REVIEW ARTICLE

Journal of Pre-Clinical and Clinical Research
www.jpccr.eu

Fibrotic remodelling in the athlete's heart – a double-edged sword of endurance training

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Kulicka N, Forszt K, Kaniak A, Gawron E, Hauzer PW, Ziółkowska D, Andrzejewska J, Ogórek A, Zielska J, Osowska A. Fibrotic Remodelling in the Athlete's Heart: A Double-Edged Sword of Endurance Training. Ann Agric Environ Med. doi: 10.26444/jpccr/208593

Abstract

Introduction and Objective. While endurance training confers significant cardiovascular benefits, emerging evidence suggests that prolonged high-volume exercise may promote myocardial fibrosis in some athletes. The aim of the review is to examine the prevalence, underlying mechanisms, imaging characteristics, and clinical significance of fibrotic remodelling in the athlete's heart – emphasizing its dual role as both physiological adaptation and potential arrhythmogenic substrate.
Review Methods. The review draws on literature published between 2017- 2024, focusing on studies involving cardiac magnetic resonance imaging (CMR), myocardial fibrosis biomarkers, and structural cardiac adaptations in endurance-trained individuals. Particular attention is given to prospective and comparative research relevant to athletic populations.
Brief description of the state of knowledge. Myocardial fibrosis has been observed in up to 21% of endurance athletes, most commonly at right ventricular insertion points. Suggested mechanisms include repetitive mechanical stress, subclinical myocarditis, and sustained inflammatory activation. While certain fibrosis patterns may reflect benign remodelling, others – such as mid-wall or ischemic late gadolinium enhancement (LGE) – have been linked to increased arrhythmic risk. Newer imaging techniques, e.g. T1 mapping and extracellular volume (ECV) analysis, offer promise but require further standardization in athletes.
Summary. Fibrotic remodelling in athletes spans a clinical spectrum. Its relevance depends on distribution, extent, and co-existing structural or electrical changes. Distinguishing physiological from pathological fibrosis remains a diagnostic challenge, underscoring the need for improved risk stratification and long-term data from athletic cohorts.

Key words

cardiac, athletic performance, endurance training, arrhythmias, myocardial fibrosis, Cardiac Magnetic Resonance Imaging, ventricular remodelling

INTRODUCTION

Endurance sports, such as ultramarathons, triathlons and CrossFit, have gained immense global popularity, drawing both elite and amateur athletes into prolonged, high-volume training. While the cardiovascular benefits of regular physical activity are well established, accumulating data raise concerns that extreme endurance regimens may exceed the threshold of physiological adaptation, particularly in aging athletes with decades of cumulative cardiac load.

A growing area of interest is myocardial fibrosis, increasingly detected in asymptomatic endurance athletes through cardiac magnetic resonance imaging (CMR) with late gadolinium enhancement (LGE). A 2020 meta-analysis of 12 studies involving 1,359 participants reported a 21.1% prevalence of LGE-positive findings in athletes versus just 3.2% in sedentary controls – an odds ratio of 7.2 (95% CI: 4.5–11.5) [1]. These fibrotic changes are often localized to right ventricular (RV) insertion points or the subepicardial lateral wall, regions subject to repetitive mechanical stress during prolonged exertion.

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Received: 05.06.2025; accepted: 24.07.2025; first published: 08.08.2025

While most athletes remain asymptomatic, the clinical meaning of these findings is debated. Some interpret them as benign adaptive responses, while others view them as markers of maladaptive remodelling with arrhythmogenic potential. As Sharma et al. note, the cardiac effects of endurance training exist on a continuum between physiological and pathological adaptation, particularly in older male athletes [2]. Parry-Williams and Sharma further highlight associations between chronic endurance activity and coronary calcification, atrial dilation, and patchy fibrosis [3], while Martinez et al. stress the diagnostic uncertainty when exercise-induced cardiac remodelling (EICR) mimics early cardiomyopathy [4].

Earlier reviews have posed the binary question of whether myocardial fibrosis is a benign bystander or a malignant substrate [5]. In contrast, the current review integrates contemporary evidence including imaging, molecular biology, and clinical outcomes, to re-interpret myocardial fibrosis in endurance athletes not as a categorical pathology, but as part of a dynamic remodelling spectrum.

