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Diagnosing COVID-19 myocarditis in athletes using cMRI

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ABSTRACT

An early report during the SARS-CoV-2 (COVID-19) outbreak noted myocardial involvement with cardiac troponin I (cTnI) levels >99th percentile in approximately 20% of hospitalized patients. Patients with cTnI elevations had higher in-hospital mortality. Additionally, myocarditis is associated with exercise-related sudden cardiac death in athletes. Therefore, reports of COVID-19 myocarditis concerned the sports cardiology community, which issued two guidelines on managing athletes with COVID-19 infection. We reviewed reports of myocardial involvement in athletes after COVID-19 infection published before June 2021.

The incidence of the diagnosis of myocarditis in athletes post-COVID-19 ranged from 0 to 15.4% based on cardiac magnetic resonance imaging (cMRI) performed 10 to 194 days after initial diagnosis of COVID-19. Only a few studies adhered to accepted myocarditis diagnostic guidelines and only two studies included a control group of uninfected athletes. There was significant heterogeneity in the method and protocols used in evaluating athletes post-COVID-19.

The incidence of COVID-19 myocarditis in athletes appears to be over-diagnosed. The evaluation of myocarditis post-COVID-19 should be individually performed and managed according to the current guidelines. This can potentially prevent needless training restrictions and the inability to participate in competitive sports.

Introduction

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2 or COVID-19) is a single-stranded RNA virus identified in Wuhan, China, on January 9, 2020. [1] It has infected more than 260 million and killed more than 5.2 million people worldwide as of Nov 28, 2021. [2] COVID-19 is the third known severe acute respiratory syndrome (SARS) coronavirus. [3] The first SARS-CoV occurred in 2002-2003 in Foshan, China. The second outbreak, Middle East Respiratory Syndrome (MERS) occurred in 2002 in the Arabian Peninsula. [4] SARS-CoV produced an exaggerated systemic inflammatory response with the production of IL-6 and IL-8 by virus-infected cells [5], resulting in direct and indirect injury to the cardiovascular system. The latest SARS version, COVID-19, can affect the heart and cause myocarditis, myocardial infarction, arrhythmias, and acute and chronic heart failure [6]. An early report in the COVID-19 epidemic found that 19.7% of hospitalized patients had cardiac injury defined as a highly sensitive troponin I (TnI) level >99th percentile. This group had a higher mortality rate of 51.2% compared to 4.5% in hospitalized patients without elevated TnI concentrations. [7] Based on a study, among 100 German COVID-19 patients, only 33% of whom had been hospitalized, 78% had increased myocardial native T1 mapping on cardiac magnetic resonance imaging (cMRI), and 60% had increased native T2 mapping consistent with diffuse myocardial fibrosis and/or edema. Furthermore, 32% had late gadolinium enhancement (LGE), and 22% had pericardial enhancement consistent with a myocardial scar and pericardial inflammation, respectively. [8]

Such findings raised concern in the sports cardiology community as in some registries; myocarditis is the third leading cause of sudden cardiac death (SCD) in athletes. [9] This concern prompted members of the American College of Cardiology's Sports and Exercise Cardiology Section to publish two return-to-play recommendations for evaluating athletes after COVID-19 infection prior to resumption of exercise. [10,11]

Here we review the criteria for diagnosing myocarditis, discuss possible cMRI differences between athletes and controls and review nine studies with cMRI results in athletes after COVID-19 infection published before June 2021. We conclude that available data have probably overestimated the frequency of myocardial involvement after COVID-19. Only a few studies adhered to accepted criteria for diagnosing myocarditis, included a control group, or interpreted the cMRI images blindly.

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Table 1
Published studies with cardiac screening post COVID-19 in athletes.

