subsequent study in professional athletes undergoing RTP cardiac testing reported a lower prevalence of cardiac involvement (0.6%) when using a testing strategy of 12-lead ECG, blood cardiac troponin assay, and transthoracic echocardiography (TTE), followed by CMR when clinically indicated.¹⁴ The prevalence, clinical correlates, and outcomes of SARS-CoV-2 cardiac involvement in competitive athletes are unclear.

The Outcomes Registry for Cardiac Conditions in Athletes was designed to address these issues. This registry collected data documenting cardiac diagnoses, noncardiac diagnoses, and attendant clinical outcomes among competitive collegiate athletes returning to organized athletics during the SARS-CoV-2 pandemic.

METHODS

The data that support the findings of this study are available from the corresponding author on reasonable request. This was a prospective observational cohort study. The primary aim was to determine the prevalence of cardiac involvement in collegiate athletes diagnosed with SARS-CoV-2 infection. Secondary aims were to assess the diagnostic yield of cardiac testing, identify risk factors for cardiac involvement, and determine the prevalence of adverse cardiac events or hospitalizations after SARS-CoV-2 infection in collegiate athletes. Deidentified data were collected from participating institutions from September 1, 2020, to December 31, 2020, inclusive of all athletes with previous SARS-CoV-2 infection. All aspects of this study were approved by the Massachusetts General Brigham Institutional Review Board (protocol 2020P002667).

Recruitment, Eligibility Criteria, and Data Collection

All National Collegiate Athletic Association institutions that implemented SARS-CoV-2 infection testing and performed cardiac preparticipation evaluations for athletes infected with SARS-CoV-2 were eligible. Athletes with confirmed SARS-CoV-2 infection by laboratory testing (polymerase chain reaction, antigen, or antibody) and who underwent local cardiovascular evaluation were included. Athletes were often tested for SARS-CoV-2 infection multiple times by participating institutions, and were included in this study if they had any positive SARS-CoV-2 test. Each athlete is only represented once in the registry based on the initial positive SARS-CoV-2 test. Evaluations included a clinical assessment and a range of cardiac testing as determined by the institution including 1 or more of the following: 12-lead ECG, cardiac troponin assay, TTE, and

CMR. Triad testing refers to the combination of ECG, troponin, and TTE. Athletes were excluded if they had only a clinical assessment with no additional cardiac testing or if they had not yet started their cardiac evaluation.

The Massachusetts General Hospital served as the central data collection center. Each participating institution submitted data using a standardized data capture tool and deidentified original clinical reports of the cardiovascular testing performed. Raw imaging data for both TTE and CMR were not available to the study investigators and diagnoses were therefore adjudicated using original clinical reports. Submitted data collection spreadsheets were reviewed for completeness and accuracy before being transcribed into a central REDCap database. Further details on recruitment and data collection methods are provided in the [Methods in the Data Supplement](#).

Key Definitions

Symptom severity was categorized using reported symptoms from the initial infection and the presence of any recurring symptoms with return to exercise as initially proposed for collegiate athletes.⁵ Prespecified symptom severity categories included asymptomatic, mild, moderate, and cardiopulmonary.⁵ Mild symptoms were defined as cough, fatigue, gastrointestinal symptoms (nausea, vomiting, or diarrhea), headache, anosmia, ageusia, rhinorrhea, sore throat, or nasopharyngeal congestion. Moderate symptoms were defined as the presence of COVID-19 toes or fingers, chills, fever, or myalgias. Cardiopulmonary symptoms were defined as chest pain, shortness of breath, palpitations, or exercise intolerance. If an athlete had symptoms in multiple categories, he or she was assigned the most severe category on the basis of the symptoms. ECGs were deemed normal or abnormal using international criteria for ECG interpretation in athletes.¹⁵ Abnormal ECG, TTE, and CMR reports were adjudicated as related to SARS-CoV-2 infection or unrelated to SARS-CoV-2 infection using prespecified definitions ([Table I in the Data Supplement](#)). A troponin measurement was considered abnormal if it was >99% upper limit of normal per the local laboratory commercial assay standards. Borderline abnormal triad testing was defined as any cardiac test that was considered abnormal by the local institution and led to CMR testing but did not meet prespecified study definitions after adjudication ([Table I in the Data Supplement](#)). These included detectable troponin level but not >99th percentile, nonspecific ECG abnormalities not fulfilling the international criteria, and TTE findings including abnormalities of unclear significance or those at the upper or lower limit of the normal range and considered to potentially represent pathology (ie, left ventricular ejection fraction 50%).

Study definitions were derived for both myocardial and pericardial involvement with imaging components adapted from the Updated Lake Louise Imaging Criteria ([Table I in the Data Supplement](#)).¹⁶ Definite myocardial involvement was defined as CMR T1 abnormality or presence of late gadolinium enhancement + T2 abnormality or CMR T2 abnormality + 1 or more supportive findings (left ventricular ejection fraction <45%, small or greater pericardial effusion, pericardial enhancement, or troponin >99% upper limit of normal). Probable myocardial involvement was defined as CMR T1 abnormality or presence of late gadolinium enhancement + 1 or more supportive findings (same as definite myocardial involvement). Possible myocardial involvement was defined as isolated CMR T1 abnormality or

presence of late gadolinium enhancement. SARS-CoV-2 pericardial involvement was defined as a small or greater pericardial effusion or pericardial enhancement on CMR. Any athlete meeting criteria for myocardial involvement of SARS-CoV-2 infection who also had pericardial involvement was labeled as definite, probable, or possible myopericardial involvement based on the definitions for myocardial involvement.