By synthesizing mechanistic insights, imaging advances, and emerging epidemiological trends, the review aims to provide clinicians and researchers with a contextual framework for interpreting fibrotic changes in athletic hearts. Ultimately, such a perspective may support more accurate risk stratification, diagnostic clarity, and

return-to-play guidance in this growing and complex population.

Cardiovascular effects of endurance training. Endurance training induces a range of favourable cardiovascular adaptations in healthy adults. Among the most consistently reported benefits are enhanced heart rate variability (HRV), improved autonomic regulation of cardiac function, increased maximal oxygen uptake (VO_2 max), better lipid metabolism, and reductions in both adiposity and arterial blood pressure, collectively contributing to a significantly reduced risk of cardiovascular morbidity and mortality [6–8]. These physiological changes foster a cardiovascular system that is both more efficient and more resilient, with endurance training also linked to faster recovery following physical or emotional stress and lower levels of perceived life stress [9].

Despite these well-documented advantages, accumulating evidence indicates a potential downside of prolonged or excessive endurance exercise. Structural remodelling of the heart – including chamber dilation and increased myocardial wall thickness – has been noted in some highly trained individuals. Moreover, the incidence of arrhythmias, particularly atrial fibrillation and sinus bradycardia, appears elevated in endurance athletes, with some studies also reporting occurrences of myocardial fibrosis [10]. While the health benefits of regular, moderate endurance exercise are undisputed, high-volume or high-intensity training regimens may pose cardiovascular risks that warrant closer scrutiny, particularly in older or veteran athletes [10]. On balance, current evidence supports moderate endurance training as the optimal strategy for promoting cardiovascular health, while highlighting the importance of monitoring individuals engaged in extreme exercise volumes [6, 10].

THE ATHLETE'S HEART – PHYSIOLOGICAL ADAPTATION OR SUBCLINICAL INJURY?

Typical features of the athlete's heart. Endurance training prompts a spectrum of structural cardiac adaptations that are generally considered physiological. Among the most prominent are increases in left ventricular (LV) end-diastolic volume, stroke volume, and diastolic filling velocities, reflecting enhanced preload and chamber compliance [11]. Notably, such changes can emerge even after relatively short training interventions.

The remodelling pattern differs by training type. Endurance athletes more commonly display eccentric or concentric hypertrophy, whereas sprint-trained individuals typically retain normal cardiac geometry and wall thickness [12]. These alterations occur without deterioration in systolic performance, reinforcing their interpretation as adaptive rather than pathological.

Adaptation of the right ventricle (RV) is especially characteristic of high-dynamic endurance sports. Compared to sedentary individuals, athletes in these disciplines exhibit elevated RV systolic pressures, increased contractile indices, such as TAPSE and tissue Doppler velocities, and segmental deformation patterns, favouring apical over basal strain, indicative of volume-induced remodelling rather than dysfunction [13]. Together, these structural and functional shifts illustrate a coordinated myocardial response to chronic volume load. While pronounced, such changes typically preserve overall cardiac function, and should be interpreted within the broader context of sport-specific physiology.

Borderline or maladaptive remodelling. Although most structural adaptations observed in endurance athletes are benign, there is growing recognition that in some cases these changes may verge on maladaptive. The nature and volume of training play a key role in determining this threshold; for instance, evidence from heart failure populations suggests that moderate-intensity continuous training improves ventricular function, while higher-intensity or less consistent regimens offer diminishing returns [14]. By analogy, excessive or unbalanced training loads in athletes may also tip cardiac remodelling into less favourable territory.

One area of concern is the right ventricle, which is disproportionately affected by chronic endurance exercise due to its thinner wall and greater exposure to volume load. Hypotheses suggest that repeated RV strain may lead to disproportionate chamber dilation, transient dysfunction, and even fibrosis, features that collectively contribute to an arrhythmogenic substrate [15]. While the notion of 'exercise-induced cardiomyopathy' remains controversial, isolated cases of veteran athletes presenting with elevated biomarkers and non-ischemic myocardial scarring suggest that a subset may indeed approach this borderline phenotype.