Study	Clark et al [12]	Rajpal et al [13]	Starekova Et al [14]	Brito et al [15]	Malek et al [16]	Martinez et al [17]	Moulson et al [20]	Hendrickson et al [18]	Daniels et al [19]
Design	Retro Obs	Prosp Obs	Retro Obs	Cross Obs	Retro Obs	Cross Obs	Prosp Obs	NA	Retro Obs
Study size (cMRI performed)	59 (59)	26 (26)	145 (145)	160 (48)	26 (26)	789 (30)	3018 (317)	137 (5)	1597 (1597)
Control group	60 military personal & athletes (h); 27 Healthy controls (i)	no	no	20 Athletes (cMRI not performed)	no	no	no	no	no
Blind read	no	no	no	no	no	no	no	no	no
Mean Age	20 (19-22)b	19.5 (1.5)a	19.6 (1.3)a	19 (18-21)b	24 (21-27)b	25 (3)a	20 (1)a	20 (18-27)c	NC
Mean Time to cMRI (SD or Median IQR)	21.5 (13-37)b	26 (10)a	15 (11-194)c	27 (22-33)c	32 (22-62)b	19 (17)a	33 (18-63)b	22 (11)a	22 (10-77)c
T1 Elevation No./total No. tested (%)	23/59 (39%). 8/60 (13%) (h), 2/27 (8%) (i)D	none	2/145 (1.3%)	9 (19%)	none	none	3/317 (0.9%)	NA	5/1597 (0.31%)
T2 Elevation, No./total No. tested (%)		4/26 (15.3%)	2/145 (1.35%)	0/48	4/26 (15%)	1/30 (3.3%)	7/317(2.2%)	0/5	31/1597 (1.9%)
LGE, No./total No. tested (%)	16/22 (27%) 10/27 (24%)(h)	12/26 (46.1%)	42/145 (28.9%)	1/48 (2%)	1/26 (4%)	2/30 (6.6%) with LGE	13/317 (4.1%)	0/5	36/1597 (2.2%)
Pericardial enhancement (x) No./total No. tested (%)	1/22 (4.5%)	2/4 (7.7%)	1/145 (0.6%)	19/48(39.5%)	1 (3.8%)	2/30 (6.6%)	10/317 (3.1%)	NA	1/1597 (0.06%)
Elevated troponin No./total No. tested (%)	none	none	5/145 (3.4%)	1/48 (2%)	4/26 (15%)e	12/789 (1.52%)	24/2719 (0.9%)	4/137 (2.9%)	6/1597 (0.37%)
Outcome in full cohort No/total No. tested (%)	2/59 (3.3%) myocarditis 1/59 (1.6%) pericarditis	4/26 (15.4%) myocarditis	2/145 (1.38%) myocarditis	0	No myocarditis	3/789 (0.38%) myocarditis; 2/789 (0.25%) pericarditis	15/3018 myocarditis (0.5%)f	No myocarditis	37/2461 myocarditis (1.5%)g

Abbreviations: cMRI, Cardiac magnetic resonance imaging; NA, not available or applicable; NC not collected; Ret, retrospective; cross, cross-sectional; pro, prospective; obs, observational
A: Mean (SD)
B: Median (interquartile range)
C: Median (range)
D: T1 Elevation, T2 Elevation and increase ECV reported together
E: High sensitivity troponin utilized
F: Reported as possible (n=6), probable (n=4) and definite (n=11) myocarditis
G: Reported as subclinical possible (n=20), subclinical probable (n=8), clinical myocarditis (n=9)
H: Military personal as athletic control group
I: Health control group

Methods

We searched PubMed for English language reports using combinations of the search terms myocarditis, myopericarditis, Post-Covid-19, Post-SARS COV-3, and athletes. Reports before June 2021 were reviewed, and those addressing COVID-19 and athletes were examined in detail.

