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Myocarditis in athletes: Challenges for return to play

Miocardite em Atletas: Desafios para o Retorno à Prática Desportiva

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Resumo

A miocardite é uma condição caracterizada por etiologias, manifestações clínicas e história natural heterogéneas. O diagnóstico da miocardite permanece desafiador e requer uma abordagem multidisciplinar que integre avaliação clínica, biomarcadores, eletrocardiografia, ecocardiografia e técnicas de imagem mais avançadas. A ressonância

magnética cardíaca emergiu como o exame *gold-standard*, que, juntamente com a biópsia endomiocárdica, constitui a base da atual classificação diagnóstica. A miocardite é também uma causa relevante de morte súbita cardíaca em indivíduos jovens, incluindo atletas. Nesse contexto, a avaliação da elegibilidade para o retorno à prática desportiva de atletas após uma miocardite aguda é complexa e está frequentemente limitada pela escassez de dados robustos baseados em evidência científica. A estratificação de risco, orientada por uma avaliação multiparamétrica abrangente e por um processo de tomada de decisão partilhada, que inclua os potenciais riscos associadas ao retorno do desporto competitivo, é fundamental para garantir a segurança do atleta. Esta revisão tem como objetivo discutir a abordagem de atletas com miocardite e abordar os desafios associados às decisões quanto ao retorno à prática desportiva.

Palavras-chave: Miocardite; Atleta; Morte Súbita Cardíaca; Tomada de Decisão Partilhada; Retorno à Prática Desportiva.

Abstract

Myocarditis is a condition characterized by heterogeneous etiologies, clinical manifestations, and natural history. Its diagnosis remains challenging and requires a multidisciplinary approach that integrates clinical evaluation, biomarkers, electrocardiography, echocardiography, and advanced imaging modalities. Cardiac magnetic resonance has emerged as the non-invasive gold-standard test, that together with endomyocardial biopsy, constitutes the foundation of the current diagnostic classification. Myocarditis is also a frequent acquired cause of sudden cardiac death in young individuals, including athletes. In this setting, the evaluation of return-to-play eligibility in athletes after acute myocarditis is complex and often limited by scarce evidence-based data. Accurate risk stratification, guided by a comprehensive multiparametric evaluation and a shared decision-making process that considers the potential risks of resuming competitive sport, is fundamental to ensuring athlete safety. This review aims to discuss the management of athletes with myocarditis and to address the challenges associated with return-to-play decisions.

Key-words: Myocarditis; Athlete; Sudden Cardiac Death; Shared-Decision; Return to Play.

Introduction

Myocarditis is histologically defined as an inflammatory infiltrate associated with myocyte necrosis not caused by ischemia and generally diffuse that may present as an acute, subacute, or chronic condition.[1] Acute myocarditis (AM) is defined by symptom duration of ≤4 weeks. Due to the spectrum of related diseases sharing similar etiologies and the potential for contiguous involvement, the 2025 European Society of Cardiology (ESC) Guidelines for the management of myocarditis and pericarditis introduce the term inflammatory myopericardial syndrome.[2]

The clinical presentation of myocarditis is highly variable, ranging from oligosymptomatic cases to those with chest pain, fatigue, dyspnea, syncope, arrhythmias, congestive heart failure, or even sudden cardiac death (SCD).[3] The natural history of this condition is highly variable; most patients recover fully and others develop dilated cardiomyopathies with heart failure symptoms and increased risk of ventricular arrhythmias, especially in the acute phase.[4] AM is considered fulminant when it presents with acute onset and hemodynamic instability, requiring advanced support such as inotropic drugs or mechanical circulatory assistance. Complicated AM refers to cases associated with left ventricular (LV) systolic dysfunction, sustained ventricular arrhythmias, advanced atrioventricular block, heart failure, or cardiogenic shock.[5]

Most cases of myocarditis have presumed an infectious cause, particularly viral, but several non-infectious conditions could be involved, including systemic diseases, autoimmune disorders, inflammatory bowel disorders, drugs and toxic reactions, chest radiation and some genetic conditions.[1,6] Diagnosis is often challenging and requires a comprehensive and multidisciplinary approach an is often a diagnosis of exclusion. The advent of multimodality imaging, particularly cardiac magnetic resonance (CMR), the gold-standard test, has significantly improved the feasibility of non-invasive clinical diagnosis.[2] A new classification system proposes integrating clinical presentation with either positive CMR findings or endomyocardial biopsy (EMB) results, alongside updated histopathological definitions and revised Lake Louise CMR criteria.[2]

