Myocarditis in the Athlete: Arrhythmogenic Substrates, Clinical Manifestations, Management, and Eligibility Decisions

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Abstract
Myocarditis is an important cause of sudden cardiac death (SCD) among athletes. The incidence of SCD ascribed to myocarditis did not change after the introduction of pre-participation screening in Italy, due to the transient nature of the disease and problems in the differential diagnosis with the athlete’s heart. The arrhythmic burden and the underlying mechanisms differ between the acute and chronic setting, depending on the relative impact of acute inflammation versus post-inflammatory myocardial fibrosis. In the acute phase, ventricular arrhythmias vary from isolated ventricular ectopic beats to complex tachycardias that can lead to SCD. Atrioventricular blocks are typical of specific forms of myocarditis, and supraventricular arrhythmias may be observed in case of atrial inflammation. Athletes with acute myocarditis should be temporarily restricted from physical exercise, until complete recovery. However, ventricular tachycardia may also occur in the chronic phase in the context of post-inflammatory myocardial scar.

Keywords
Myocarditis · Athletes · Sport · Ventricular tachycardia · Ventricular fibrillation · Atrial fibrillation · Atrioventricular block · Sudden death

Abbreviations
AM Acute myocarditis
SCD Sudden cardiac death
EMB Endomyocardial biopsy
LGE Late gadolinium enhancement
CMR Cardiac magnetic resonance
AV Atrioventricular
LV Left ventricular
AF Atrial fibrillation
VF Ventricular fibrillation
VT Ventricular tachycardia
ICD Implantable cardioverter defibrillator

Introduction
Myocarditis is an inflammatory disease of the heart muscle most often caused by infectious agents (infective myocarditis), autoimmune conditions, or pharmacological and environmental toxins (non-infective myocarditis) [1]. Infective myocarditis due to viral agents (coxsackievirus, adenovirus, parvovirus, and herpes virus) is fairly the most common form of the disease and is usually characterized by a benign prognosis [2]. The majority of patients with acute myocarditis (AM) have a mild and transient cardiac involvement, most often mimicking the clinical features of acute coronary syndromes, and a favorable clinical course with a complete clinical recovery; acute heart failure and sudden cardiac death (SCD) are less common clinical presentations [1, 3, 4]. Post-inflammatory myocardial sequelae are not so rare and consist of repair subepicardial-midmural myocardial fibrosis which occurs as a result of the healing process of a previous acute myocardial lesion [5]. There is growing evidence that non-ischemic myocardial scar, either post-myocarditis or due to other myocardial diseases, may act as a substrate of...
life-threatening ventricular arrhythmias and arrhythmic cardiac arrest, mostly in young people and athletes [6].

The present article reviews the etiology, pathogenesis, clinical manifestations, risk stratification, and management of myocarditis occurring in the athlete, with particular reference to the different arrhythmogenic substrates and mechanisms acting in the acute and chronic disease settings.

**Myocarditis in the Athletes**

Although definite evidence is lacking, athletes may be at higher risk of developing myocarditis than the general population. Sports activity can influence the susceptibility to infections, depending on the intensity and duration of the physical exercise. While moderate physical activity may improve immunological defenses [7–9], intense and prolonged training and competition lower the immunity by reducing salivary secretory immunoglobulin A, lactoferrin, and lysozyme and altering the T cell response, all mechanisms that may increase the vulnerability of athletes to viral infections [7, 8, 10–14]. Non-infective myocarditis, i.e., immune-mediated and toxic myocarditis, may also be favored.

Sports activity may also worsen the pathobiological course of AM [15–18]. Murine models of myocarditis by coxsackievirus B3 showed a striking augment in the virulence of the pathogen agents when the animals were forced to swim [15]. The acceleration of coxsackie B3 myocarditis course induced by exercise was also reported by Kiel et al., who observed that exercise increased both the extent of myocardial necrosis and overall mortality [16]. Worsening of the myocardial pathological damage can occur not only in viral myocarditis but also in autoimmune myocarditis as demonstrated by the increase of both humoral and cellular immunities against the heart muscle in mice exercised by treadmill [19].