Results

We identified nine reports of cMRI results in athletes being evaluated for possible COVID-19 myocarditis. [12,13,14,15,16,17,18,19,20] Four of these studies evaluated all COVID-19 infected athletes using cMRI prior to return to athletic activity. [12,13,14,16] The rest performed cMRI selectively based on one or more inclusion criteria (abnormal electrocardiogram (ECG), echocardiogram or troponin (cTn) or symptoms [15,17,18] or used a cardiac screening protocol). [20] cMRI was performed between 10[19] and 194[14] days after COVID-19 diagnosis. The study cohorts ranged from 26[13] to 3018[20] athletes. cMRI performed on 5[18] to 1597[19] athletes. Myocarditis was diagnosed in 0[15,16,18] to 15.38% [14] of the athletes. Pericardial effusion or enhancement was found in 0[19] to 58% [15] athletes. Two studies included either college students, as non-athlete controls, or military personal and college athletes as athletic controls [12,15], although only one study provided cMRI data on the control groups. [12] The two largest studies gathered data from 42 (n=3018 athletes) [20] and ten universities (n=2461 athletes) [19] but did not standardize the data collection techniques. cMRI was performed in 317 athletes [20] and 1597 athletes [19], respectively. All nine studies used different study designs (Table 1).

Discussion

Myocarditis

Myocarditis is most commonly secondary to viral infections. [21] Timely management of fulminant myocarditis carries a survival rate of >90%. However, the acute phase of myocarditis has a significant risk for malignant arrhythmia and SCD. [22,23] Myocarditis accounts for 9% of new idiopathic dilated cardiomyopathy worldwide. [24,25] Clinical suspicion is based on cardiac symptoms, abnormalities on the electrocardiogram, echocardiogram, or elevated troponin levels. Endomyocardial biopsy remains the gold standard for diagnosing myocarditis, but is used less frequently because cMRI is widely available and has less risk.

Lake Louise cMRI criteria for diagnosis of myocarditis

The 2009 Lake-Louise Criteria (LLC) established cMRI criteria for diagnosing myocarditis. LLC required at least two of the following three for the diagnosis: edema (T2 weighted ratio), hyperemia (early gadolinium enhancement), and necrosis/fibrosis (late

gadolinium enhancement). The LLC explicitly states that their application is limited to symptomatic patients with high clinical pretest probability. [26] The LLC does not address its use on asymptomatic patients. Expert recommendations from the American College of Cardiology and the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases require 2 of 4 criteria to diagnose myocarditis in asymptomatic patients. These diagnostic criteria include: ECG abnormalities, an elevated troponin, functional or structural abnormalities on echocardiography or cMRI tissue characterization demonstrating edema, hyperemia, or LGE. [27] cMRI criteria alone does not meet the guidelines for diagnosing myocarditis.

LLC updated in 2018 requires at least one T1 and one T2 criterion for diagnosing myocarditis. T1 criteria reflect myocardial inflammation and necrosis, whereas T2 criteria reflect myocardial edema, a sign of cellular injury. T1 criteria includes elevated myocardial relaxation times, increased extracellular volume fraction (ECV), and LGE. T2 criteria include elevated T2 myocardial relaxation times, T2 signal intensity ratio, and visible myocardial edema. 2018 LLC has a sensitivity of 87.5% and a specificity of 96.2% in symptomatic patients if accompanied by laboratory signs of inflammation (CRP mean ± SD 55.2 +/- 70.9) and injury (Troponin I (ng/ml) mean ± SD 13.9 +/- 37.4) and after excluding coronary artery disease. [28]