Myocarditis is frequently associated with malignant ventricular arrhythmias and is a common acquired cause of SCD in young individuals, with a reported

prevalence among autopsied cases ranging from 1.1% to 12%. Although myocarditis can occur at any age, it predominantly affects young, previously healthy individuals; there is a clear predominance among, men with incidence approximately 2.2 times higher than that of women.[7,8] In this setting, myocarditis is also a relevant entity among athletes, causing 2% to 9% of SCD cases in this population.[9] This circumstance presents substantial challenges in the management of athletes diagnosed with myocarditis, particularly concerning the assessment of eligibility for participation in sports and the formulation of evidence-based return-to-play (RTP) strategies. Accurate risk stratification, supported by a multiparametric evaluation and a shared decision-making process, which carefully considers the potential risks of resuming competitive sports, is a critical component of management in this context.

The present review aims to discuss the management of athletes with myocarditis and to address the challenges associated with RTP decisions.

Myocarditis as cause of sudden cardiac death in athletes

Several studies have examined the epidemiology and the causes of SCD in athletes. Table 1 summarizes the main results of the most relevant studies, highlighting the reported incidence of myocarditis.

Acute myocarditis is a relevant cause of SCD in athletes and in some reports is the third most common cause in young athletes, after autopsy-negative SCD and coronary artery anomalies.[10] Van Camp et al.[11] analyzed non-traumatic deaths among high school and college athletes over a 10-year period (1983–1993) in the USA. Among 160 deaths, SCD was identified in 100 cases and an additional seven deaths were presumed to be associated with primary cardiac arrhythmia. The most frequent cause was hypertrophic cardiomyopathy (HCM), while myocarditis accounted for seven cases (6.5%).

Corrado et al.[12] evaluated the causes of sudden death in young individuals aged 12–35 years over a 21-year period in the Veneto region, Italy. A total of 55 deaths were reported, 49 (89%) of which were considered SCD, mainly due to arrhythmogenic cardiomyopathy (12 cases), while myocarditis was identified in five (9.8%) cases. A study performed in the United Kingdom analyzed SCD cases in athletes, mainly amateurs, who

were referred to a tertiary cardiac pathology center for definitive etiological determination over a 12-year period. Of 118 cases, 89 were of cardiac origin, in which the predominant cause was idiopathic LV hypertrophy (36 cases), whereas only one case of myocarditis was identified (3.0%).[13] Maron et al.[14] studied 1866 sudden death cases in American competitive athletes (aged from 8 to 39 years) over a 27-year period (1980–2006), of which 1049 were considered to be of probable cardiac origin and 690 were confirmed as SCD. The leading cause was HCM (251 cases), while myocarditis accounted for 41 events (5.9%). A similar incidence of myocarditis as cause of SCD in athletes have been reported in other studies.[15-18] A study performed by Finocchiaro et al.[19] evaluating athletes over a period of approximately 20 years (1994–2014) revealed sudden arrhythmic death syndrome (SADS) as the most frequent cause of death (149 cases; 42%), being myocarditis implicated in only in seven cases (2.0%). In more recent studies myocarditis has been associated with 4.0%–7.0% of SCD in athletes.[20-22,24] For example, in the FIFA Sudden Death Registry (2014 to 2018), Egger et al.[22] reported 174 cases SCD with a cardiac origin, eight (4%) due to myocarditis. However, a recent European study by Böhm et al.[23] showed that myocarditis was the third most common cause of SCD, with a higher prevalence, both in autopsy-confirmed cases (16%) and in cases without autopsy assessment, which also included survivors of sudden cardiac arrest (11.2%).