Outcomes for this study included adverse cardiovascular events (new clinically significant arrhythmias, clinical heart failure, or sudden cardiac arrest or death) or hospitalizations related to SARS-CoV-2. Follow-up time was defined as the date of symptom onset or date of positive SARS-CoV-2 test if asymptomatic (or if symptoms were not reported) to the date of last cardiovascular outcomes update from each institution.

Statistical Analysis

Patient characteristics, SARS-CoV-2 symptoms, and cardiac testing results were described using basic descriptive methods including frequency distributions, means and SDs, and medians and interquartile ranges; 95% CIs for all prevalence estimates were calculated by exact binomial distribution. Because of the small number of observed SARS-CoV-2 cardiac cases and the known small sample bias with maximum likelihood estimation, the Firth penalized logistic regression method was used to assess unadjusted associations with definite, probable, and possible SARS-CoV-2-related cardiac involvement. A multivariable Firth logistic regression model using backwards variable selection was built to identify independent predictors of SARS-CoV-2 myocardial/pericardial involvement. The final model was determined on the basis of goodness of fit using Akaike information criteria. Odds ratios (ORs) and 95% CIs are reported. Secondary analyses modeling only definite and probable SARS-CoV-2-related cardiac involvement were also conducted using the same approach. Statistical analyses were performed using SAS (version 9.4; SAS Institutes) and R: A Language and Environment for Statistical Computing (R Core Team; <https://www.R-project.org/>).

RESULTS

Study Population

Among 19 378 athletes enrolled during the study period, 3384 (17.5%) tested positive for SARS-CoV-2 infection and 3018 (15.6%) met study inclusion criteria ([Figure 1 in the Data Supplement](#)). Baseline characteristics for the cohort and for the subgroup with SARS-CoV-2 cardiac involvement are presented in Table 1. The cohort, comprising athletes from 42 colleges or universities, includes representation from 26 distinct sporting disciplines including American-style football (36%), baseball (9%), cross country/track and field (8%), lacrosse (6%), and basketball (6%) ([Table II in the Data Supplement](#)). The majority of athletes were asymptomatic (33%) or had mild symptom burden (29%), most commonly accounted for by loss of taste or smell (40%), headache (39%), and sore throat (31%) during the acute phase of SARS-CoV-2 infection ([Figure 1](#)). Cardiopulmonary symptoms (chest pain, shortness of breath, palpitations,

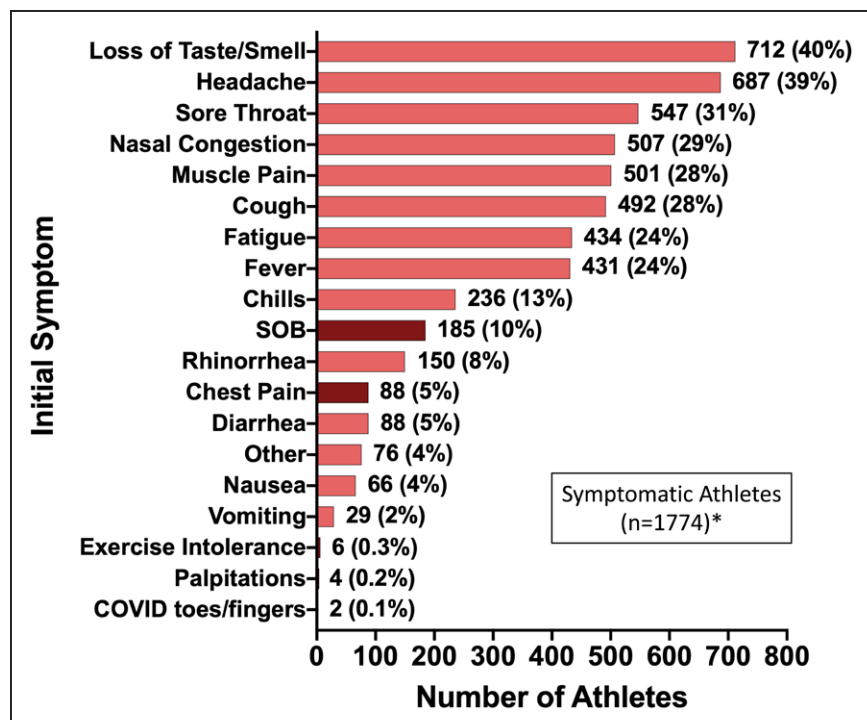


Figure 1. Prevalence of initial symptoms in athletes with symptomatic SARS-CoV-2 infection.

Maroon bars indicate cardiopulmonary symptoms. *Athletes with known initial symptoms. COVID indicates coronavirus disease; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; and SOB, shortness of breath.

or exercise intolerance) during the acute illness or on return to exercise were reported by 13% of athletes. The most commonly used (74%) cardiovascular testing protocol was triad testing (Table 1). A total of 198 athletes underwent CMR performed as part of the primary cardiovascular screening protocol irrespective of symptom burden or the results of other testing and 119 underwent CMR for clinical indications (Table III in the Data Supplement). The time from initial diagnosis of SARS-CoV-2 infection to the completion of each component of cardiac triad testing and CMR is shown in Figure 2. Abnormal triad testing possibly related to SARS-CoV-2 cardiac involvement was detected by TTE (24 of 2556 [0.9%]), 12-lead ECG (21 of 2999 [0.7%]), and cardiac troponin testing (24 of 2719 [0.9%]), including 5 athletes tested with high-sensitivity troponin I and the other 19 athletes tested with conventional troponin I or troponin T assays. Of athletes with abnormal triad testing possibly related to SARS-CoV-2, 65 had a single abnormal triad test, 2 athletes had 2 abnormal triad tests (12-lead ECG and TTE), and no athlete had abnormalities on all 3 triad tests.