Myocarditis has been traditionally regarded as cause of life-threatening ventricular arrhythmias and SCD in the athletes. The incidence of SCD due to AM in the athlete ranges from 2 to 12% of all fatalities [20–24]. Data from the registry of juvenile SCD of the Veneto region of Italy showed that the mortality did not significantly change before and after the implementation of systematic pre-participation screening in Italy (from 0.3/100,000 to 0.15/100,000, p = 0.42) [25]. Failure to identify athletes with myocarditis by pre-participation screening reflects the acute and unpredictable nature of the inflammatory process, which often is clinically silent and may lead to electrical ventricular instability and cardiac arrest in otherwise asymptomatic individuals. Furthermore, highly trained athletes tend to underestimate or hide symptoms to avoid limitations to competitive sports eligibility.

**Challenges in the Diagnosis of Acute Myocarditis in the Athlete**

Definitive diagnosis of AM relies on histologic demonstration of myocardial inflammatory infiltrates and myocyte degeneration and death on endomyocardial biopsy (EMB). However, the use of EMB is uncommon in the routine clinical practice and according to current guidelines the invasive approach for histologic examination of myocardial samples is reserved to selected clinical scenarios that do not include most common benign and self-limiting presentation [26]. In the absence of EMB confirmation, the clinical diagnosis of myocarditis is strongly suspected or highly probable in the presence of ≥2 criteria including compatible clinical symptoms, electrocardiographic abnormalities, elevation of myocardio cytolysis markers, evidence of non-ischemic morphofunctional ventricular abnormalities, and increased T2 signal for myocardial edema/regional late gadolinium enhancement (LGE) at cardiac magnetic resonance (CMR) [1].

Personal and family history, physical examination, and 12-lead ECG are crucial in the initial evaluation of athletes with suspected myocarditis. Symptoms, such as chest pain, dyspnea, and fatigue, possibly start after a respiratory or gastrointestinal infection.

At the electrocardiogram, repolarization abnormalities are the most common alterations in AM, being detected in 40% of patients; ST segment elevation (typically concave and diffuse without reciprocal changes) is more frequent than ST segment depression (6% vs 2%), and negative T waves (36%) are observed mainly in lateral leads [27]. These ECG changes are typically transient in AM [28], and even when present, their significance might be uncertain since athletes frequently show repolarization abnormalities (i.e., early repolarization) [29]. The most notable ECG feature of early repolarization is ST segment elevation, which may vary on morphology, location, and degree; T wave inversion can also be observed particularly in black athletes but confined to leads V1–V4 [30]. Pathologic Q waves are reported in 13% of patients with AM [27], whereas they are rare in athletes [31]. PR segment depression and low voltages can reveal the existence of pericardial inflammation and effusion and tend to disappear throughout the healing process. Other ECG changes include atrioventricular (AV) and bundle branch blocks. Electrocardiographic differential diagnosis between AM and athlete’s heart is summarized in Table 1.

Biomarkers indicative of myocardial injury are usually elevated in myocarditis; however, the majority of healthy athletes after prolonged exercise show values of cardiac troponin above the 99th percentile [32].

Echocardiography may solve the dilemma, identifying signs of the disease such as global left ventricular (LV) enlargement and dysfunction, localized wall motion abnormalities, and relaxation alterations suggestive of the presence of
inflammatory lesions [33, 34]. Modest pericardial effusion and increased reflectivity of the pericardial leaflets may be observed. Of note, both patients with myocarditis and athletes may show mild depression of the LV ejection fraction at rest, but in healthy athletes, wall motion abnormalities are always absent. Moreover, exercise echocardiography can exacerbate regional wall motion abnormalities and further reduce the ejection fraction in patients with myocarditis while systolic function typically normalizes in healthy athletes during exercise [35].