Across the available evidence, SADS, HCM and arrhythmogenic cardiomyopathy emerge as the most frequent causes of SCD in young athletes, while myocarditis accounts for a smaller but meaningful proportion of cases. Furthermore, there is a clear male predominance in cases of myocarditis and sports-related SCD, which may be partly explained by the higher proportion of male athletes engaged in high-intensity physical activity worldwide. Nonetheless, a biological component cannot be excluded, as female athletes may benefit from a hormonally mediated protective immune response that reduces the risk of myocarditis and its progression to fatal arrhythmias and SCD.[25] The sports reported as most frequently being associated with myocarditis-related SCD included football, basketball, cycling, gymnastics, and long-distance running, which largely reflects the popularity and participation rates of specific disciplines within each country or region studied.[26-28] Although most SCD events occurred during or immediately after physical exertion, a significant proportion, up to 40% in some series,

were reported at rest, which is challenging for exercise prescription and RTP.[19] Despite regional and methodological differences, these studies collectively highlight that myocarditis remains an important cause of SCD, warranting careful consideration in cardiovascular (CV) evaluation and risk stratification of athletes.

Specificities of myocarditis in athletes

Athletes may have a higher risk of developing myocarditis than the general population, with several sport-specific factors contributing to their increased susceptibility to this condition. Some behavioral and physiological requests of competition are associated with reduced immune competence, largely due to sleep deprivation, frequent travel and circadian rhythm disruption caused by jet lag.[29] The increasing demands of competitive sports require athletes to perform in shorter competition cycles, with limited recovery time and frequent participation in events across diverse global locations. Consequently, recurrent air travel and time zone shifts can disrupt circadian homeostasis and impair immune function. Moreover, certain sports, particularly endurance disciplines, are often performed under extreme environmental conditions such as high temperatures, cold exposure, altitude, or low humidity, which may transiently suppress immune activity.[29,30]

Furthermore, athletes participating in team and contact sports experience greater exposure to pathogens due to close physical proximity, which increases the likelihood of viral transmission. This is particularly relevant, as viral infections are the primary cause of myocarditis in both athletes and the general population.[1,30]

On the other hand, strenuous exercise induces a systemic inflammatory response characterized by mobilization of neutrophils and monocytes, associated with transient immune suppression. This involves several mechanisms, including a reduction in the number of naïve T cells, particularly CD4+ T cells, decreased natural killer cell cytotoxicity and diminished thymic output.[30,31] Intense physical activity also elevates cortisol, the primary stress hormone, while reducing the concentrations of immune-supporting factors such as salivary secretory immunoglobulin A, lactoferrin, and lysozyme.[30] This inflammatory milieu contributes to an increased predisposition to both supraventricular

and ventricular arrhythmias, the latter of which can heighten the risk of malignant arrhythmias and SCD.[29,31]

Another important factor is the emotional and psychological burden experienced by competitive athletes. Chronic competitive stress and the constant pressure to succeed or achieve peak performance may lead to the use of antidepressant medication or doping agents, which can adversely affect CV health and further increase the risk of myocarditis.[29,31]

Figure 1 illustrates the relationship between high-intensity exercise and the potential increased risk of myocarditis and other CV events in athletes, as modulated by behavioral, environmental, and immunological factors.

Clinical presentation and diagnosis

Myocarditis classically presents with chest pain, which is typically retrosternal and may be described as burning or oppressive. This pain often occurs in the context of a recent viral infection within the preceding two weeks, such as upper respiratory tract symptoms, gastrointestinal symptoms, or a general flu-like syndrome with malaise and headache.[2] The clinical presentation is highly variable, ranging from mild and non-specific symptoms that may go unrecognized to severe forms with acute or advanced heart failure, malignant ventricular arrhythmias, and, in rare cases, SCD.[2,5]

In athletes, symptoms are frequently overlooked or misinterpreted. This may be related to high pain tolerance, the frequent occurrence of musculoskeletal symptoms during viral illnesses, the attribution of chest discomfort to benign musculoskeletal causes, and a strong psychological motivation to continue training or competing.[10] As a result, clinicians should maintain a high index of suspicion when evaluating athletes with compatible symptoms. Additionally, the preceding viral illness may be very mild or entirely asymptomatic, further contributing to diagnostic delay.[2]

The diagnostic evaluation of suspected myocarditis should begin with a detailed clinical history and thorough physical examination, followed by a 12-lead electrocardiogram (ECG), laboratory assessment including high-sensitivity cardiac troponin, and

transthoracic echocardiography[2]. In the presence of a normal high-sensitivity troponin, clinically relevant myocarditis is highly unlikely and can be safely excluded in most cases. However, very mild elevations in high-sensitivity troponin may be observed in athletes after strenuous exercise and should be interpreted with caution, particularly in the absence of compatible symptoms or supportive imaging findings.