Advanced imaging has helped to clarify these patterns. Cardiovascular magnetic resonance (CMR) studies in asymptomatic endurance athletes have revealed balanced biventricular enlargement with preserved function in most, but also occasional evidence of late gadolinium enhancement (LGE) in non-ischemic patterns, sometimes consistent with prior silent myocarditis [16]. Importantly, these findings were independent of training volume, suggesting that underlying susceptibility or inflammatory history may modulate risk as much as the load itself.

It is also worth noting that borderline performance may stem from non-cardiac contributors. Impaired chronotropic response, altered microvascular regulation, and suboptimal oxygen utilization have been implicated as limiting factors, even in the presence of structurally normal hearts [17].

The distinction between adaptation and injury in athletes remains fluid, with cumulative load, genetics, and inflammatory triggers all contributing to how the myocardium responds over time.

Interpreting electrical remodelling. Electrical adaptations are frequently observed in endurance athletes, yet their clinical significance remains debated. Findings such as prolonged filtered QRS durations are relatively common – reported in nearly half of middle-aged endurance athletes – despite entirely normal structural imaging [18]. This suggests that some ECG anomalies may reflect physiological myocardial hypertrophy rather than covert pathology. Other cohorts, including elite paralympic athletes with substantial left ventricular (LV) enlargement, have demonstrated preserved systolic function and no structural abnormalities, despite notable ECG changes, further supporting a benign interpretation in many cases [19]. However, relying solely on ECG to evaluate myocardial remodelling can be misleading. Standard voltage criteria, such as the Sokolow-Lyon index, often show weak correlation with actual LV mass, especially in young endurance athletes who may display normal tracings despite substantial chamber enlargement [20].

These inconsistencies underscore the importance of integrating electrical findings with structural and functional assessment tools. ECG abnormalities in athletes should not be pathologized in isolation. Multimodal evaluation, including advanced imaging, is essential to accurately characterize the physiological versus pathological nature of observed changes. Moreover, most of the available data pertain to elite or high-volume athletes, and may not extrapolate to recreational populations with lower training burdens.

Myocardial fibrosis in athletes – patterns, pathways, and risk factors. Although endurance exercise promotes numerous cardiovascular benefits, emerging evidence suggests that, under specific conditions, it may also contribute to myocardial fibrosis – a structural alteration with potential electrical and clinical consequences. The development of fibrosis appears to be multifactorial, modulated by cumulative training load, age, gender, genetic predisposition, and prior subclinical myocarditis. While often asymptomatic, myocardial fibrosis has been implicated in arrhythmogenic events and adverse cardiac remodelling.

The following section explores the distribution patterns of fibrosis, hypothesized pathophysiological mechanisms, and key individual risk factors in athletic populations.

Prevalence and anatomical patterns of fibrosis in endurance athletes. Growing evidence suggests that myocardial fibrosis is significantly more common in endurance athletes than in sedentary individuals. A 2020 meta-analysis involving 1,359 participants reported a 21.1% prevalence of late gadolinium enhancement (LGE) among athletes, with an odds ratio of 7.2 (95% CI: 4.5–11.5), compared to controls [1]. The most consistently observed pattern involves localized fibrosis at the right ventricular (RV) insertion points and the subepicardial lateral wall, with prevalence estimates as high as 23.7% in athletes with remodelling versus 3.3% in non-athletic controls [21, 22].

These anatomical patterns appear modulated by age, gender, and training volume. Younger athletes tend to show higher LGE prevalence than veterans (27.7% vs. 19.9%), although more extensive fibrosis is often noted in older male athletes [21]. Farooq et al. [23] identified mid-wall LGE in the basal inferolateral left ventricle in 48% of competitive male endurance athletes over the age of 50, compared to just 15% of age-matched controls, h`ighlighting a distinctive, non-ischemic distribution associated with long-term training exposure.

While ventricular fibrosis remains the primary focus of most imaging studies, atrial involvement is gaining attention. In a comparative CMR study, Peritz et al. [24] found significantly greater left atrial fibrosis burden in athletes (15.5% vs. 9.6%; $p = 0.002$), raising questions about whether such remodelling represents benign adaptation, or an arrhythmogenic substrate in evolution.