CMR imaging has emerged and progressively gained importance as a non-invasive modality for the diagnosis of AM by identifying regions with myocardial edema (T2-weighted sequences) and providing information on myocardial damage/necrosis by the technique of LGE. Specific imaging criteria (“Lake Louis” criteria) can be used to confirm the diagnosis of AM based on demonstration of epicardial and/or midmyocardial edema, hyperemia, and myocardial fibrosis/LGE [36]. Edema and LGE are usually absent in athletes, although the latter can be the sign of a previous silent myocarditis or of an underlying cardiomyopathy (see below) [6, 35].

### Arrhythmias in Acute Myocarditis

The mechanisms involved in the pathogenesis of myocarditis are not fully understood and still remain an open field of research. The majority of the studies focused on viral and autoimmune myocarditis, postulating a three-phase process from acute injury to chronic dilated cardiomyopathy [1]. Acute injury leads to cardiac damage, exposure of intracellular antigens, and activation of the innate immune system. Inflammatory infiltrates and dying myocytes predispose to the development of tachyarrhythmias, either supraventricular or ventricular [5].

It has been reported that in up to 24% of cases, the first clinical manifestation consists of arrhythmias (either tachy- or bradyarrhythmia), syncope, or SCD [37–40]. Table 2 reports the prevalence of various arrhythmias in AM.

### Supraventricular Arrhythmias

Sinus tachycardia is a common finding in AM as a result of the systemic inflammation and heart failure. Adrenergic stimulation occurring in atrial inflammation is involved in the onset of atrial tachyarrhythmias [3]. In fact, the inflammatory process of AM often extends to the atria; atrial myocarditis in the absence of ventricular involvement has also been reported [41–44].

Since either local or systemic inflammation is associated with induction and maintenance of atrial fibrillation (AF) in the general population [45, 46], it is not surprising that this arrhythmia represents a common complication in myocarditis, with a reported prevalence of 2.5–14% [27, 39, 47]. The postulated mechanism is that the inflammatory process causes an increased automaticity of the atrial myocardium resulting in the occurrence of atrial ectopic beats, which may trigger AF [48]. In patients with Wolff-Parkinson-White syndrome who died suddenly, Basso et al. demonstrated the presence of inflammatory infiltrates in the atrial myocardium close to the accessory pathway, a finding which substantiates a cause-effect relationship between atrial myocarditis and rapid pre-exited AF degenerated into ventricular fibrillation (VF) [49]. Inflammatory-mediated increase of ectopic activity arising from the pulmonary vein, which is a well-recognized trigger of AF [48, 50–52], may also induce focal atrial tachycardia and in patients with dual AV node pathways or accessory pathways, paroxysmal supraventricular tachycardia [53]. Atrial inflammation leading to myocyte necrosis and fibrotic changes creates a structural myocardial substrate which worsens the electrical atrial instability and favors the arrhythmia permanence [54].

<table>
<thead>
<tr>
<th>Repolarization abnormalities</th>
<th>Acute myocarditis</th>
<th>Athlete’s heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>- ST segment elevation</td>
<td>Diffuse</td>
<td>Localized (usually in the anterior leads) or diffuse</td>
</tr>
<tr>
<td>- ST segment depression</td>
<td>Possible</td>
<td>Absent</td>
</tr>
<tr>
<td>- T wave inversion</td>
<td>Mainly in lateral or inferior leads</td>
<td>Confined to V1-V4 in Afro-Caribbean athletes</td>
</tr>
<tr>
<td>Q waves</td>
<td>Pathological (amplitude ≥ 25% of the ensuing R wave and/or ≥ 0.04 s in duration)</td>
<td>Non-pathological (≥ 4 mm in depth but &lt; 25% of the ensuing R wave and &lt; 0.04 s in duration)</td>
</tr>
<tr>
<td>Voltages of the QRS</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>PR segment depression</td>
<td>Possible</td>
<td>Absent</td>
</tr>
<tr>
<td>AV block</td>
<td>Any degree</td>
<td>First-degree or second-degree type 1</td>
</tr>
<tr>
<td>Bundle branch block</td>
<td>Left or right bundle branch block</td>
<td>Incomplete right bundle branch block</td>
</tr>
</tbody>
</table>