When myocarditis is suspected, CMR is the key non-invasive imaging modality. CMR enables comprehensive assessment of left and right ventricular regional and global function, evaluation of pericardial effusion, and detailed myocardial tissue characterization. CMR findings compatible with myocarditis are defined by the updated Lake Louise criteria and require a combination of markers of myocardial inflammation and injury.[32] These criteria include evidence of myocardial edema, demonstrated by regional or global hyperintensity on T2-weighted images or increased myocardial T2 values on T2-mapping, together with markers of myocardial injury such as increased native T1 values and/or extracellular volume fraction, and the presence of non-ischemic patterns of late gadolinium enhancement (LGE). The presence of non-ischemic myocardial injury in the absence of active myocardial inflammation does not support the diagnosis of AM. In such cases, CMR findings cannot reliably distinguish between healed myocarditis and myocardial injury related to other etiologies. For this reason, the timing of CMR is crucial, as delayed imaging may lead to false-negative results for active inflammation. In this setting, CMR should be ideally performed within the first two weeks after clinical presentation. Importantly, athletes exhibit a higher prevalence of LGE compared with the general population, commonly focal and located at the right ventricular insertion points and this may represent a benign or physiological finding rather than active or clinically relevant myocardial disease.[33] Beyond diagnostic purposes, CMR findings also carry prognostic value. Parameters such as LV ejection fraction (LVEF) and the extent of myocardial injury, particularly the presence and burden of LGE, have been associated with clinical outcomes and may inform risk stratification.[33]

Diagnosis and risk stratification of myocarditis are summarized in Table 2. According to the 2025 ESC clinical practice guidelines, a clinical diagnosis of myocarditis can be categorized as definite, possible, or rejected/unlikely, depending on the combination of clinical presentation and evidence of myocardial inflammation demonstrated by CMR

and/or EMB.[2] A clea4r diagnosis requires a compatible clinical presentation together with either a positive EMB or CMR fulfilling two imaging criteria, including at least one T2-based criterion (myocardial edema) and one T1-based criterion (myocardial injury). A possible diagnosis may be established in patients with an appropriate clinical presentation and one positive imaging criterion on CMR or uncertain EMB findings. In patients with a compatible clinical presentation but without supportive imaging or biopsy findings, the diagnosis of myocarditis is considered unlikely or rejected. Additional supportive parameters include the standard diagnostic work-up, such as serum biomarkers of myocardial injury, ECG abnormalities, and echocardiographic evidence of LV ventricular dysfunction or wall motion abnormalities.[2]

Based on clinical presentation and imaging findings, patients can be stratified into different risk categories, which guide further management. High-risk clinical features include presentation with acute heart failure or cardiogenic shock, and severe dyspnea (NYHA class III–IV) refractory to optimal medical therapy. Additional high-risk features are cardiac arrest or syncope, sustained ventricular tachycardia or ventricular fibrillation, and high-grade atrioventricular block. High-risk imaging features include newly developed moderate or severe LV systolic dysfunction (LVEF <40%) and extensive LGE. In these settings, EMB is recommended to establish a definitive diagnosis and guide therapy and admission to an intensive care unit is recommended. Intermediate-risk clinical features include newly developed or progressive dyspnea, non-sustained ventricular arrhythmias, and persistent or relapsing elevation of cardiac troponin. Intermediate-risk imaging features include newly mild LV systolic dysfunction (41% to 49%), regional wall motion abnormalities, or preserved LVEF with LGE involving at least two myocardial segments. Low-risk patients typically present with stable or mild symptoms, preserved LVEF, and absent or limited LGE. In low- and intermediate-risk patients, CMR should be preferred over EMB for initial diagnostic evaluation.[2]

Follow-up CMR imaging is recommended three to six months after the acute episode to assess resolution of myocardial inflammation and to quantify residual myocardial scarring. In cases of ongoing active myocardial inflammation or persistent LV systolic dysfunction, EMB should be performed to identify specific etiologies that may benefit from targeted therapy, including giant cell myocarditis, cardiac sarcoidosis, and

eosinophilic myocarditis, for which immunosuppressive treatment such as corticosteroids may be indicated.[2]

Myocarditis “in the field”

To illustrate the practical approach of athletes suffering AM two paradigmatic case reports are shown.