While most studies suggest an elevated prevalence of myocardial fibrosis among endurance athletes, long-term outcomes remain variable. For example, Sanchis-Gomar et al. [25, 26] found no significant biomarker elevations or LGE in the majority of former elite athletes, with only two showing small, non-ischemic patches, indicating that endurance-induced fibrosis is not universal. This discrepancy may reflect cohort differences in training status, imaging modality, or age, suggesting that myocardial fibrosis may not be universal even in elite endurance populations.

Collectively, these findings suggest that myocardial fibrosis in athletes reflects not a singular process, but a spectrum of structural changes shaped by training intensity, duration, and individual susceptibility. Ongoing research is needed to clarify the long-term clinical relevance of these patterns and their progression over time.

Molecular and pathophysiological mechanisms. Myocardial fibrosis results from the activation of cardiac fibroblasts into matrix-producing myofibroblasts under the influence of mechanical stress, inflammation, or injury [27, 28]. This process is modulated by a variety of molecular signals, including transforming growth factor-β (TGF-β), angiotensin II, proinflammatory cytokines (e.g., IL-6, TNF-α), and Wnt/β-catenin pathways. These cascades stimulate collagen synthesis, alter matrix crosslinking, and contribute to electrical remodelling of the myocardium [28]. In the context of endurance training, particularly under high loads or in the presence of subclinical myocarditis, these pathways may become maladaptive. Endurance exercise during unresolved viral myocarditis, for instance, has been shown to exacerbate interstitial fibrosis through sustained inflammatory activation and collagen organization [28, 29]. Additional contributors include right ventricular pressure overload, repetitive mechanical stretch at the RV insertion points, and low-grade myocyte injury as reflected by transient troponin elevations post-exercise [29]. Evidence from murine models reinforces this hypothesis. Da Rocha et al. [30] demonstrated that excessive endurance loading in mice leads to increased collagen deposition, suppressed AMPKα signalling, and enhanced expression of proinflammatory cytokines and pathological hypertrophy markers, changes consistent with fibrosis driven by chronic biomechanical and neurohumoral stress.

Altogether, the available evidence supports a model in which molecular responses to endurance training unfold along a physiological–pathological spectrum. While moderate exercise maintains extracellular matrix (ECM) equilibrium, sustained or high-intensity training may activate signalling pathways that promote fibrotic remodelling (Fig. 1).

Chronic endurance exercise induces volume and pressure overload, triggering mechanical and neurohormonal responses that activate inflammatory cascades. These processes promote fibroblast transformation, extracellular matrix (ECM) disorganization, and collagen deposition, ultimately contributing to myocardial fibrosis. Illustrated pathways highlight both subclinical myocardial injury and systemic signaling contributing to fibrotic remodelling.

Modifiable and non-modifiable risk factors. The development of myocardial fibrosis in endurance athletes appears to result from a complex interplay between training characteristics and individual predispositions. Among modifiable risk factors, training intensity and cumulative load play dominant roles. Athletes engaging in high weekly volumes – particularly exceeding 10 hours – exhibit elevated left ventricular (LV) mass, increased extracellular volume, and elevated systolic pressure, which have all been associated with fibrotic remodelling [21, 31]. The type of sport further modulates cardiac strain; mixed-discipline events – such as triathlons – may impose greater haemodynamic and adrenergic stress than pure endurance sports, amplifying arrhythmogenic risk [32].

