Chapter 23
The Impact of COVID-19 on Sports Cardiology

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Background

The coronavirus disease-19 (COVID-19) is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and spread predominantly by respiratory droplets. COVID-19 garnered international attention as it rapidly developed into a global pandemic in early 2020 and its dramatic morbidity and mortality necessitated the implementation of public health measures designed to limit close contact and large public gatherings. To achieve that goal, organized sports from the recreational level to the Olympic Games were postponed, altered, or canceled altogether. As we gradually make progress in the fight against COVID-19 and our society focuses on the means to restore and restart all facets of normal life, including the resumption of organized athletics, sport and health organizations continue to face significant challenges designing and implementing safe return-to-play (RTP) strategies.

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Cardiac Considerations

Early data from critically ill and hospitalized COVID-19 patients demonstrate the common association between COVID-19 and myocardial injury, defined as a cardiac troponin level greater than the 99th percentile upper reference limit [1–4]. Several mechanisms of myocardial injury have been proposed and include direct viral myocardial injury, microvascular injury, cytokine and stress mediated cardiomyopathies, acute coronary syndromes, pulmonary emboli, and systemic hyperinflammatory responses [5, 6] (Fig. 23.1). SARS-CoV-2 is thought to enter the body through the angiotensin-converting enzyme 2 (ACE2) receptor, a receptor present in the lungs, myocardium, and on vascular endothelial cells [5, 7]. As such, direct viral myocardial injury is a potential mechanism for myocarditis and has been supported by autopsy data demonstrating viral presence, progeny, and shedding in cardiac tissue [6]. Other theoretical mechanisms of direct viral injury include infection-mediated vasculitis, as the ACE2 receptor is expressed in arterial and venous endothelial cells, or an indirect immunological response and resultant hypersensitivity reaction [5, 8]. Microvascular injury is also a proposed mechanism for

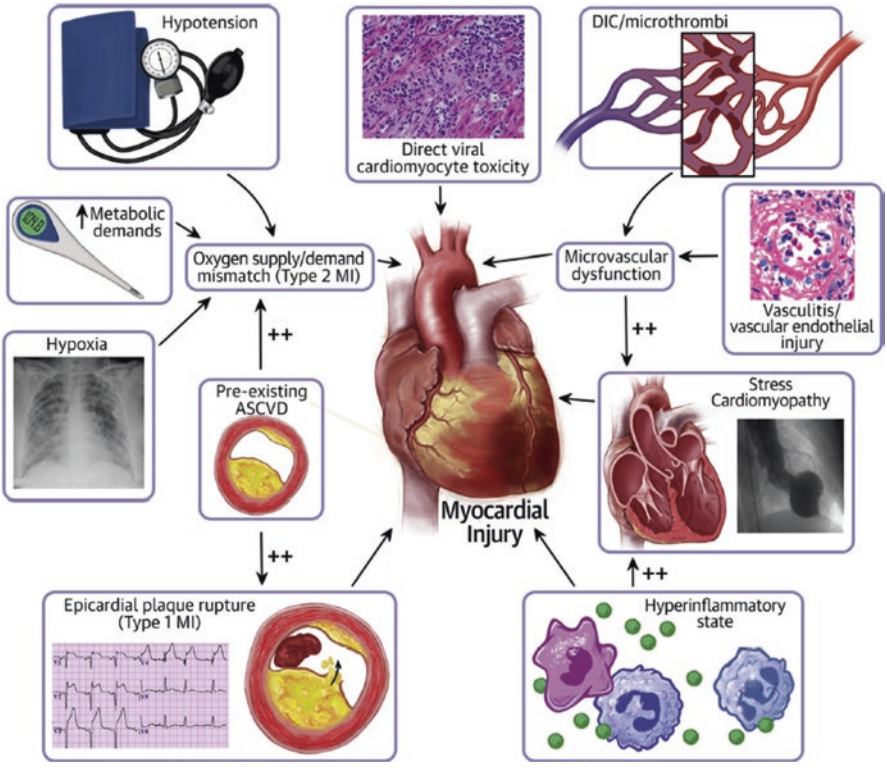

Fig. 23.1 Potential mechanisms of myocardial injury in COVID-19. (Reprinted with permission from Atri et al. [5] (Elsevier))

myocardial injury given the significant proportion of patients with severe COVID-19 meeting criteria for disseminated intravascular coagulation (DIC) [9]. Immune activation as a result of severe COVID-19 is thought to trigger DIC, microvascular dysfunction, and subsequent myocardial injury [5].