Athlete A:

A 26-year-old male professional football player presented with burning retrosternal chest pain occurring one week after a flu-like illness. The initial viral syndrome consisted of nasal congestion, cough, and fever lasting two days. As symptoms improved, the athlete resumed training. Approximately one week later, he reported a reduction in exercise capacity, dyspnea during high-intensity exertion, and the onset of burning chest pain lasting several hours.

On physical examination, resting heart rate was 82 bpm, blood pressure 122/78 mmHg and cardiopulmonary auscultation was normal. There were no clinical signs of heart failure, including peripheral edema. The ECG demonstrated diffuse ST-segment elevation (Figure 2) and the laboratory testing revealed elevated high-sensitivity cardiac troponin. CMR imaging demonstrated extensive myocardial inflammation, with increased signal intensity on T2-weighted images and subepicardial LGE in a non-ischemic distribution, LV and right ventricular systolic function were preserved, and no pericardial effusion was identified.

The athlete was admitted, and during the four-day hospital stay, symptoms progressively improved, with no evidence of heart failure or documented cardiac arrhythmias. High-sensitivity troponin levels declined steadily and was discharged without the need for anti-inflammatory therapy for symptom control.

He was advised to abstain from competitive sports for three months and was scheduled for follow-up evaluations, including clinical reassessment, repeat CMR imaging, ECG, 24-hour Holter monitoring, and exercise stress testing.

Athlete B:

A 30-year-old male professional cyclist presented with palpitations, reduced exercise tolerance during high-intensity efforts, and chest discomfort, occurring approximately two weeks after a self-limited episode of gastroenteritis. The gastrointestinal illness did not lead to interruption of training and resolved spontaneously.

Physical examination at presentation was unremarkable. The ECG showed sinus rhythm with premature ventricular contractions, laboratory evaluation revealed elevated cardiac biomarkers, and transthoracic echocardiography demonstrated dilation of the cardiac chambers with mild LV systolic dysfunction. Although the pre-test likelihood of coronary artery disease was low, a coronary computed tomography angiography was performed, excluding coronary artery disease. CMR demonstrated mild LV systolic dysfunction, increased myocardial T2 values, consistent with myocardial edema, and LGE in a non-ischemic pattern involving the LV free wall (Figure 3). During a one-week hospitalization, telemetry revealed isolated and couplet premature ventricular contractions over the first three days, which resolved spontaneously thereafter. High-sensitivity cardiac troponin levels declined progressively throughout the admission. One month after discharge an echocardiographic reassessment was performed, showing normalization of LV systolic function.

Abstinence from exercise training for three months was recommended, with follow-up including repeat CMR, 24-hour Holter monitoring, and exercise testing prior to RTP.

Return to play after acute myocarditis

Return to play after AM remains challenging and extensively debated topic. It has been suggested that exercise may exacerbate myocardial inflammation and necrosis through several mechanisms, increasing the risk of arrhythmias and SCD.[34,35] In this context, particularly during the acute phase of myocarditis, patients are advised to refrain from exercise. The recommended duration of this restriction depends on the clinical presentation, illness duration, LV systolic function, and extension of LGE on CMR. CMR

plays a key role in identifying ongoing or healed process and in risk stratification, particularly by evaluating T2-weighted images and LGE uptake.[36-38]

After the restriction period and to ensure the resolution of the disease, a comprehensive evaluation should be conducted, including 24-hour Holter monitoring, laboratory analysis (troponin and biomarkers of inflammation), exercise stress testing and CMR (if myocardial edema or LGE was present during the acute illness). Current guidelines recommend that RTP should be based on absence of symptoms, normalization of LVEF and serum biomarkers, absence of ongoing inflammation by CMR and complex ventricular arrhythmias, either on 24-hour Holter monitoring or during exercise stress testing.[36-38]

The clinical significance of persistent LGE in asymptomatic athletes following the index event remains controversial, but an extensive LGE on baseline CMR after AM increases the risk of adverse cardiac outcomes, namely SCD or life-threatening arrhythmias.[39,40] Furthermore, in a retrospective study, dynamic changes in LGE after AM were reported in follow-up control CMR three to 12 months after diagnosis.[41] Dynamic changes in LGE were highly variable with <50% decrease or an increase being independently associated with CV events, underscoring the relevance of follow-up CMR for risk stratification.[42]