Cardiac Involvement After SARS-CoV-2 Infection

A total of 21 athletes (0.7% [95% CI, 0.4–1.1]) had definite, probable, or possible SARS-CoV-2 cardiac involvement (Table 2). This included 6 of 198 (3.0% [95% CI, 1.1–6.5]) athletes identified by primary screening CMR, of whom 3 (1.5% [95% CI, 0.3–4.4]) had definite or probable cardiac involvement (Figure 3A), and 15 of 2820 (0.5% [95% CI, 0.3–0.9]) athletes identified by 1 or more elements of cardiac triad testing or moderate or

greater symptoms followed by clinically indicated CMR, of whom 12 (0.4% [95% CI, 0.2–0.7]) had definite or probable cardiac involvement (Figure 3B). For athletes in the clinically indicated CMR cohort with at least 1 abnormal triad test (n=34), 6 (17.7% [95% CI, 6.8–34.5]) had definite or probable cardiac involvement.

In the overall cohort, 137 of 3018 athletes (4.5% [95% CI, 3.8–5.3]) who underwent any form of cardiac screening had abnormal testing (Figure 4). Eighty-one (2.7% [95% CI, 2.1–3.3]) had abnormal cardiac testing possibly related to SARS-CoV-2 infection and 56 (1.9% [95% CI, 1.4–2.4]) had cardiac abnormalities unrelated to SARS-CoV-2 infection (a combination of known and new cardiac findings). Among athletes with 1 or more abnormal triad test results potentially related to SARS-CoV-2 (n=67), 37 underwent CMR, whereas the remaining 30 were ultimately deemed to have isolated testing abnormalities unrelated to SARS-CoV-2 (Table IV in the Data Supplement). Of the 37 athletes who underwent CMR for an abnormal triad test, only 2 (5.4%) had multiple abnormal tests (Figure 4). Fourteen athletes had normal triad testing but were found to have CMR abnormalities potentially related to SARS-CoV-2.

Predictors of SARS-CoV-2 Cardiac Involvement

Significant univariable predictors of definite, probable, or possible cardiac involvement included White Hispanic race (OR, 7.6 [95% CI, 2.2–26.1]), basketball participation (OR, 5.1 [95% CI, 1.8–14.5]), cardiopulmonary symptoms during acute infection or on resumption of exercise (OR, 4.2 [95% CI, 1.4–12.4]), and 1 or more abnormal triad test results (OR, 48.2 [95% CI, 18.5–125.6]) potentially related to SARS-CoV-2 infection (Table V in

Table 1. Patient Characteristics for the Total Cohort and for Athletes With Definite, Probable, or Possible Myocardial or Pericardial SARS-CoV-2 Involvement

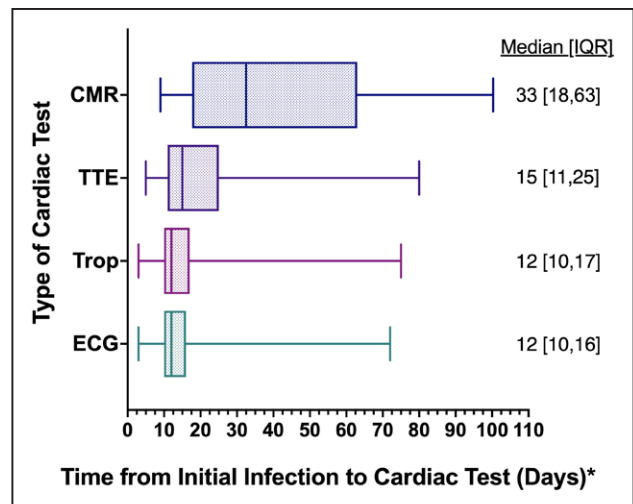
Patient characteristics*	Total cohort (n=3018)	Myocardial/pericardial SARS-CoV-2 involvement (n=21)
Female	957 (32)	10 (48)
Age, y	20 (1)	20 (2)
Male BMI, kg/m ²	27 (5)	25 (2)
Female BMI, kg/m ²	23 (3)	25 (5)
White non-Hispanic	1922 (64)	10 (48)
Black	829 (27)	8 (38)
White Hispanic	87 (3)	3 (14)
Mixed	62 (2)	0
Other†	80 (3)	0
Preexisting conditions*		
Sickle cell trait	31 (1)	1 (5)
Diabetes	10 (0.4)	1 (5)
Hypertension	10 (0.4)	0
Hyperlipidemia	7 (0.3)	1 (5)
Asthma (mild, intermittent)	154 (6)	0
Asthma (mild, persistent, or greater)	68 (2)	0
Immunosuppressive agent	5 (0.2)	0
Structural/valvular cardiac disease	18 (0.6)	0
Electrical cardiac disease	14 (0.5)	0
Symptom categories*		
Asymptomatic	887 (33)	5 (24)
Mild	789 (29)	4 (19)
Moderate	663 (25)	4 (19)
Cardiopulmonary	337 (13)	8 (38)
SARS-CoV-2–positive test		
PCR	2465 (82)	18 (86)
Antibody	263 (9)	2 (10)
Antigen	234 (8)	1 (5)
Unknown	56 (2)	0
Cardiovascular testing performed		
ECG + troponin + TTE	2231 (74)	0
ECG + troponin + TTE + CMR	188 (6)	18 (86)
ECG only	172 (6)	0
ECG + troponin	159 (5)	0
ECG + troponin + CMR	124 (4)	3 (14)
ECG + TTE	121 (4)	0
Other‡	23 (0.8)	0

Values are presented as n (%) or mean (SD). BMI indicates body mass index; CMR, cardiac magnetic resonance imaging; PCR, polymerase chain reaction; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; and TTE, transthoracic echocardiography.