The sensitivity and specificity of the components of 2018 LLC are highly variable among individual studies, likely reflecting subjectivity in their assessment. A meta-analysis of 22 studies evaluating the performance of the individual LLC components reported a sensitivity and specificity for T1 mapping of 64-98% and 67-100%, T2 mapping of 57- 94% and 60- 92%, ECV of 67-94% and 56-90%, native T2 weighted of 45-100% and 43-100%, and LGE of 30-95% and 39-100% respectively. [29] Native T1 relaxation time achieved the highest diagnostic accuracy, although accuracy varied between 61% and 99%. [29] These wide ranges in results from the available studies are likely due to differences in cMRI timing from presentation, cMRI field strengths, and variabilities in thresholds. Interestingly, of all the cMRI findings, only LGE correlated with mortality or sudden cardiac death with a hazard ratio of 12.8%. [30]

cMRI and echocardiogram in athletes

The intensive exercise training program for athletes results in physiologic cardiac remodeling. [31] Endurance-trained athletes have enlargement of all four cardiac chambers to accommodate the higher rest and exercise stroke volumes. This adaptation usually occurs with mild left ventricular wall hypertrophy (LVH). Alternatively, primarily resistance exercise training produces LVH with minimal enlargement in chamber size. [32] Transiently elevated markers of cardiac injury and stress, including cardiac troponins T (cTnT) and I (cTnI), and brain natriuretic peptide (BNP) have been observed after an intense or prolonged exercise session. [33] From limited cMRI performed on athletes, LGE has been found in athletes primarily at the hinge points between the left and right ventricles. [16] LGE consistent with overt myocardial scarring found in 11.4% of 158 veteran athletes (VETS) (age mean ± SD 55± 8) with >40 years of competitive exercise compared to no scarring in 89 healthy volunteers (HV) (age mean 50 ± 13); among the VETS with scarring, 56% had a non-ischemic distribution. [34] There is a paucity of normative cMRI data for athletes. One study reported higher left ventricular native T1 values in athletes than non-athletes (1230.5 +/- 38.8 vs. 1174. +- 36.4; p<0.001). [35] ECV is lower in athletes than in untrained individuals (mean ± SD = 22.5 ± 2.65 vs. 24.5 ± 2.25; p=0.02), and athletes with high peak V02 values (>60ml/kg per min) had the lowest ECV. [36] Thus, the physiologic changes from exercise training can mimic laboratory and structural abnormalities used to diagnose myocarditis.

Post-COVID 19 myocarditis in athletes

The reports on cardiac effects of COVID-19 in athletes are difficult to compare due to differences in athlete selection criteria and cMRI protocols, although all studies utilize cMRI to evaluate myocardial involvement. Four studies performed cMRI on all COVID-19 positive athletes, [12,13,14,16] whereas the University of West Virginia study performed cMRI only on athletes with symptoms, echocardiographic or cTnI abnormalities. [15] A collaborative study from 42 universities included 3018 athletes. cMRI was performed in 198 athletes routinely and in 119 athletes because of symptoms or because of an abnormal echocardiogram, electrocardiogram, or cTnI. [20] An analysis of 789 professional athletes performed cMRI only in 30 athletes with ECG, echocardiogram, and cTnI abnormalities with or without symptoms. [17] All studies used adaptations of the LLC to diagnose cardiac involvement, suggesting that cMRI abnormalities indicate myocarditis. However, LLC is specifically designed to diagnose myocarditis only when there is a high pretest clinical suspicion. Only two studies included a control group. The Vanderbilt study included 60 “retrospectively selected” military personnel as “athlete” controls and 27 healthy adults as general controls. It is not clear when the cMRIs for the military personnel and other controls were interpreted. [12] Interestingly, among the COVID-19 infected athletes, 39% percent had T1, T2, or ECV cMRI abnormality, whereas 13% of the military personnel and 8% of the healthy controls had similar abnormalities. [12] More importantly, LGE was noted in 27% of infected athletes and 25% of military personnel. [12] This inclusion of an athlete control, even if retrospective, is an improvement over no controls. Nevertheless, all of these cMRI studies can be criticized because none used simultaneously recruited controls or read the cMRI’s blindly. cMRI interpretation is subjective and readers can overestimate myocardial involvement in patients referred with a COVID-19 diagnosis. Most studies of athletes reporting cardiac involvement found only mild abnormalities, specifically elevated T1and T2 values. These findings can be suggestive of myocarditis in the appropriate clinical setting, but elevated T1[35] and LGE [12,34] have been reported in asymptomatic athletes without COVID-19 infection. On the other hand, one study detected LGE in 26.2% of 145 athletes with COVID-19 but most of the LGE was at the RV/septal insertion site. [14] Another detected pericardial enhancement in 40% of 48 athletes with COVID-19 symptoms or an abnormal cardiac screening. [15] These findings are highly suggestive of myocarditis but higher than expected, so require confirmation. In contrast, the registry from ten universities, which included 2461 COVID-19 positive athletes of whom 1597 underwent cMRI, reported only 9 (0.32%) athletes with symptoms suggestive of myocarditis and concurrent cMRI changes satisfying LLC. [19]