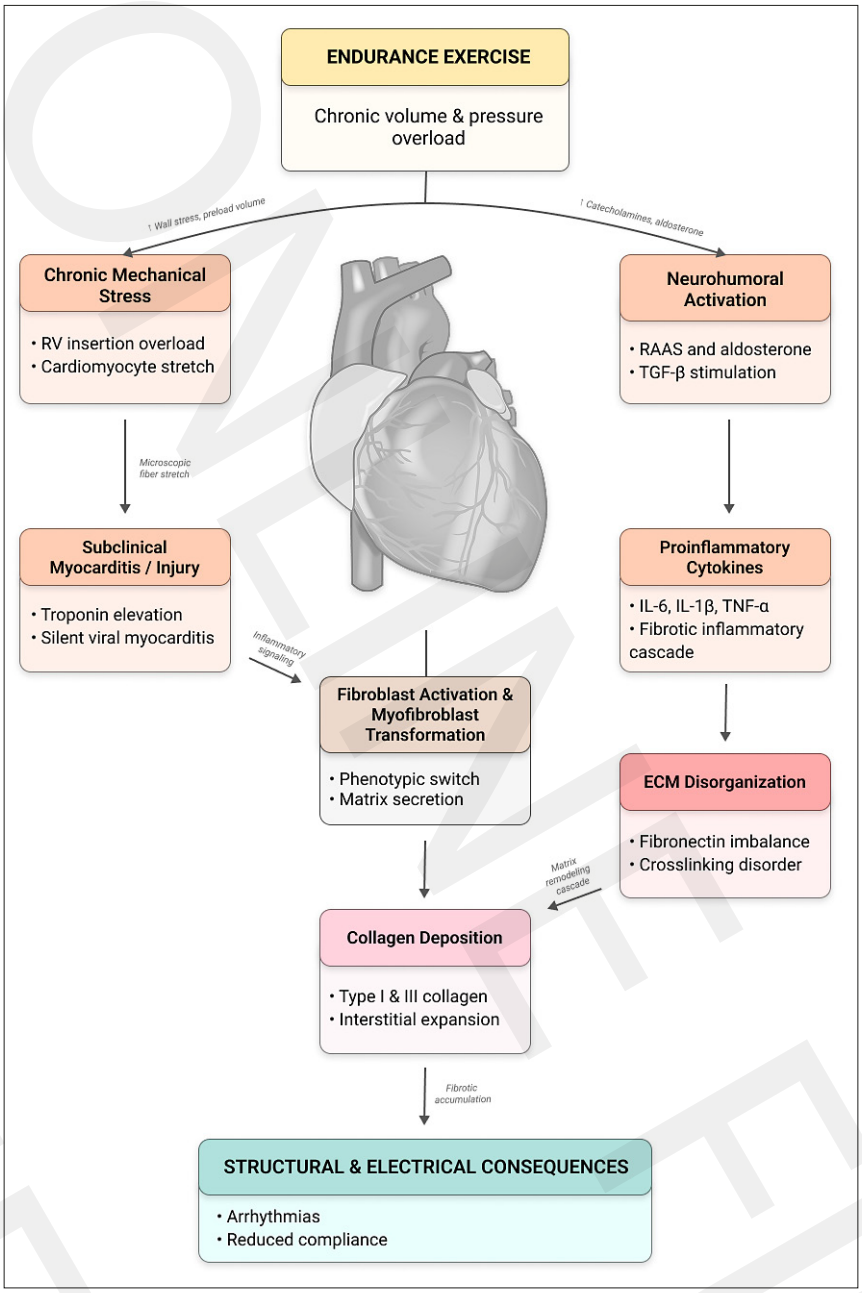

Figure 1. Pathophysiological mechanisms linking endurance exercise to myocardial fibrosis

Structural adaptations also differ by training modality. Endurance athletes frequently show LV dilation and fibrosis at right ventricular (RV) insertion points, whereas strength athletes more often develop concentric hypertrophy without fibrosis [33]. These sport-specific phenotypes support the importance of tailoring the interpretation of imaging findings to the athlete's training profile. However, not all studies consistently identify fibrosis in high-load disciplines, suggesting that individual variability, genetic predisposition, or methodological differences may moderate the apparent association between training modality and fibrotic outcomes. Non-modifiable factors, including age, gender, and body size, further shape the fibrotic response. While younger athletes tend to exhibit localized insertion-point fibrosis, older athletes more commonly demonstrate diffuse or mid-wall patterns [21]. Male gender has been associated with higher prevalence of coronary calcifications and myocardial fibrosis, possibly due to hormonal differences and pro-fibrotic gene expression, whereas estrogen may exert protective effects in females [21]. Larger body surface area also correlates with atrial enlargement and early diastolic dysfunction, adding to the mechanical load on cardiac structures [31].

An underappreciated contributor is viral myocarditis. Training during or shortly after asymptomatic viral infection, particularly with cardiotropic viruses, e.g. coxsackievirus B3, may promote sustained inflammation and fibrosis, especially in male athletes [34].

