




Return to play after myocarditis: time to abandon the one-size-fits-all approach?

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MYOCARDITIS AND THE RISKS IN ATHLETES

During the COVID-19 pandemic, physicians around the world were confronted with concerns that myocarditis, a non-ischaemic inflammatory disease of the myocardium, could occur more frequently following SARS-CoV-2 infection than after other respiratory viruses. This concern was particularly pertinent for athletes because exercise continuation during acute myocarditis may increase the severity of myocardial damage¹ and act as a trigger of lethal arrhythmias and sudden cardiac death in individuals with myocardial inflammation and scar. Despite the recognition that sports should be avoided during acute myocarditis, the timing of exercise resumption in the postacute phase, when symptoms have resolved, is almost entirely dependent on expert consensus.^{2–4} This commentary examines the rationale underpinning current return-to-play (RTP) recommendations and proposes a more individualised approach.

THE DIAGNOSIS OF MYOCARDITIS AND THE CHALLENGE OF SUBCLINICAL DISEASE

Cardiac MR (CMR) imaging is the most useful tool for diagnosing myocarditis and has excellent sensitivity for detecting myocardial inflammatory alterations.⁵ However, the clinical context is critically important because CMR diagnostic criteria for myocarditis were generated in the setting of patients presenting with signs and symptoms suggestive of myocarditis. In the COVID-19 era,

isolated CMR findings meeting criteria for myocarditis have been detected with increasing frequency due to RTP screening programmes in athletes following SARS-CoV-2 infection, but there is limited evidence on the risks and prognosis of these CMR features in the absence of symptoms.⁶ Data from the Big Ten COVID-19 Cardiac Registry for detecting COVID-19-related cardiac involvement reported that 3% of athletes were suspected to have myocarditis of which 20 of 37 athletes had subclinical disease based on CMR findings alone (ie, in the absence of cardiac symptoms, elevated troponin level and/or abnormal ECG or echocardiogram findings).⁷ Should these individuals be managed in a similar fashion as those with clinically overt myocarditis and refrain from moderate-intensity to high-intensity exercise for a period of 3–6 months?^{2,3} The scale of the COVID-19 pandemic has highlighted this issue, but it is worth noting that myocarditis is a recognised postinfectious complication from other pathogens and the relative risk of various infectious agents is not well established. Thus, based on previous experience, CMR screening of asymptomatic athletes should be discouraged in the absence of supportive signs or symptoms suggesting myocarditis.⁸ Hence, the term ‘subclinical myocarditis’ should not be used as current RTP recommendations apply only in the appropriate clinical context.

A CRITICAL LOOK AT CURRENT RTP RECOMMENDATIONS

The rationale underpinning current European Society of Cardiology and American Heart Association (ESC/AHA) RTP recommendations^{2,3} following myocarditis is fuelled by two important observations: (1) acute myocarditis is an important cause of sudden cardiac death in young athletes and (2) exercise continuation during acute myocarditis markedly increases the severity of myocardial inflammation and necrosis in animal models.¹ These considerations have led experts to prudently advise that people should avoid vigorous exercise and be excluded from competitive

sport for at least 3 months following a diagnosis of myocarditis, irrespective of age, sex or the initial severity of disease. There are no studies that have assessed the incidence of myocarditis complications, their relationship to exercise or comparisons with recovery time. Given that the expected rate of serious complications is very small, such evidence is likely to remain elusive.

COULD CMR REFINE CURRENT RTP RECOMMENDATIONS?

In the current era of personalised medicine, a more individualised approach that recognises the heterogeneity of myocarditis presentations seems desirable. Although there is a paucity of evidence regarding the incidence of complications, there are some surrogates of disease activity that may provide guidance. Inflammatory alterations of the myocardium reveal a rapid and consistent decrease in a time frame that is shorter than the restriction period of 3 months.⁹ T1 and T2 relaxation times are the only single parameters of ongoing myocardial inflammation/oedema to show significant differences between patients with myocarditis and control subjects at 4–8 weeks follow-up examinations.⁹ Although these differences in T1 and T2 relaxation times increase the sensitivity for diagnosing subtle cases of acute myocarditis, their value for predicting clinical events in the convalescence phase has not been investigated. Thus, CMR criteria by itself, although useful for early diagnosis in symptomatic patients, are insufficient to guide clinical decision-making and should always be considered in the appropriate clinical context.