**Table 1** Electrocardiographic differential diagnosis between acute myocarditis and athlete’s heart
Atrioventricular Blocks

A V block is traditionally associated with myocarditis, being reported in 10% of biopsy-proven AM patients [37]. While first-degree AV block is frequently encountered, advanced AV block is uncommon and observed in specific variants of myocarditis, such as giant cell myocarditis, sarcoidosis, and bacterial or protozoal AM.

Giant cell myocarditis is a rare and rapidly progressive inflammatory heart disease usually characterized by an ominous prognosis [55]. In a recent series of patients diagnosed with giant cell myocarditis, distal AV conduction block was reported in 28% of cases [56].

High-degree AV block is typically observed in cardiac sarcoidosis (i.e., idiopathic granulomatous myocarditis), which is also complicated by ventricular tachycardia (VT) or VF and SC which may represent the first clinical manifestation of the disease [57–59]. Autopsy studies reported the presence of myocardial granulomas in 20–30% of patients, with a patchy fashion distribution, although some regions are preferred including the interventricular septum and atria, potentially justifying the conduction defects [60].

Myocardial inflammation by bacterial and protozoal infection typically causes bradyarrhythmia. In this regard, Lyme and diphtheria myocarditis are both bacterial infection characterized with various degree of AV block (Fig. 1) [61, 62]; Chagas disease is a protozoal infection caused by Trypanosoma cruzi, which can present clinically as AM with right bundle branch block or left anterior fascicular block potentially leading to complete heart block [63].

Several mechanisms have been proposed to explain the onset of ventricular arrhythmias in AM. First, myocarditis may cause myocardial ischemia similar to coronary artery disease. Ischemic mechanisms include mural thrombi, coronary artery embolization, and arteritis [64]. In addition, vasoactive kinins and catecholamines produced during the acute phase of viral infection can cause coronary artery spasm [65].

Another mechanism contributing to the arrhythmic phenotype of AM is myocardial edema (Fig. 2): local inflammation, cytokines release, and cell death constitute the basis for its formation, resulting in some cases in LV tumefaction (i.e., inflammatory pseudo-hypertrophy) [33, 36]. A recent study demonstrated that the electrocardiographic expression of transmural edema consists is the inversion of the T waves in the ECG leads exploring the affected LV wall, and interestingly such ECG abnormalities normalized completely during follow-up, underscoring its transient and acute nature (Fig. 3) [28]. There is experimental evidence that acute inflammation can modify the electrophysiological properties of the myocardium through multiple mechanisms, favoring ventricular arrhythmias [62–66].

Finally, AM has been associated with clinical deterioration of pre-existing cardiac diseases, such as hypertrophic cardiomyopathy [66]. Arrhythmogenic right ventricular cardiomyopathy (ARVC) patients suffering arrhythmic storms frequently displays sign of acute inflammation at CMR or EMB (so-called hot-phase), although the inflammatory mechanism (viral versus immune-mediated) remains to be established [67–69].

Ventricular Arrhythmias

Ventricular arrhythmias vary from simple isolated ventricular ectopic beats to complex and life-threatening tachycardias. Anzini et al. documented the presence of non-sustained VT in 28% of AM patients, whereas sustained VT or VF represented the first clinical manifestation in 7% of patients [39].

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Arrhythmias in the Chronic Phase

Three arrhythmic mechanisms may explain the occurrence of life-threatening ventricular arrhythmias in patients after an episode of AM: 1) recurrent myocarditis or persistent myocardial inflammation; 2) residual LV dysfunction and 3) post-inflammatory myocardial scar.