In a prospective 5-year study, athletes with ischemic LGE had a markedly higher incidence of sudden cardiac death (28%) compared to zero cases among those with non-ischemic patterns [35]. Additionally, these individuals demonstrated a 4-fold increase in ventricular couplets and triplets during Holter monitoring (33% vs. 8%; $p = 0.02$) [27].

Epidemiological data underscore the clinical consequences of these combined factors. The lifetime risk of atrial fibrillation is elevated in athletes, particularly those who begin high-volume training at a younger age or engage in high-strain disciplines. Newman et al. [32] found that athletes under the age of 55 had a greater relative risk of AF than older cohorts, a trend possibly linked to increased vagal tone, adrenergic stimulation, and atrial stretch. These functional changes likely interact with structural remodelling, including fibrosis, to promote arrhythmogenesis over time.

Gender-related differences in myocardial fibrosis (MF) among athletes. MF is increasingly being recognized among endurance athletes. In a systematic review of 3,814 athletes, Allwood et al. [21] found that while overall LGE prevalence was similar between genders (19.7% in men vs. 16.4% in women), fibrosis patterns differed significantly. Nearly all cases in women were minor and localized at RV insertion points, whereas men more frequently exhibited non-ischemic or ischemic fibrosis, often with clinical relevance. Women also showed higher extracellular volume (ECV), possibly reflecting structural differences rather than pathological change. Hormonal and haemodynamic factors may contribute: female athletes tend to have lower peak blood pressure during exertion, less LV hypertrophy, and shorter cumulative training exposure. Estrogen may exert antifibrotic effects via PI3K/AKT and CaMK signalling, limiting collagen deposition and promoting adaptive remodelling, as confirmed in animal models [25].

These findings suggest that female athletes, while not immune to fibrosis, present with less clinically concerning patterns, likely due to protective molecular and physiological mechanisms.

DIAGNOSTIC TOOLS AND INTERPRETATION CHALLENGES

Role of Cardiac Magnetic Resonance (CMR). Cardiac magnetic resonance (CMR) is the cornerstone of non-invasive myocardial tissue characterization, offering unmatched resolution and diagnostic accuracy without ionizing radiation. Its role in athletic populations is particularly valuable, as it captures both focal scarring via late gadolinium enhancement (LGE) and diffuse interstitial changes through parametric mapping, including native T1 and extracellular volume (ECV) quantification.

Beyond simply detecting abnormalities, CMR supports nuanced differentiation between physiological and pathological remodelling—an essential distinction in endurance athletes. As Androulakis et al. [22] underscore, its sensitivity to subtle tissue alterations enables clinicians to assess structural adaptations in the context of training load, age, and sex. Moreover, the technique's reliability is reinforced by Han et al. [36], who showed that CMR-derived ECV aligns closely with histologic fibrosis and sets the standard for validating alternative imaging modalities like CT-ECV.

Beyond its diagnostic capabilities, CMR plays a pivotal role in discerning whether myocardial changes in athletes represent benign remodelling or the onset of disease.

Late Gadolinium Enhancement (LGE) – focal fibrosis detection and diagnostic ambiguity. Late gadolinium enhancement (LGE) is the most established CMR technique for visualizing focal myocardial fibrosis. In endurance athletes, particularly those with prolonged training histories, LGE often appears at the right ventricular (RV) insertion points—regions subjected to intense mechanical load. For instance, Domenech-Ximenos et al. reported a 37.6% prevalence of LGE in competitive triathletes training over 12 hours per week, compared to just 2.8% in sedentary controls, with all enhancement confined to RV insertion sites [37]. These athletes also showed elevated global extracellular volume (ECV), despite preserved chamber size and function, suggesting coexisting subtle diffuse matrix remodelling.

While RV insertion-point fibrosis has long been considered a benign byproduct of physiological adaptation, this interpretation is being challenged. In non-athletic populations, LGE has been strongly associated with adverse outcomes. For example, in patients with myocarditis or dilated cardiomyopathy, LGE independently predicted higher risk of death, ventricular arrhythmias, and major cardiac events—sometimes more so than traditional measures such as ejection fraction [38, 39]. Although these results cannot be directly translated to athletic populations, they underscore the need for caution when LGE is present, particularly outside classic adaptation zones or in conjunction with other risk markers. Importantly, meta-analytic data in non-athletic populations demonstrate that the prognostic impact of LGE depends on its extent and distribution, further supporting the need for nuanced interpretation in athletic cohorts [40].