COVID-19 myocarditis has emerged as a critical issue in the discussions regarding RTP strategies for athletes [3, 10, 11]. Because myocarditis is a prominent etiology of sudden cardiac death (SCD) among athletes, accounting for approximately 4–7.5% of SCD in athletes [12], current guidelines addressing myocarditis recommend against competition or strenuous training for 3–6 months after diagnosis [12–16]. Recent studies of cardiac magnetic resonance imaging (CMR) in patients recovering from COVID-19 have demonstrated findings suggestive of residual myocardial inflammation, and as such, highlighted concerns about COVID-19-related myocarditis [17, 18]. Additionally, after several media reports of athletes diagnosed with presumed COVID-19 myocarditis, widely publicized discussions arose regarding the feasibility of continuing or resuming athletic training and competition in the midst of the pandemic [19].

While inflammatory heart disease, including pericarditis and myocarditis, poses an enhanced risk to athletes on resumption of training and competition, the true incidence and prevalence of COVID-19-related subclinical inflammatory heart disease, particularly among athletes who were asymptomatic or mildly symptomatic, remain unknown and existing data are limited to small observational case series [18, 20–22]. Current studies evaluating the incidence of myocardial injury in asymptomatic or mildly symptomatic athletes provide highly variable findings [18, 21, 22, 23]. Adding to the diagnostic difficulty in detecting potential subclinical COVID-19 cardiac injury in athletes is the fact that certain abnormalities described in association with myocarditis, such as troponin elevation, ECG abnormalities and imaging findings including increased left ventricular (LV) wall thickness, increased chamber size, and mild reduction in ventricular ejection fraction, can be seen as attributes of the athlete's heart [24–26].

Recognizing these challenges, the American College of Cardiology Sports and Exercise Cardiology Council, and other organizations, generated RTP recommendations for athletes recovering from COVID-19 [10, 20, 27–31]. While many athletes will be asymptomatic or minimally symptomatic, some may have experienced more pronounced viral symptoms such as prolonged fever with myalgias, chest pain, reduced exercise tolerance, or shortness of breath. Ascertained symptom burden should guide the next steps in the evaluation of the athlete, with an assumption of a correlation of potential risk of cardiac sequelae of COVID-19 with the severity of initial COVID-19 viral illness [31]. Although there has been some variation between published RTP cardiovascular screening recommendations, a conservative approach consisting of the combination of an electrocardiogram (ECG), transthoracic echocardiogram (TTE), and cardiac biomarker evaluation (troponin evaluation) was initially put forth by the ACC Sports and Exercise Cardiology Council in May 2020 for all athletes with prior mild to severe COVID-19 viral illness [10, 20, 27–29, 31, 32]. RTP without additional cardiovascular risk stratification was deemed reasonable in asymptomatic athletes who test positive for COVID-19, as long as

clinical observation is available and there is stepwise and pragmatic training intensification [31].

The decision to pursue additional downstream testing, including CMR, should be based on concerns raised on the initial screening exams. The widespread use of advanced imaging methods, such as CMR, as part of the initial pre-participation screening process of athletes has not been recommended. There remain valid concerns that increased testing will lead to increased sensitivity, but will invariably decrease specificity for detecting clinically relevant cardiac pathology, especially given the challenges in distinguishing potential COVID-19 cardiac pathology from adaptive remodeling in athletes [31]. The initial RTP recommendations were put forth with the understanding that the screening approach will evolve as more data on the prevalence of COVID-19 cardiac involvement and the diagnostic performance of screening measures become available.