*Partial data are available for the following characteristics: age, n=3006 (total cohort); sex, n=3017 (total cohort); BMI, n=2621 (total cohort) and n=20 (myocardial/pericardial SARS-CoV-2 involvement); race, n=2980 (total cohort); preexisting conditions, n=2800 (total cohort); and symptom categories, n=2676 (total cohort).

†The "Other" category includes Asian, American Indian, Native Hawaiian, Pacific Islander, and self-selected other.

‡Includes other combinations not listed.

**Figure 2. Time from initial infection to cardiac testing.**

*Time from initial infection calculated as the longest time from date of initial symptom onset or date of positive severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) to date of cardiac test. The total athletes with known time frame were as follows: cardiac magnetic resonance imaging (CMR), n=302; ECG, n=2765; troponin (Trop), n=2537; and transthoracic echocardiography (TTE), n=2406. Midline is the median; box, interquartile range (IQR); whiskers, 95% CI.

the Data Supplement). On multivariable analysis, after adjusting for sex and race, cardiopulmonary symptoms (OR, 3.1 [95% CI, 1.2–7.8]) and any abnormal triad test result (OR, 37.4 [95% CI, 13.3–105.3]) were predictive of cardiac involvement (Table VI in the Data Supplement).

Clinical Outcomes

No adverse cardiac events (median follow-up period, 130 days [interquartile range, 97–160]) were reported in athletes with definite, probable, or possible SARS-CoV-2 cardiac involvement (n=21). Among the entire cohort of athletes positive for SARS-CoV-2 (n=3018; median follow-up period, 113 days [interquartile range, 90–146]), we observed 1 adverse cardiac event (successfully resuscitated sudden cardiac arrest). This athlete underwent CMR 17 days after SARS-CoV-2 infection symptom onset without findings suggestive of acute cardiac involvement, rendering the cause of this event uncertain and likely unrelated to SARS-CoV-2. Ten athletes were either hospitalized (n=5) or treated in the emergency department (n=5) for noncardiac complications of SARS-CoV-2 infection. Noncardiac SARS-CoV-2 complications included submassive pulmonary embolism (n=1), symptomatic large pleural effusion (n=1), and palmar desquamation secondary to mild multisystem inflammatory syndrome (n=1).

DISCUSSION

We report data characterizing collegiate athletes undergoing cardiovascular evaluation after SARS-CoV-2

Table 2. Clinical Characteristics and Cardiac Evaluations for Athletes With Myocardial or Pericardial Involvement From SARS-CoV-2 as Adjudicated by CMR

Athlete number	Sex	Race/ethnicity	Past medical history	Symptom severity/duration, d: initial symptoms/exertional symptoms (SARS-CoV-2 test)	ECG	Troponin, ng/mL	TTE	CMR	Days from symptoms to CMR*	Certainty of involvement
Athletes with isolated myocardial involvement from SARS-CoV-2 (n=11)										
1	M	White	None	Moderate/"few days": headache, myalgias/NA (antibody)	Normal	Troponin I <0.01	LVEF 60% to 65%, no WMAs, no pericardial effusion	LVEF 58%, T1 LGE at the inferior (basal) segment, T2 increased signal consistent with mild myocardial edema, no pericardial effusion	39	Definite
2	M	Black	None	Mild/5: congestion, cough, loss of taste/smell, sore throat/NA (PCR)	Normal	Troponin T <0.01	Abnormal: LVEF 45% to 50%, no WMAs, no pericardial effusion	LVEF 41%, T1 LGE could not be assessed due to allergy, T2 global hyperintensity of LV myocardium c/w edema, no pericardial effusion	42	Definite
3	M	Black	T1D	Asymptomatic: NA (PCR)	Normal	Abnormal: hsTroponin 20	Not performed	LVEF 57%, T1 no LGE, T2 increased signal in LV myocardium consistent with edema, no pericardial effusion	34	Definite
4	F	Hispanic/Latino	None	Mild/2: loss of taste/smell, sore throat/unknown (PCR)	Abnormal: LAE, RAE	Troponin I <0.01	LVEF 60%, no WMAs, no pericardial effusion	LVEF 50%, T1 abnormal mapping and LGE in inferior (basal, mid), inferolateral (basal), anterolateral (mid) segments, T2 abnormal mapping in basal inferior segment, no comment on effusion	72	Definite
5	M	Black	None	Asymptomatic: none (PCR)	Normal	Troponin T <0.01	LVEF 50%, no WMAs, no pericardial effusion	LVEF 42%, increased T1 mapping, no LGE, increased T2 mapping, trace pericardial effusion	38	Definite
6	M	Black	None	Cardiopulmonary/9: chills, congestion, fevers, loss of taste/smell, myalgias, sore throat/exertional CP, DOE (PCR)	Normal	Troponin I <0.03	LVEF 53%, no WMAs, no pericardial effusion	LVEF 56%, normal T1 mapping, LGE of the inferolateral (apical) segment, normal T2 mapping without edema, no comment on effusion	29	Possible
7	M	White	ADHD	Asymptomatic: none (antibody)	Abnormal: RAE, LAE	Troponin I <0.01	LVEF 60% to 65%, no WMAs, no pericardial effusion	LVEF 61%, T1 LGE at inferior (basal), inferolateral (basal), inferoseptal (basal) segments, T2 no myocardial edema, no pericardial effusion	36	Possible
8	M	Black	ADHD, anxiety	Mild/1: headache, sore throat/exertional CP (PCR)	Normal	Troponin I <0.01	LVEF 60%, no WMAs, no pericardial effusion	LVEF 55%, abnormal T1 with LGE in inferior (basal), inferolateral (basal) segments, T2 normal mapping, trivial pericardial effusion	68	Possible
9	F	Black	None	Mild/3: loss of taste/smell/none (PCR)	Normal	Troponin I <0.01	LVEF 55%, no WMAs, no pericardial effusion	LVEF 54%, T1 abnormal ECV fractions midsegment, no LGE but myocardium appearance of "inhomogenous null," T2 normal mapping, no comment on effusion	74	Possible
10	M	Black	Sickle cell trait	Cardiopulmonary/2: fever, vomiting, SOB/DOE (PCR)	Normal	Troponin I <0.017	LVEF 50%, no WMAs, no pericardial effusion	LVEF 50%, T1 mapping normal, LGE present in inferior (mid), inferolateral (mid) segments, T2 mapping normal but degraded by motion artifact, no comment on effusion	84	Possible
11	M	White	None	Cardiopulmonary/5: fever, headache, congestion, "lung pain"/none (PCR)	Normal	Troponin I <0.01	LVEF 65%, no WMAs, no pericardial effusion	LVEF 59%, T1 abnormal ECV fractions, LGE present in inferolateral (basal), inferior (basal) segments, T2 normal mapping, trivial pericardial effusion	56	Possible