Innovative approaches may soon offer alternatives to contrast-enhanced imaging. Zhang et al. introduced a virtual native enhancement (VNE) method that reproduces LGE-like tissue contrast using only native T1 and cine MRI data—offering potential for safer, faster fibrosis screening in high-volume athletic cohorts [41].

Overall, while LGE is a powerful tool, its presence in athletes should not be reflexively dismissed as benign. Careful interpretation, incorporating fibrosis pattern, location, and accompanying clinical features, remains essential.

Parametric mapping and interpretation of pitfalls in athletics' hearts. Parametric CMR techniques—including native T1 and T2 mapping, as well as extracellular volume (ECV)—offer a sensitive, non-invasive window into diffuse myocardial remodelling not detectable with traditional late gadolinium enhancement (LGE). These methods are particularly relevant for athletes, in whom subtle interstitial changes may precede overt structural abnormalities.

In a series of meta-analyses and reviews, native T1 mapping was found to outperform the Lake Louise Criteria in sensitivity for active myocarditis, while ECV mapping uniquely identified diffuse matrix expansion not visible via LGE [42, 43]. Wang and Li additionally emphasized that these markers provide continuous, tissue-level insight into fibrosis and inflammation, potentially eliminating the need for biopsy in certain clinical scenarios [44]. However, their application in asymptomatic athletes is still emerging. Small shifts in native T1 or ECV may reflect benign adaptive remodelling—such as increased plasma volume or augmented capillary density—rather than pathology. The risk of overdiagnosis is heightened in the absence of sport- and gender-specific normative reference values.

Indeed, in elite female athletes, features such as right ventricular enlargement or T-wave inversions may mimic

arrhythmogenic right ventricular cardiomyopathy (ARVC), despite benign outcomes [45]. Similarly, a meta-analysis in male athletes showed that RV dilation was common yet unaccompanied by dysfunction, reinforcing the need to distinguish physiological from pathological patterns [46].

In summary, while parametric mapping provides powerful tools for tissue-level characterization, their interpretation in athletic hearts must be contextualized. Without longitudinal data and athlete-specific cut-offs, even advanced imaging may blur the line between adaptation and disease.

CLINICAL IMPLICATIONS – IS FIBROSIS A WARNING SIGN?

Prognostic value of LGE in non-athletic populations. In individuals with non-ischemic cardiomyopathy, the presence of myocardial fibrosis, detected via late gadolinium enhancement (LGE), has emerged as a robust predictor of adverse cardiovascular outcomes. A meta-analysis encompassing over 7,800 patients across 36 studies reported that LGE was associated with a nearly 4-fold increased risk of ventricular arrhythmias or sudden cardiac death (HR: 3.76), as well as significantly elevated all-cause mortality and major cardiovascular events over a follow-up period extending up to 4.8 years [47]. These associations remained significant, regardless of left ventricular ejection fraction.

Subsequent pooled analyses of more than 10,000 patients with non-ischemic cardiomyopathy confirmed these findings, revealing increased odds of death (OR: 2.9), ventricular arrhythmias (OR: 4.6), and hospitalization for heart failure (OR: 3.4) in LGE-positive individuals [48]. Additional data demonstrated that LGE not only predicts arrhythmic events but also correlates with the absence of reverse remodelling in dilated cardiomyopathy, whereas LGE-negative patients typically exhibit more favourable outcomes [49]. Mechanistically, these prognostic effects are thought to arise from the arrhythmogenic properties of fibrotic tissue. Patchy or mid-wall scarring disrupts electrical homogeneity, fostering slow conduction zones and re-entrant circuits that increase the susceptibility to malignant ventricular arrhythmias. Although often subclinical, such structural abnormalities are considered markers of irreversible myocardial remodelling.

It is important to note that these data are derived from populations with established cardiac disease. While the physiological remodelling observed in endurance athletes may lead to similar LGE patterns, extrapolating these risk associations directly to athletic cohorts remains problematic. Nonetheless, the consistent link between LGE and adverse outcomes in pathological contexts underscores the need for cautious and context-sensitive interpretation of fibrotic findings in athletes, particularly when detected outside classic adaptation zones.