Practical Use of Cardiac Testing for COVID-19 Myocarditis

In the assessment for potential COVID-19 cardiac pathology, it is important to understand the strengths and limitations of the cardiac tests currently recommended in RTP cardiac screening algorithms. Appropriate use and interpretation of the results will help enhance detection of disease and athlete protection while minimizing false-positive results that could adversely affect athletes by causing unnecessary delays in RTP or disqualification. The four tests that have been addressed in the most detail in the evaluation of athletes in the RTP screening algorithm include cardiac biomarkers (troponin), ECG, echocardiography, and CMR.

Troponin

Several COVID-19 RTP documents have recommended measuring high-sensitivity cardiac troponin (hs-cTn) levels to assess for the biochemical presence of myocardial injury and diagnose subclinical myocarditis [20, 27, 31, 32]. However, strenuous exercise may also cause an elevation in troponin that peaks and returns to baseline approximately 24–48 hours after exercise [26, 31, 33, 34]. As such, hs-cTn testing should not be done within this timeframe and testing should be repeated following an isolated abnormal result [10]. Persistently elevated troponin levels should prompt characterization of the myocardium with echocardiography and CMR [10]. It is important to note that the data linking elevated hs-cTn to worse outcomes in COVID-19 was derived from hospitalized patients. The full implications of elevated hs-cTn among younger, asymptomatic, or mildly symptomatic athletes is currently not known [10, 35]. Because there are no established reference ranges for hs-cTn in athletes, results need to be incorporated with other clinical data obtained in the screening process to properly interpret and act on the result.

Electrocardiography

The 12-lead ECG is simple, inexpensive, and useful in detecting conditions associated with SCD. While myocarditis, or myopericarditis, may manifest on an ECG in the form of premature ventricular contractions, arrhythmias, ST and T wave abnormalities, pseudoinfarction pattern (Q waves and ST elevation), bundle branch and atrioventricular (AV) blocks, the sensitivity of the ECG for detecting myopericarditis remains less than 50% [31, 36]. Moreover, many of the physiological, adaptive electrical changes commonly seen on athletes' ECGs, such as repolarization abnormalities and tall T waves, could be misinterpreted as myopericarditis [31] (Fig. 23.2). The specificity of ECG changes for diagnosing myocarditis is expected to be low but comparison with previous ECGs is of paramount importance [31].

Echocardiography

Given their relative accessibility compared with other forms of advanced cardiac imaging, and excellent diagnostic capabilities, echocardiograms have been recommended as first-line imaging exams in several RTP strategies for symptomatic COVID-19-positive athletes [27–29, 31, 32]. The presence of left or right ventricular systolic or diastolic dysfunction, or more than a trivial pericardial effusion, are important findings on echocardiography, especially if the findings are new in comparison with available prior studies. These abnormalities should prompt consideration of additional imaging to exclude COVID-19-related inflammatory heart disease. Occasionally, elite endurance athletes may demonstrate a low normal to mildly reduced resting left and right ventricular systolic function [37–39]. In this

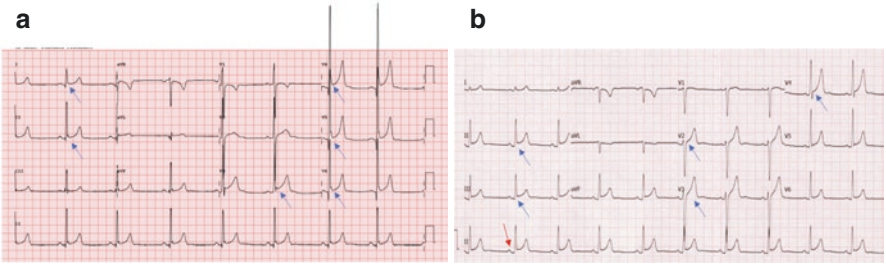

Fig. 23.2 Challenges with ECG screening in athletes post COVID-19. ECGs performed on two athletes highlighting challenges with differentiating normal changes associated with athletic training and pathology. Panel A is a normal healthy endurance athlete's ECG showing diffuse ST segment elevation due to early repolarization (arrows). Panel B is an ECG from a 23-year-old soccer player presenting with positional pleuritic chest pain, elevated high-sensitivity troponin (> 5000 ng/l) with confirmed myopericarditis on MRI; the ECG also shows diffuse ST segment elevation (blue arrows) but also subtle PR depression (red arrows). (Reprinted with permission from Phelan et al. [11], Elsevier)