TOWARDS AN INDIVIDUALISED AND INTEGRATED APPROACH

As acknowledged in current ESC/AHA guidelines,^{2,3} even in athletes with persisting myocardial scar in the postacute phase, the RTP decision-making comes down to the exclusion of cardiac dysfunction and complex ventricular arrhythmias. Thus, according to guidelines, the athlete with significant illness, substantial scar (>5% of myocardial mass)¹⁰ and recovered left ventricular (LV) function and the athlete with transient, self-limited chest pain receive the same recommendation of exclusion from competition for 3 months. One might speculate that in this second scenario, the absence of scar, ventricular arrhythmias and LV dysfunction could be sufficient to enable RTP prior to 3 months. In figure 1, we propose a more individualised approach for RTP in which

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Individualised approach for return-to-play after ACUTE MYOCARDITIS

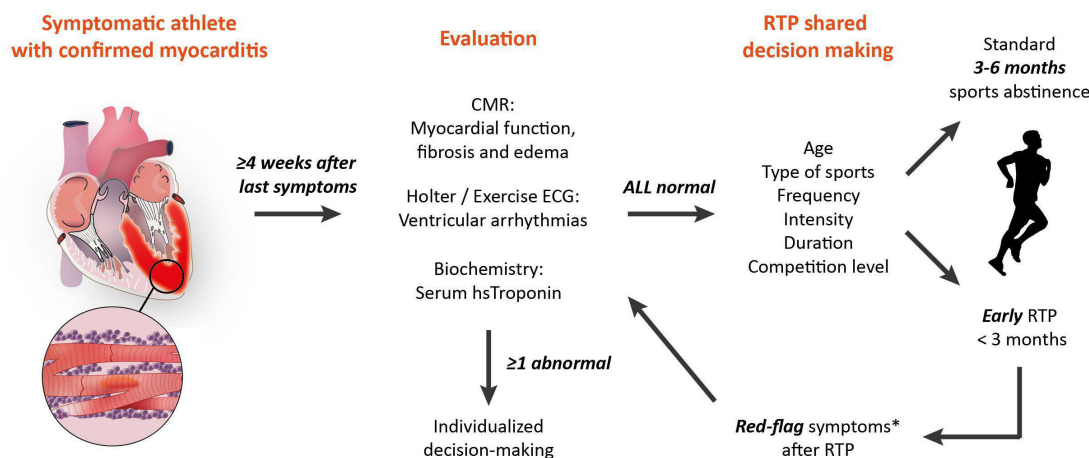


Figure 1 Illustration of an individualised and integrated approach that may be useful for risk stratification and guidance on timing of return to sport after acute myocarditis. CMR, cardiac MR; RTP, return to play. *Red-flag symptoms include exertional chest pain, unexplained dyspnoea, syncope and palpitations.

repeat testing may be performed 4 weeks after disappearance of clinical signs and symptoms related to myocarditis. We recognise that the 4-week interval—after resolution (not onset) of symptoms—remains arbitrary, but reasonable given previous CMR data on the evolution of myocardial inflammation/oedema in the postacute phase. At this time interval, subtle differences in T1 and T2 relaxation times may still be present,⁹ but there are no data indicating that these findings (in isolation) present a higher arrhythmic risk. While awaiting more scientific evidence to confirm the appropriate timing of RTP following acute myocarditis, decision-making should rely most on the presence or absence of cardiac dysfunction, myocardial scar by CMR and complex ventricular arrhythmias during exercise or Holter monitoring. In the authors' view, individuals without symptoms or identifiable risk factors at least 4 weeks after resolution of symptoms may be considered for early RTP after careful individualised evaluation, discussion regarding theoretical risks and awareness of 'red-flag' symptoms (ie, exertional chest pain, unexplained dyspnoea, syncope, palpitations) that would prompt reassessment. This evaluation for early RTP ideally includes a contrast-enhanced CMR, exercise ECG test and serum troponin. A structured reconditioning programme is also recommended prior to full RTP, including supervised training with progressive workloads and intensity and monitoring for new cardiovascular symptoms.

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