LGE in athletes – from diagnostic uncertainty to evidence-based risk stratification. Late gadolinium enhancement (LGE) is a well-established marker of adverse outcomes in cardiomyopathy, yet its prognostic value in endurance athletes remains uncertain. In this population, LGE typically occurs in asymptomatic individuals with preserved function and is most often confined to regions of mechanical stress, such as the right ventricular insertion points.

Robust data from non-athletic cohorts, however, challenge the assumption of benignity. Meta-analyses involving over 15,000 patients with non-ischemic cardiomyopathy have shown that the presence of LGE is linked to a 3 – 5-fold increased risk of ventricular arrhythmias and sudden cardiac death, as well as significantly higher cardiovascular and all-cause mortality [39,50]. This evidence indicates that fibrosis, regardless of its underlying cause, has the potential to disrupt myocardial conduction, facilitate re-entrant circuits, and increase susceptibility to arrhythmias. Whether the same holds true for athletes with structurally normal hearts remains unclear. There are no large prospective studies tracking LGE-positive athletes to determine clinical outcomes. However, a 5-year follow-up of endurance athletes found that ischemic LGE, present in just 2% of participants, was associated with a 28% incidence of sudden cardiac death, while those with non-ischemic patterns experienced no such events [35]. This highlights the importance of not only the presence but also the pattern and location of fibrosis in risk assessment.

Kiaos et al. [51] further demonstrated that in non-athletes, LGE involving ≥10% of left ventricular mass predicted a 5-fold increase in sudden cardiac death, out-performing traditional prognostic markers such as wall thickness or ejection fraction. Whether such thresholds apply to athletes is unknown. In response, Ahmad et al. [27] called for athlete-specific risk stratification tools that integrate advanced imaging, electrophysiological findings, and training metrics, resources still absent from standard clinical care.

Technological advances such as MR fingerprinting, T1ρ imaging, and AI-driven tissue characterization, are promising, yet remain unvalidated in athletic populations [52]. Without normative data and outcome-based validation, their utility in return-to-play decisions remains constrained.

Until more evidence becomes available, fibrosis in athletes should be interpreted with individualized caution. Clinical decisions, particularly regarding eligibility for high-intensity training, should consider fibrosis distribution, arrhythmic burden, and personal or family history. A structured protocol incorporating ECG, Holter monitoring, cardiopulmonary testing, and serial imaging remains essential for accurate risk stratification [53].

To bridge current gaps, prospective multi-centre registries are urgently needed. These should follow LGE-positive athletes across fibrosis types, sport disciplines, gender, and training exposure, with long-term tracking of arrhythmias, function, and outcomes to enable evidence-based models for screening, counselling, and return-to-play management.

SUMMARY

Myocardial fibrosis in endurance athletes reflects a complex and often subclinical continuum between adaptive remodelling and emerging pathology. Although commonly localized to right ventricular insertion points and considered benign, certain fibrosis patterns, such as mid-wall or ischemic, may represent arrhythmogenic substrates with potential prognostic implications.

Cardiac magnetic resonance (CMR) remains the gold standard for fibrosis detection, but interpreting findings in athletes is complicated by physiological overlap and the absence of validated, outcome-based thresholds. While

studies in cardiomyopathy cohorts suggest that even limited fibrotic burden portends elevated risk, direct extrapolation to healthy athletes remains speculative. This underscores a pressing knowledge gap. Without longitudinal, phenotype-specific data, clinicians risk either over-medicalizing benign adaptations or missing early signs of cardiovascular vulnerability. Addressing this uncertainty requires prospective, multi-centre studies that stratify athletes by fibrosis pattern, sport type, gender, and training load, correlated with arrhythmic, functional, and performance outcomes.

Until such evidence becomes available, clinical decision-making must be individualized. Risk stratification should incorporate fibrosis location and extent, symptomatology, training history, and history of myocarditis. Return-to-play decisions are best guided through a multidisciplinary lens, engaging cardiologists, sports medicine physicians, and allied professionals, to ensure that performance optimization does not compromise long-term cardiovascular health.

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