(Continued)

Table 2. Continued

Athlete number	Sex	Race/ethnicity	Past medical history	Symptom severity/duration, d: initial symptoms/exertional symptoms (SARS-CoV-2 test)	ECG	Troponin, ng/mL	TTE	CMR	Days from symptoms to CMR*	Certainty of involvement
Athletes diagnosed with myopericardial involvement of SARS-CoV-2 (n=6)										
12	F	Hispanic	None	Cardiopulmonary/14 to 20: cough, fever, SOB/NA (PCR)	Normal	Troponin <0.01	Abnormal: stress, TTE, exercise-induced WMA, basal, midinferior wall, small pericardial effusion	LVEF 54%, increase of native T1, no LGE, increase of T2 mapping, small pericardial effusion	Unknown	Definite
13	F	White	NA	Cardiopulmonary/9: chest pain, loss of taste/smell, myalgias/exertional CP (PCR)	Abnormal: prolonged QTc (481 ms)	Troponin I <0.01	LVEF 65%, no WMAs, no pericardial effusion	LVEF 45%, T1 LGE in the inferior (mid) segment, T2 associated edema present in this segment on FIESTA imaging, small pericardial effusion	123	Definite
14	F	White	Pre-HTN, HLD	Moderate/2 to 3: headache, loss of taste/smell, myalgias, pinkeye/NA (PCR)	Normal	Troponin T <0.01	LVEF 60%, no WMAs, trivial pericardial effusion	LVEF 62.5%, abnormal T1 mapping with LGE in antero-septal (basal), inferior (basal), inferolateral (basal) segments, no comment on T2 mapping, small pericardial effusion	18	Probable
15	M	Black	None	Moderate/3 to 4: headache, myalgias/NA (PCR)	Abnormal: Black athlete repolarization pattern, TWI V5 to V6, RAE	hsTroponin <6	Not performed	LVEF 60%, T1 normal mapping, LGE of the anterolateral (mid, apical) segments, T2 normal/no evidence of edema, small pericardial effusion	31	Probable
16	F	White	None	Asymptomatic: none (PCR)	Normal	Troponin I <0.01	LVEF 60%, no WMAs, trivial pericardial effusion	LVEF 55%, T1 abnormal ECV fractions in LGE areas of inferior (basal, mid), inferoseptal (basal), inferolateral (basal) segments, T2 mapping upper limits of normal, small pericardial effusion	41	Probable
17	F	White	None	Moderate/2: chills, fever, headache, loss of taste/smell, myalgias/none (PCR)	Normal	Troponin I <0.03	LVEF 60% to 65%, no WMAs, trivial pericardial effusion	LVEF 58%, T1 mapping normal, LGE present in inferolateral (mid) and anterolateral (mid) segments, T2 mapping normal, small pericardial effusion	22	Probable
Athletes diagnosed with isolated acute pericardial involvement of SARS-CoV-2 (n=4)										
18	M	White	None	Mild/4 to 7: headache, loss of taste/smell/none (PCR)	Normal	hsTroponin I <6	Abnormal: LVEF not reported, WMA not reported, small pericardial effusion	LVEF "normal," no myocardial inflammation, fibrosis, or edema present, Medium-sized pericardial effusion	20	Definite
19	F	White	None	Cardiopulmonary/4 to 7: nasal congestion, fatigue, sore throat, myalgias/exertional CP, DOE (PCR)	Normal	Troponin <0.01	LVEF 60% to 65%, no WMAs, no pericardial effusion	LVEF 65%, T1 no LGE present, T2 normal mapping without edema, small pericardial effusion	71	Definite
20	F	Hispanic	None	Asymptomatic: none (PCR)	Normal	hsTroponin 21	Not performed	LVEF 67%, T1 normal mapping, no LGE present, T2 normal mapping, small pericardial effusion	26	Definite
21	F	White	None	Cardiopulmonary/5: cough, fatigue, sore throat/exertional CP (antigen)	Normal	Troponin I <0.01	Abnormal: LVEF 55% to 60%, no WMAs, small pericardial effusion	LVEF 58%, T1 normal mapping, no LGE present, T2 normal mapping, small pericardial effusion	83	Definite

The mean age was 21±0.4 years (range, 17 to 24), with athletes from sporting disciplines including baseball, basketball, cheerleading, cross-country/track and field, football, lacrosse, soccer, softball, tennis, and volleyball. ADHD indicates attention-deficit/hyperactivity disorder; c/w, consistent with; CMR, cardiac magnetic resonance imaging; CP, chest pain; DOE, dyspnea on exertion; ECV, extracellular volume fraction; *FIESTA*, fast imaging employing steady-state acquisition; HLD, hyperlipidemia; hs, high sensitivity; HTN, hypertension; *LAE*, left atrial enlargement; LGE, late gadolinium enhancement; LV, left ventricular; LVEF, left ventricular ejection fraction; NA, not applicable; PCR, polymerase chain reaction; *RAE*, right atrial enlargement; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; SOB, shortness of breath; T1D, type 1 diabetes; TTE, transthoracic echocardiography; TWI, T-wave inversion; and WMA, wall motion abnormality.