Table 2 Prevalence of arrhythmias in patients with acute myocarditis

<table>
<thead>
<tr>
<th>Arrhythmia</th>
<th>Prevalence (%)</th>
<th>Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sustained supraventricular tachycardia</td>
<td>0.8</td>
<td>Anderson (2014)</td>
</tr>
<tr>
<td>Atrial fibrillation/atrial flutter</td>
<td>2.5–14</td>
<td>Imazio (2008)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anzini (2013)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ukena (2011)</td>
</tr>
<tr>
<td>Non-sustained ventricular tachycardia</td>
<td>28</td>
<td>Anzini (2013)</td>
</tr>
<tr>
<td>Sustained ventricular tachycardia or ventricular fibrillation/flutter</td>
<td>7.3–9.7</td>
<td>Anzini (2013)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anderson (2014)*</td>
</tr>
<tr>
<td>AV blocks (any degree)</td>
<td>10</td>
<td>Caforio (2007)</td>
</tr>
</tbody>
</table>

*Pediatric population
Fig. 1 Serial electrocardiograms and CMR imaging of a young athlete with acute Lyme myocarditis, presenting with cardiogenic shock and third-degree AV block. A 19-year-old male football player was admitted for cardiogenic shock. The ECG at admission showed complete AV block (a). The CMR performed in the acute phase revealed circumferential edema with the highest signal intensity in the interventricular septum and inflammatory “pseudo-hypertrophy” (b). One week after the myocarditis onset, the ECG demonstrated the restoration of normal AV conduction (c). The CMR before discharge showed consistent reduction of myocardial edema and reduction of the ventricular wall thickness (d). Lab tests conducted during hospitalization confirmed the infection from *Borrelia burgdorferi*.

Fig. 2 Coronary angiography and CMR imaging of patient with biopsy proven acute myocarditis who suffered out-of-hospital cardiac arrest. Out-of-hospital cardiac arrest occurred in a 41-year-old male with biopsy-proven AM. Coronary angiography showed normal right (a) and left (b) coronary arteries. CMR showing myocardial edema (two chambers, long axis view; T2-weighted sequences) (c) and transmural LGE (two chambers, long axis view; T1-weighted inversion recovery post-contrast sequences) (d) affecting the mid-apical inferior LV wall. Modified and reproduced with permission from [90].
The healing process of AM can cause a myocardial scar that can act as a substrate for life-threatening ventricular tachyarrhythmias even in subjects with a normal LV function. Post-inflammatory fibrosis typically shows a “band” pattern and involves the subepicardial/midmyocardial layers of the infero-lateral LV wall [36]. Such lesion can be evidenced “in vivo” by gadolinium enhanced CMR sequences, while it is usually undetectable by echocardiography because fibrosis involves a small myocardial area and is confined to outer wall layers without reaching the subendocardium, which is the part of the LV wall that most contributes to myocardial contractility [70]. The resting ECG is often unremarkable and this explains why the affected athlete is missed at pre-participation screening and its prevalence among middle-aged athletes engaged in long-term endurance sports activities is low (17/613, 2.8%) (Table 3) [71–84].

The role of myocardial fibrosis as a source of ventricular arrhythmias was demonstrated by an outcome study on 405 patients with clinically suspected myocarditis. During a mean follow-up of 4.4 years, major arrhythmic events occurred only in patients with evidence of LV myocardial LGE/scar at CMR [85].

Myocardial scar with a subepicardial/midmyocardial (i.e., non-ischemic) distribution has been increasingly reported as a myocardial substrate for life-threatening ventricular arrhythmias and cardiac arrest mostly in young people and athletes (Fig. 4) [6, 86–88]. A previous study reported that, during a mean 3-year follow-up, 6 (22%) athletes with ventricular arrhythmias and a non-ischemic LV scar experienced major arrhythmic events such as appropriate implantable cardioverter defibrillator (ICD) shock, sustained VT and SCD compared with none of control athletes with ventricular arrhythmias in

**Fig. 3** Cardiac magnetic resonance findings in a representative patient with inferior T wave inversion. ECG at the time of CMR showing the presence of negative T waves in inferior leads (a). Long-axis three-chamber view showing infero-lateral myocardial edema in T2-weighted sequences (b). Semiquantitive analysis by CMR42, Circle Cardiovascular Imaging Inc. software showing the transmural arrangement of myocardial edema (c). Modified and reproduced with permission from [28].