European Society of Cardiology guidelines recommend that RTP should be guided by the absence of myocardial inflammation and fibrosis on follow-up CMR. However, individuals with persistent LGE but no evidence of myocardial edema or other red flags on complementary testing should be assessed on an individual basis. They may be allowed to resume competitive sports. Conversely, those with extensive myocardial scarring (>20% LGE), especially with persistent LV dysfunction, should be advised against engaging in moderate- or high-intensity exercise.[36,38]

Beyond its extent, the prognostic relevance of LGE location within the myocardium remains unclear. However, the ITAMY - ITalian multicenter study on Acute MYocarditis study[34] analyzed the role of CMR and LGE in predicting outcomes in patients with AM and preserved LVEF. During a median follow-up of more than four years, the composite endpoint of cardiac death, appropriate implantable cardioverter-defibrillator therapy, resuscitated cardiac arrest, and hospitalization for heart failure was evaluated according

to LGE location. Midwall LGE in the anteroseptal myocardium was independently associated with a worse prognosis compared with other patterns (inferolateral LGE, other LGE patterns, or absence of LGE). Although more robust data are required, these findings may warrant consideration in RTP decision-making.

The optimal duration of exercise restriction following AM remains controversial. Traditionally, abstinence from moderate- to high-intensity exercise has been recommended for three to six months. However, more recent recommendations suggest that, in uncomplicated cases, this period may be reduced to one month after resolution of symptoms.[2,38] In fact, some data have suggested that resolution of inflammation, initially detected by CMR, can occur four to six weeks after diagnosis.[44,45] As these recommendations are mainly based on expert opinion, an individualized approach and a shared decision-making process are critical.[46] Figure 4 presents a proposed management strategy for RTP in athletes following AM.

An early return to very low-intensity physical activity may be considered in carefully selected cases, following an individualized, case-by-case approach. This should occur under close clinical supervision, with thorough arrhythmic risk assessment and a personalized exercise prescription developed in collaboration with the team's medical staff. Acceptable activities may include stretching, technical or skill-based drills, and exercises with low dynamic and low static components, while strictly avoiding significant CV load and competitive training. Importantly, allowing a limited degree of training may confer mental health benefits, help prevent detraining and loss of technical skills, and promote continued engagement with the sporting environment and team dynamics.

Challenges and the future

Despite myocarditis being a relatively frequent condition in athletes, several gaps and challenges persist in its management. In particular, the optimal duration of exercise restriction, the clinical significance of persistent LGE and the appropriate timing for repeat CMR are still unclear.

To date, no studies have specifically investigated the incidence of complications following AM and their association with exercise, which, due to the expectedly low event rate, would be difficult to obtain. Growing evidence has linked myocarditis to the "hot phase"

of arrhythmogenic cardiomyopathy.[47,48] Indeed, a substantial proportion of cases carry genetic variants associated with cardiomyopathies, which are particularly relevant for athletes with mutations linked to arrhythmogenic cardiomyopathy, especially in desmosomal proteins such as desmoplakin.[49] The differential diagnosis between this newly recognized clinical entity and isolated AM remains challenging. Clinicians should suspect an underlying genetic substrate in the presence of disproportionate, recurrent, or unexplained ventricular arrhythmias; recurrent episodes of myocarditis; family history of cardiomyopathy or SCD; LGE with atypical patterns and distributions; or phenotypic features suggestive of arrhythmogenic cardiomyopathy. In such cases, a comprehensive and multimodal approach to identifying red flags, including a detailed family history, serial imaging studies, ambulatory rhythm monitoring and genetic testing, is essential and should be undertaken.[50] This distinction is clinically relevant, as the identification of an inherited arrhythmogenic cardiomyopathy has major implications for risk stratification and RTP decisions. Given the well-established association between high-intensity exercise and both arrhythmic risk and disease progression, this type of activity is generally discouraged in these patients.[49,50]

Despite existing recommendations, approximately half of patients with AM do not receive counseling regarding sporting activity. Among those who do, adherence is generally high but varies according to activity level. Competitive athletes tend to receive counseling more frequently than recreational athletes but have lower adherence to the recommendations. Therefore, it is crucial to improve guideline-based counseling on sports participation for patients recovering from AM.[51] Current RTP protocols are similar across all athletes, but further studies are warranted to explore the relationship between AM and its complications in specific sports.