*If the patient was asymptomatic, time is listed as the time from positive SARS-CoV-2 testing to CMR.

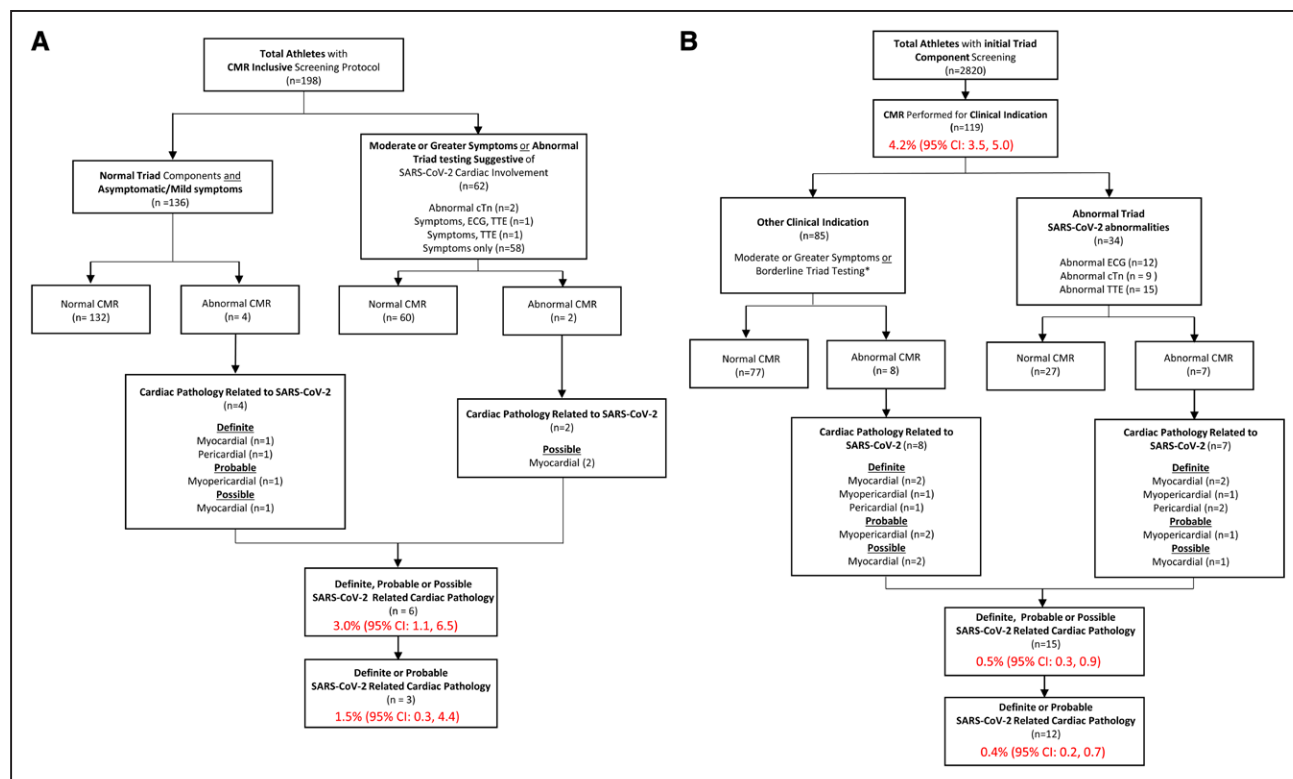


Figure 3. Results of primary CMR versus clinically indicated CMR screening protocols.

A, Athletes undergoing primary CMR screening protocol. **B**, Athletes undergoing clinically indicated CMR screening protocol. *Borderline triad testing as indication for CMR included detectable troponin level but not >99th percentile, nonspecific ECG abnormalities not fulfilling international criteria, and transthoracic echocardiogram (TTE) findings including abnormalities of unclear significance or those at the upper or lower limit of the normal range and considered to potentially represent pathology (ie, left ventricular ejection fraction 50%). CMR indicates cardiac magnetic resonance imaging; cTn, cardiac troponin; and SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

infection with the following key findings. First, cardiac evaluation conducted in accordance with current recommendations,^{5,7} which suggest risk stratification based on SARS-CoV-2 symptom burden to determine the role of triad testing and CMR, had a diagnostic yield for definite, probable, or possible cardiac involvement of 0.5% (95% CI, 0.3–0.9). Second, the prevalence of definite, probable, or possible SARS-CoV-2 cardiac involvement, derived from the first multicenter cohort of competitive athletes undergoing screening CMR, was 3.0% (95% CI, 1.1–6.5). Our data suggest these represent the lower and upper estimates (0.5% to 3.0%) of cardiac involvement after SARS-CoV-2 infection in young otherwise healthy individuals. Third, clinical predictors of SARS-CoV-2 cardiac involvement include cardiopulmonary symptoms during acute infection or on return to exercise and any abnormality on triad testing suggestive of SARS-CoV-2 cardiac involvement. In this cohort, a stepwise approach that used the presence of moderate severity or cardiopulmonary symptoms or any abnormal triad test to trigger a diagnostic CMR would have identified 9 of 11 (81.8%) athletes diagnosed with definite or probable myocardial or myopericardial involvement. Last, no athlete diagnosed with definite, probable, or possible cardiac involvement had an adverse cardiac event through the follow-up period.