Our interpretation of the literature linking COVID19 with myocardial involvement in athletes is that, it is impossible to determine the incidence of myocarditis in athletes after COVID-19 since none of the studies adhered to the LLC, included an appropriate control group, or blindly interpreted the cMRI scans. Future investigations should consider these measures in their study design to examine the risk of myocardial involvement in athletes with COVID-19. Exaggerated risk estimates significantly affect the athletic community with unnecessary restrictions on training and competitive sport, thus increasing the need for additional medical evaluations.

Approach to athletes with myocarditis

Making an unequivocal diagnosis of myocarditis in athletes is difficult because of physiologic changes of the ECG [37], echocardiogram [38], and cMRI that can occur with exercise training. Athletes with myocarditis should be restricted from exercise. Pharmacological management of myocarditis in athletes is based on ex-

pert opinion. Non-steroidal anti-inflammatory agents (NSAIDs) and colchicine are recommended for symptoms of chest pain and evidence of pericardial involvement. Some cardiologists use NSAIDs and/or colchicine in asymptomatic individuals with myocarditis for 4 to 6 weeks under the unproven assumption that these agents prevent ongoing inflammation and myocardial injury. [39] Others continue these agents longer if there is evidence of ongoing myocardial involvement by cardiac biomarkers. [39] The current European and American guidelines recommend 3-6 months of exercise restriction. These guidelines require a normal left ventricular ejection fraction (LVEF), cTnI, and 24-hour ECG monitoring before permitting a return to exercise and competitive sports. [40,41] Others permit a return to low-level exercise training after only one month if an echocardiogram, cMRI, and cardiac biomarkers are normal. [42] There are no data on "safe" levels of exercise in the 3-6 month abstinence period after the diagnosis of myocarditis. [43] There are also no clear guidelines on managing athletes with persistently abnormal cardiac biomarkers or cardiac imaging. We have recommended that such athletes continue to avoid maximal exercise training and effort, albeit also recognize that such caution may be unnecessarily restrictive. [39] Athletes without symptoms, abnormal cardiac biomarker or echocardiographic abnormalities, but who had an abnormal cMRI done for routine screening after COVID-19 do not meet the LLC for myocarditis. Therefore, they should be treated less restrictively than those with symptomatic myocarditis. Such athletes may not have myocardial involvement at all but simply cMRI abnormalities of uncertain significance. Maximal exercise testing and possibly prolonged rhythm monitoring should be considered in all athletes with any cardiac abnormalities after COVID-19 before return to full activity to evaluate if arrhythmias are present and frequent.

There are no data on management of athletes with myocarditis after COVID-19 immunization. [42] Most of these athletes are diagnosed with myocarditis because of symptoms so they should be treated as standard myocarditis with exercise restriction and anti-inflammatory medications as appropriate.

Ultimately, medical treatment of athletes and the timing to return to training and competition should be made on an individual basis because definitive data on this issue are not available. [42] In the interim, facilities where athletes train and compete should have action plans to deal with cardiac emergencies. [44] Also, coaches, trainers, and other athletes should be trained to recognize cardiac arrest and perform CPR. [44] Such preparation and training can have benefits far beyond the present epidemic.

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