Finally, given that exercise restriction can significantly impact athletes' mental health, this factor should be incorporated into a shared decision-making process, carefully weighing individual risks and benefits in each case.[26,27]

Conclusions

Myocarditis is a clinically significant cause of SCD in athletes, presenting considerable challenges in diagnosis, risk stratification, and RTP decision-making. A comprehensive multiparametric evaluation is essential, with CMR playing a central role in both diagnosis and prognosis. RTP decisions should be individualized, based on complete clinical, functional, and imaging recovery, and made through a shared decision-making process, with progressively shorter restriction periods for uncomplicated cases. Further research is needed to better understand the interaction between exercise and myocarditis and to develop evidence-based strategies that ensure athlete safety.

Ethics in publishing

1. Does your research involve experimentation on animals?:

No

2. Does your study include human subjects?:

No

3. Does your study include a clinical trial?:

No

4. Are all data shown in the figures and tables also shown in the text of the Results section and discussed in

the Conclusions?:

Yes

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Figures' legends

Figure 1. Mechanisms linking sports and risk of myocarditis.

Figure 2. ECG showing diffuse ST-segment elevation.

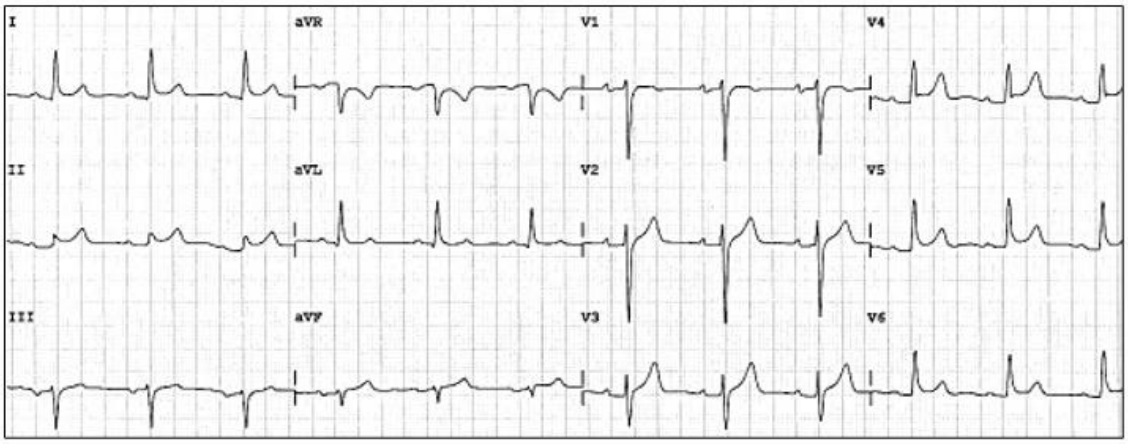

Figure 3. CMR showing LGE in a non-ischemic pattern involving the LV free wall (upper panels) and increased myocardial T2 values (lower panels) consistent with myocardial edema.

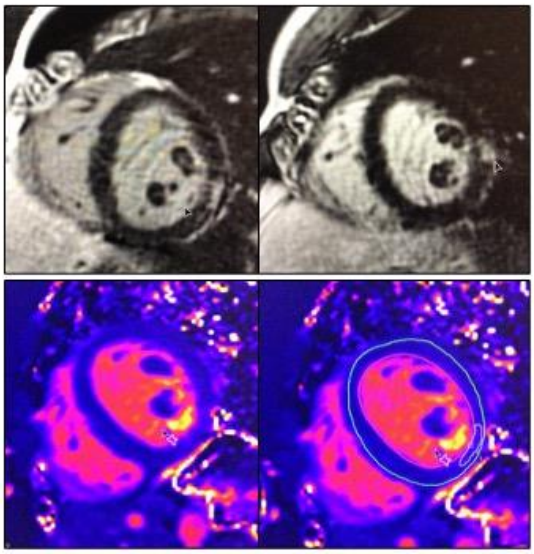

Figure 4. Proposed management strategy for RTP in athletes following AM.