instance, stress echocardiography demonstrating normal augmentation of left ventricular wall segments and normal exercise hemodynamics is a useful tool to help distinguish features of the athlete's heart from potential cardiac pathology. However, in the context of concern for COVID-19 myocarditis, this should only be undertaken after a CMR has ruled out active myocardial inflammation. Implementation of echocardiography may be limited by cost and access in areas of high disease prevalence [31].

Cardiac Magnetic Resonance Imaging

The important diagnostic role of CMR for the detection myocarditis and pericarditis in patients with high clinical suspicion is well established. CMR should only be performed during the acute illness if needed for immediate patient management decisions, otherwise, it should be performed >10 days after initial diagnosis in order to limit exposure to hospital and MRI staff [31, 40]. Some small CMR-specific studies demonstrated a significant prevalence of cardiac involvement in patients who recovered from COVID-19. In a cohort of 26 patients in Wuhan, China, recovering from moderate to severe COVID-19 pneumonia, 31% had late gadolinium enhancement (LGE), and there were additional markers of myocardial inflammation in a high prevalence of patients including increased global native T1, T2, and extracellular volume [41]. Puntmann et al. studied a German cohort of 100 middle-age patients recovering from COVID-19 illness and reported that 78% of this cohort had evidence of myocardial injury on CMR (median 71 days post-diagnosis) [17]. Importantly, the cohort had a mean (SD) age of 49 (14) years and a clinically significant burden of preexisting conditions (hypertension 22%; diabetes 18%; intrinsic lung disease 21%), and 36% had ongoing symptoms at the time of CMR. It is evident that this cohort may not be generalizable to younger, healthier athletes. Notably, a revised manuscript was ultimately published because of inaccurate data analysis and inconsistencies within the data.

Athlete-specific CMR studies have reported highly variable rates of cardiac abnormalities in athletes who recovered from COVID-19. In one single-center study of 26 athletes (mean age 19) with prior asymptomatic or mildly symptomatic COVID-19 illness, and with normal ECG, hs-cTn, and echocardiogram, CMR demonstrated that 46% of these athletes had LGE and 15% had CMR findings suggestive of myocarditis [18]. Another observational case series of 46 collegiate athletes (mean age 19) who recovered from COVID-19 and underwent a screening CMR demonstrated 41% of athletes had pericardial hyperenhancement, suggesting pericarditis. Only 1 athlete had myocardial LGE and no athletes had abnormal native T2 values [22]. In contrast to these studies, a third CMR study evaluating 12 Hungarian athletes recovering from COVID-19 (median age 23) showed no evidence of myocardial or injury [21], and a CMR-based study of 145 collegiate athletes recovered from COVID-19 showed that the prevalence of detected myocarditis was 1.4% [43].

Given the variability associated with the results of these three small-cohort studies, it is clear that large, multicenter, controlled, and blinded CMR studies are needed.

At present, there are insufficient data to recommend CMR for all athletes with confirmed or suspected COVID-19 or for those without clinical suspicion for myocarditis [10]. As learned during the introduction of ECG screening for athletes, widespread CMR-based screening without standardized measurements and normative data may lead to high false-positive rates, unnecessary subsequent testing, and needless medical disqualifications [10, 42].