The prevalence and clinical implications of cardiac sequelae after SARS-CoV-2 infection among people not requiring hospitalization have yet to be determined. Defining disease prevalence is of paramount importance to understand diagnostic testing performance and inform recommendations for population-based screening. Among a German cohort recovering from SARS-CoV-2 infection (mean age, 49 years; 33% hospitalized for symptoms of SARS-CoV-2 infection), 78% were reported to have cardiac involvement as defined by abnormalities on CMR.⁹ Recent single-center cross-sectional studies of collegiate athletes undergoing screening CMR after SARS-CoV-2 infection have documented highly variable prevalence estimates of cardiac involvement, ranging from 1.4% to 56%.^{10–13} The basis for this heterogeneity is multifactorial with contributions from variable study designs, limited sample sizes, and inconsistent definitions of cardiac involvement. Findings from the present study, leveraging multicenter CMR data, and the use of strict and conservative definitions that adhere to contemporary clinical imaging criteria suggest that SARS-CoV-2 cardiac involvement among collegiate athletes is less common than previously reported. This finding is similar to the prevalence rate of cardiac involvement recently

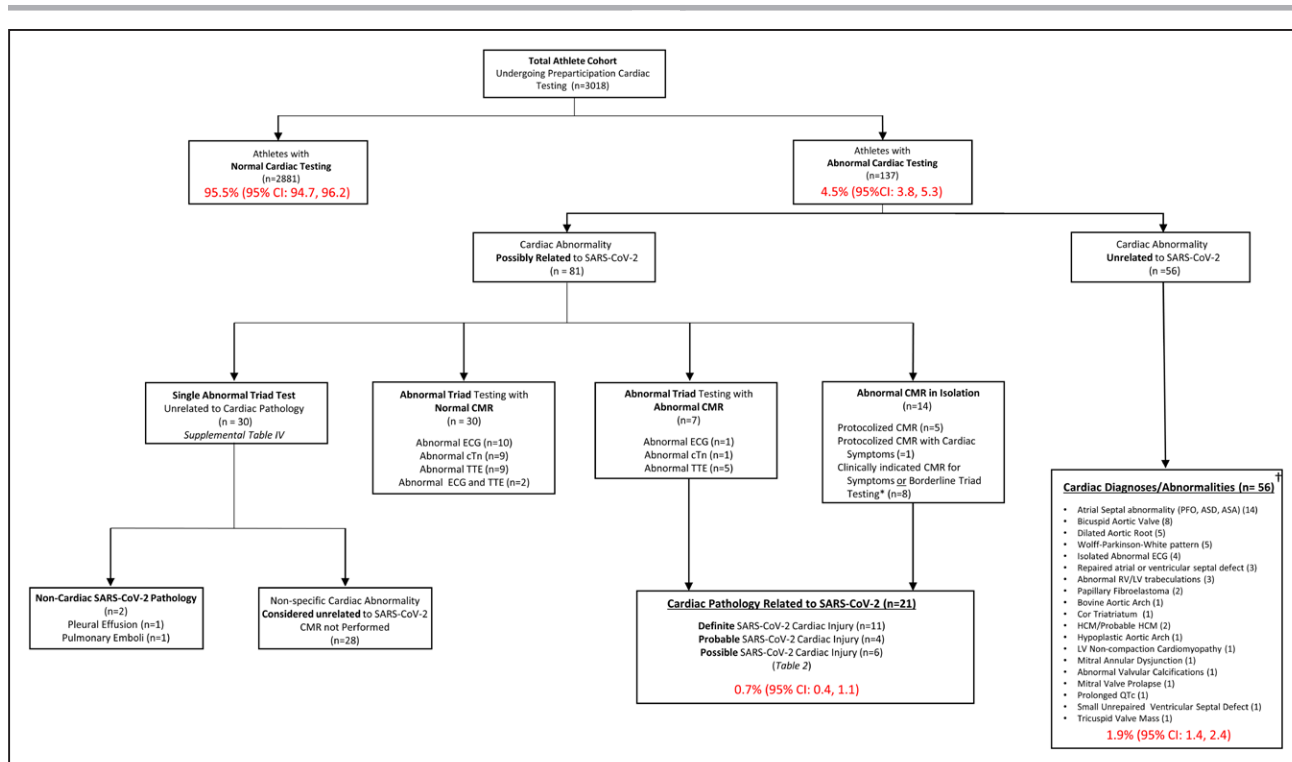


Figure 4. Cardiovascular testing results among NCAA athletes after SARS-CoV-2 infection.

*Borderline triad testing as indication for CMR included detectable troponin level but not >99th percentile, nonspecific ECG abnormalities not fulfilling international criteria, and TTE findings including abnormalities of unclear significance or those at the upper or lower limit of the normal range and considered to potentially represent pathology (ie, left ventricular ejection fraction 50%). †New and known diagnoses. ASA indicates atrial septal aneurysm; ASD, atrial septal defect; CMR, cardiac magnetic resonance imaging; cTn, cardiac troponin; HCM, hypertrophic cardiomyopathy; LV, left ventricular; NCAA, National Collegiate Athletic Association; PFO, patent foramen ovale; RV, right ventricular; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; and TTE, transthoracic echocardiogram.

reported among professional athletes (0.6%) who were assessed using triad testing followed by clinically indicated CMR.¹⁴