[Image of ECG and CMR findings]
the absence of LV scar. In 5 of 6 athletes the event occurred during effort [6].

Although isolated LV LGE with subepicardial/midmyocardial distribution is traditionally interpreted as the consequence of a previous viral myocarditis, there is increasing evidence that it may reflect a segmental inherited cardiomyopathy, namely, a left-dominant ARVC (Fig. 5). This ARVC variant is characterized by fibrofatty myocardial replacement of the LV and predisposes to life-threatening ventricular arrhythmias similarly to the classic right-ventricular dominant counterpart. Other genetically determined cardiac diseases may be associated with non-ischemic LV scars unrelated to previous AM [85]. Hence, in the absence of an overt clinical history of a previous AM, the finding of a non-ischemic LV scar in an asymptomatic athlete deserves proper clinical attention and cannot be simply dismissed as a sign of a healed remote inflammatory process. Particularly, affected athletes should be accurately evaluated for family history, symptoms and the arrhythmogenic potential inherent to the myocardial fibrosis.

The available data indicate that in the presence of positive family history, symptoms, or ventricular arrhythmias, especially if worsened by exercise, athletes with an isolated non-ischemic LV scar should prudently refrain from practicing sports activity with high cardiovascular demand.

### Eligibility to Sports Activity

Athletes with proven myocarditis should be temporarily restricted from exercise programs including competitive and leisure-time sports activity [5]. Past European and American consensus documents recommended a minimum of 6 months of inactivity from the onset of the disease [86, 87]. This time period has been shortened to 3 months in more recent recommendations, but it can be extended to 6 months according to the clinical severity and duration of the illness, LV function,

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**Table 3** Prevalence and pattern of distribution of LGE in athletes

<table>
<thead>
<tr>
<th>Authors, year of publication</th>
<th>No.</th>
<th>Mean age (years)</th>
<th>Males (%)</th>
<th>Inclusion criteria</th>
<th>%LGE</th>
<th>LGE pattern in athletes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mohlenkamp 2008</td>
<td>102</td>
<td>57</td>
<td>100</td>
<td>Marathon runners (≥ 5 marathons in the last 3 years), age &gt; 50 years old</td>
<td>12 (4 controls)</td>
<td>5: subendocardial</td>
</tr>
<tr>
<td>Breuckmann 2009</td>
<td>14</td>
<td>33</td>
<td>57</td>
<td>Marathon runners, moderately trained</td>
<td>0</td>
<td>7: midmyocardial spot</td>
</tr>
<tr>
<td>Mousavi 2009</td>
<td>17</td>
<td>34</td>
<td>100</td>
<td>Marathon runners, mean 7 h training/week</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>O’Hanlon et al. 2010</td>
<td>15</td>
<td>32</td>
<td>47</td>
<td>Half marathon runners, non-elite</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Oomah et al. 2011</td>
<td>12</td>
<td>57</td>
<td>100</td>
<td>Endurance elite, various sports, &gt; 50 years old</td>
<td>50 (0 controls)</td>
<td>4: junctional</td>
</tr>
<tr>
<td>Wilson et al. 2011</td>
<td>40</td>
<td>37</td>
<td>90</td>
<td>Endurance, &gt; 10 h/training, high performance</td>
<td>13</td>
<td>1: subendocardial</td>
</tr>
<tr>
<td>La Gerche et al. 2012</td>
<td>95</td>
<td>35</td>
<td>77</td>
<td>Endurance, &gt; 7 h/week for &gt; 2 years</td>
<td>2.1</td>
<td>1: junctional</td>
</tr>
<tr>
<td>Mangold 2013</td>
<td>40</td>
<td>41</td>
<td>100</td>
<td>Triathlon running, &gt; 5 h/week for &gt; 2 years</td>
<td>0</td>
<td>4: spots in the septum</td>
</tr>
<tr>
<td>Franzen et al. 2013</td>
<td>33</td>
<td>47</td>
<td>100</td>
<td>Endurance, &gt; 10 h/week for &gt; 10 years</td>
<td>3 (0 controls)</td>
<td>1: inferior wall spot</td>
</tr>
<tr>
<td>Bohm et al. 2016</td>
<td>10</td>
<td>40–70</td>
<td>?</td>
<td>long-term sports activity</td>
<td>20</td>
<td>1: inferior wall spot</td>
</tr>
<tr>
<td>Sanchis-Gomar et al. 2016</td>
<td>152</td>
<td>54</td>
<td>70</td>
<td>&gt; 40 years of age, ran ≥ 10 miles or cycled ≥ 30 miles per week for ≥ 10 years, and competed in ≥ 10 endurance events</td>
<td>16 (11 controls)</td>
<td>7: subendocardial</td>
</tr>
<tr>
<td>Merghani et al. 2017</td>
<td>83</td>
<td>43</td>
<td>65</td>
<td>Triathlon, &gt; 10 h/week for &gt; 3 years</td>
<td>11 (0 controls)</td>
<td>8: subepicardial/midmyocardial stria</td>
</tr>
<tr>
<td>Tahir et al. 2018</td>
<td>102</td>
<td>54</td>
<td>70</td>
<td>&gt; 40 years of age, ran ≥ 10 miles or cycled ≥ 30 miles per week for ≥ 10 years, and competed in ≥ 10 endurance events</td>
<td>16 (11 controls)</td>
<td>5: subepicardial/midmyocardial stria</td>
</tr>
</tbody>
</table>