Clinical Experience

Clinical experience gained in 2020 through widespread performance of COVID-19 RTP cardiac testing and gradual return of organized sports has fortunately yielded very few cases of relevant cardiac pathology in young athletes thus far. The major US professional sporting leagues were among the first sporting organizations to return to full-scale sport activity in the setting of the COVID-19 pandemic, with provision of extensive health and safety measures as recommended by public health, infectious disease, and cardiac consultants. A program for pre-participation RTP cardiac testing, in alignment with the initial May 2020 ACC recommendations, was implemented by each of these leagues for all athletes that tested positive for COVID-19. A study of the collective results of the systematic cardiac RTP COVID-19 screening program utilized by these professional leagues demonstrated that the prevalence of clincally detectable inflammatory heart disease in professional athletes who underwent RTP cardiac screening was 0.6% [43]. Safe return to professional sporting activity has been achieved thus far with no cardiovascular events occurring within these professional leagues during and on completion of competitive play in 2020. The implementation of RTP cardiac screening by the professional leagues has provided a large-scale practical paradigm demonstrating the clinical efficacy of the ACC expert consensus-generated screening recommendations in achieving safe return to intensive sport activity. In parallel to the experience with professional athletes, a registry of National Collegiate Athletic Association (NCAA) athletes is planned to assess the impact of COVID-19 on cardiovascular pathology and the risk of myocardial injury for collegiate athletes afflicted with COVID-19.

Updated Expert Consensus Recommendations

Based on the data and clinical experience generated after publication and practice of the initial RTP screening algorithm, members of the American College of Cardiology's Sports and Exercise Cardiology Council published updated RTP screening recommendations in October 2020. These recommendations do not

advocate for cardiovascular (CV) risk stratification among athletes with prior asymptomatic or mild COVID-19 viral illness (defined as nonspecific and self-limited fatigue, anosmia or ageusia, nausea, vomiting, diarrhea, headache, cough, sore throat, and nasopharyngeal congestion), who remain asymptomatic after completion of appropriate self-isolation [10].

For high school athletes younger than 15 years recovering from moderate to severe COVID-19 illness (defined as persistent fever [temperature 100.4 °F] or chills, myalgias, severe lethargy, and hypoxia or pneumonia and/or CV symptoms such as dyspnea and chest pain, tightness, or pressure at rest or during exertion), evaluation by a pediatrician or pediatric cardiologist has been recommended to determine the need for further CV risk stratification [10]. For high school athletes older than 15 years following asymptomatic to mildly symptomatic COVID-19 illness, the updated ACC recommendations do not advocate for CV risk stratification. However, for high school athletes who had systemic or CV specific symptoms, a similar approach to symptomatic older athletes is recommended [10].

For masters-level athletes, routine RTP CV assessment is not recommended considering the logistics required for widespread screening and the low risk of clinically significant cardiac injury after mild infection. However, risk stratification may benefit master's athletes older than 65 years, particularly individuals with preexisting CV disease and those with moderate to severe prior COVID-19 infection (Fig. 23.3).

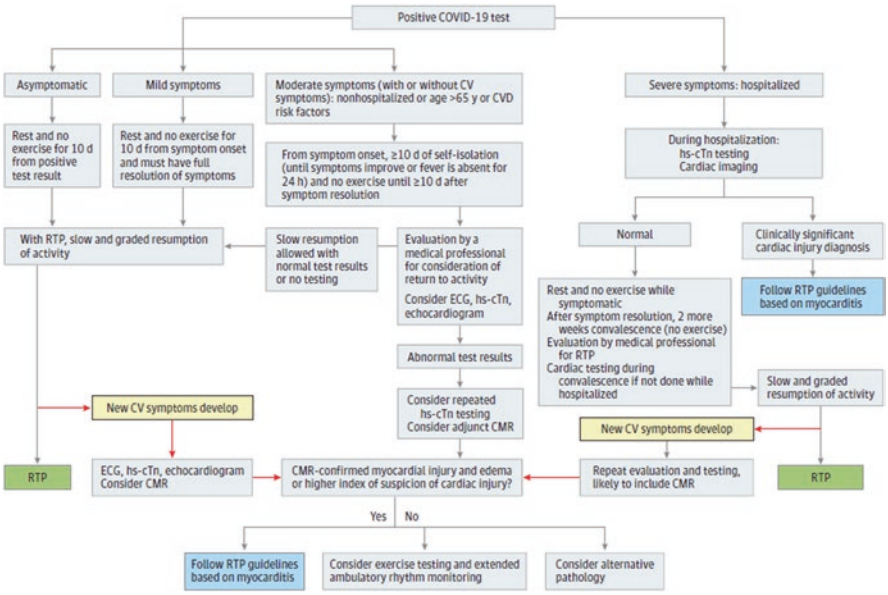

Fig. 23.3 American College of Cardiology October 2020 COVID-19 Return-to-Play Algorithm for recreational masters athletes. (Reprinted with permission from Kim et al. [10], American Medical Association)