CMR has emerged as the noninvasive reference standard for confirming myocarditis among patients with disease-specific symptoms or other cardiac testing suggestive of this diagnosis. Numerous factors including the paucity of normative CMR data in athletes, interreader interpretation variability, financial cost, and access to testing underlie uncertainties about the use of CMR as a primary screening tool. Our data highlight important tradeoffs pertaining to use of CMR as a screening modality versus its use to confirm cardiac involvement. Among athletes undergoing clinically indicated CMR based on symptom burden or an abnormal triad test (n=119), 12.6% (95% CI, 7.2–17.9) were diagnosed with definite, probable, or possible cardiac involvement. This diagnostic yield was 4-fold higher than that observed when CMR was used as part of a primary screening protocol (6 of 198 [3.0%; 95% CI, 1.1–6.5]). However, 3 of 6 of athletes with definite, probable, or possible cardiac involvement undergoing screening CMR were asymptomatic or had mild symptoms and normal triad testing. CMR detected isolated late gadolinium enhancement or abnormal T1 data, age indeterminate markers of myo-

cardial fibrosis, or abnormal tissue architecture in a small number of athletes with no other features suggestive of acute SARS-CoV-2 cardiac involvement. Although we strongly suspect that these findings were unrelated to recent SARS-CoV-2 infection, we categorized these findings as “possible cardiac involvement,” underscoring the need to better understand their prevalence and clinical significance among young competitive athletes. In aggregate, these issues illustrate the inherent tradeoff between a CMR-inclusive screening protocol that may enhance sensitivity and a clinically driven approach that maximizes specificity.

Autopsy series have established myocarditis as an important cause of sudden death during exercise.^{2–4} Before the SARS-CoV-2 pandemic, myocarditis was routinely diagnosed among patients presenting with postviral cardiovascular symptoms, malignant arrhythmias, or myocardial dysfunction. Diagnostic CMR criteria and sport eligibility recommendations were developed on the basis of considerable experience with this clinical presentation.^{16–18} It remains unclear whether isolated cardiac abnormalities detected through CMR-based screening harbor a risk similar to typical clinically diagnosed myocarditis and how often these findings occur in other viral infections. Our SARS-CoV-2 cardiac involve-

ment prevalence estimate is similar to documented prevalence rates of myocarditis after influenza infection.¹⁹ Although many collegiate athletes in this cohort did not undergo CMR as part of their RTP screening algorithm, we observed no adverse cardiac events in this group. Whereas longer follow-up is of critical importance, our data suggest that the potential for subclinical cardiac involvement characterized by isolated CMR abnormalities is of low short-term clinical risk.

There are several limitations of this study. First, screening protocol heterogeneity across participating institutions may have affected our estimates of prevalence and diagnostic yield. Nonetheless, this study examines the largest number of athletes who have undergone some form of cardiac testing after SARS-CoV-2 infection (n=3018) and the largest multicenter CMR-inclusive screening cohort (n=198) of competitive athletes to date. Second, diagnostic testing results reported for both TTE and CMR were extracted from the primary clinical reports provided by participating institutions. Whereas this approach captures the actual clinical experience of our cohort, the unblinded clinical interpretation of the testing may have resulted in a degree of observer bias on the part of the interpreting physician. Unbiased imaging adjudication by a centralized core facility represents an important area of future work. Third, there is no uniformly accepted definition of cardiac involvement attributable to SARS-CoV-2 infection. We therefore used fundamental components of the Updated Lake Louise Imaging Criteria¹⁶ to generate de novo definitions of possible, probable, and definite cardiac involvement and presented full clinical profiles of athletes meeting these criteria. Future work is warranted to validate this approach. Last, we acknowledge the relatively short duration of our clinical follow-up and underscore the need for ongoing clinical surveillance of this cohort. However, the median of 113 days (interquartile range, 90–146) follow-up captured coincides with the period of highest risk of adverse events during exercise after acute myocardial injury.

Implications for RTP SARS-CoV-2 Cardiac Testing

Findings from this study provide an opportunity to assess and refine consensus recommendations for preparticipation cardiac screening of athletes after SARS-CoV-2 infection.^{5,7} Given the low prevalence of cardiac involvement and the absence of adverse events during short-term clinical surveillance related to SARS-CoV-2 infection among both collegiate and professional athletes,¹⁴ we propose the following considerations regarding future RTP cardiac testing after SARS-CoV-2 infection. First, our data support that athletes with asymptomatic or mild symptom burden from acute SARS-CoV-2 infection do not require additional cardiac testing before resumption of organized athletics. Second, triad test-

ing in the form of a 12-lead ECG, cardiac troponin, and TTE should be considered among athletes after acute SARS-CoV-2 infection who experience moderate systemic or cardiopulmonary symptoms, with the presence of cardiopulmonary symptoms representing a particularly important marker of risk. Last, our data suggest that the diagnostic yield of CMR will be optimized by confining its use to athletes presenting with increased clinical pretest probability of acquired myocardial pathology as defined by the presence of SARS-CoV-2–related cardiopulmonary symptoms or abnormalities on triad testing. These recommendations are generally consistent with the most recent RTP cardiac testing guidelines.

CONCLUSION

In a large prospective registry of collegiate athletes, SARS-CoV-2 infection occurred in 17.5% of athletes. Clinical evaluation revealed a low prevalence (0.5% to 3.0%) of definite, probable, or possible SARS-CoV-2 cardiac involvement and a low risk of adverse cardiac events during short-term follow-up. The clinical relevance of cardiac testing abnormalities after SARS-CoV-2 infection among athletes without other clinical features of myocardial involvement requires additional study. Future studies with extended clinical follow-up and appropriate control populations including athletes without SARS-CoV-2 infection are needed to better inform risk and the refinement of evidence-based screening strategies.

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Supplemental Materials

Methods

Data Supplement Figure 1

Data Supplement Tables I–VI

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