LGE late gadolinium enhancement

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The table above summarizes the prevalence and pattern of distribution of late gadolinium enhancement (LGE) in athletes. The data show the number of athletes (No.), mean age, male percentage, inclusion criteria, pre-valence of LGE, and the pattern of distribution in athletes. The study includes a wide range of athletes from different sports backgrounds, including marathon runners, endurance athletes, and triathletes. The pattern of LGE distribution varies widely, with subendocardial, subepicardial/midmyocardial, junctional, and transmural patterns being observed in different studies.
and extent of the inflammatory process on CMR [88, 89]. This interval has been considered sufficient to ensure clinical and biological resolution of the disease. However, clinical reassessment is indicated before resuming competition, including 12-lead ECG, echocardiography, exercise testing, 24-h Holter monitoring, and blood sample. The athlete is allowed to restart training and competition if serum biomarkers and LV function normalize and major ventricular or supraventricular tachyarrhythmias are excluded. Clinical follow-up every 6 months, particularly within the first 2 years, is recommended [88]. If the evaluation indicates the persistence of active myocarditis or the development of pro-arrhythmic scars, the athlete should be considered not eligible to competitive sports.

With regard to non-ischemic LV myocardial fibrosis evidenced by CMR, the recent ESC recommendations clearly state that athletes with LGE and frequent or complex
ventricular arrhythmias should refrain from competitive sports [88]. Athletes with persistent LGE and no LV dysfunction or frequent/repetitive arrhythmias can resume physical activity, but a strict clinical surveillance is recommended, as they may represent a category of subjects at higher risk of SCD.

Conclusions

Myocarditis is a well-known cause of SCD among athletes. Pre-participation screening is scarcely sensitive in identifying affected patients due to the transient nature of the disease and raises problems of differential diagnosis with athlete’s heart. In the acute phase, athletes should be temporarily restricted from physical activity until full recovery. Myocardial scars can cause life-threatening arrhythmias which may occur in healed athletes, so that long-term clinical surveillance is warranted.

Compliance with Ethical Standards

Conflict of Interest  The authors declare that they have no conflict of interest.

Ethical Approval  This article does not contain any studies with human participants or animals performed by any of the authors.

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