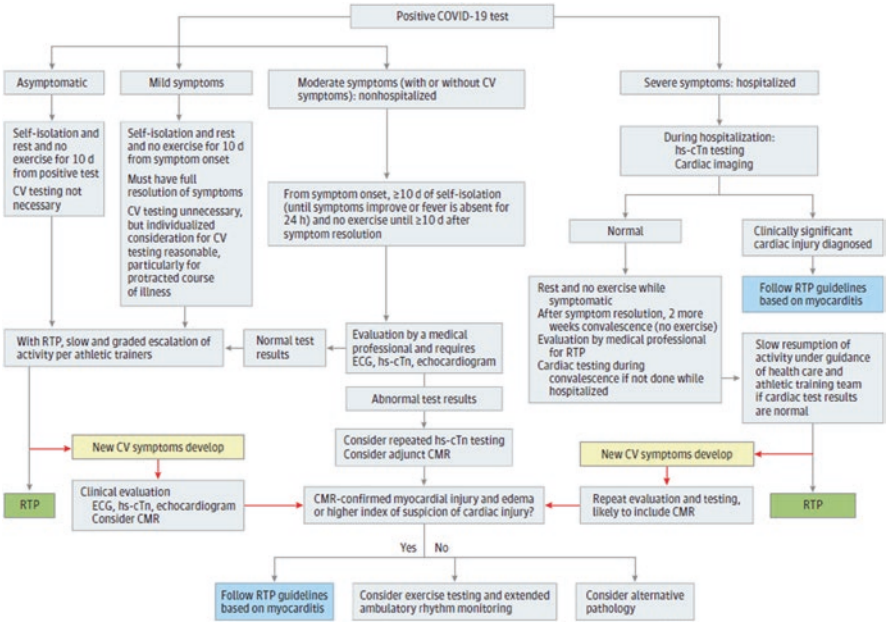

Fig. 23.4 American College of Cardiology October 2020 COVID-19 Return-to-Play Algorithm for adult athletes in competitive sports. (Reprinted with permission from Kim et al. [10], American Medical Association)

In line with the Centers for Disease Control and Prevention (CDC) recommendation to reduce self-isolation from 14 to 10 days from the time of documented infection, the updated RTP algorithm reduced the time period recommended for complete exercise abstinence from 14 days to 10 days from the date of the positive test result for asymptomatic COVID-19 infection [10]. A graded resumption of exercise remains recommended after this 10 day period. Furthermore, given the available data suggesting that RTP CV risk stratification appears to be low yield in competitive athletes with mild, self-limited disease, competitive athletes with mild COVID-19 illness may resume training in a graded fashion without pre-participation RTP testing after 10 days of symptom resolution. However, CV risk stratification is appropriate for competitive athletes with prior moderate or severe COVID-19 illness [10] (Fig. 23.4).

Conclusions

The sporting world has been heavily impacted by the COVID-19 pandemic. Concerns of potential COVID-19 cardiac pathology have driven the formation of expert consensus RTP algorithms designed to protect the athletic heart. While early experience suggests that implementation of targeted cardiac screening can promote

safe return to sport, the collection of large-scale and prospective clinical and imaging data is essential to enhance our understanding of the short- and long-term cardiac sequelae of COVID-19, and to provide data-driven approaches to the counseling and screening of competitive athletes and highly active people. The inclusion of sports cardiologists with expertise in the performance and interpretation of athlete cardiac testing will remain essential in the COVID-19 RTP process to optimize and streamline downstream testing and to minimize the potential for unnecessary disqualification or delays in return to play.

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