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Myocardial Fibrosis in Long-Distance Runners: From Physiological Adaptation to Potential Pathology - Prevalence, Imaging Characteristics, and Clinical Relevance

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Abstract

In recent years, the popularity of long-distance running has increased considerably. Regular physical activity is widely recognized as an effective strategy for improving cardiorespiratory fitness, reducing the risk of cardiovascular disease, diabetes, obesity, multiple cancers, and ultimately contributing to increased life expectancy. Despite these well-established benefits, accumulated evidence suggests that prolonged exposure to high-volume endurance exercise may be associated with adverse structural cardiac remodeling in endurance athletes. Studies employing mainly cardiac magnetic resonance imaging (CMR) have identified myocardial abnormalities that may exceed the spectrum of physiological adaptation. Available data indicate

the presence of myocardial fibrosis in some endurance athletes. Further research is needed to determine the clinical implications of these findings; however, several studies indicate that such changes may serve as a substrate for cardiac arrhythmias and, consequently, increase the risk of a sudden cardiac death. The aim of this review is to summarize current evidence regarding structural myocardial changes in long-distance runners, with particular emphasis on the prevalence, mechanisms, diagnostic assessment, and potential clinical consequences of myocardial fibrosis.

Materials and Methods: A literature review was conducted using studies available in the PubMed and Google Scholar databases. The analysis included primarily original research articles, meta-analyses, and review papers focusing on myocardial fibrosis in endurance athletes.

Keywords: myocardial fibrosis, endurance athletes, marathon runners, late gadolinium enhancement, cardiac MRI, sudden cardiac death

1. Introduction

Over the past decades, participation in marathon running has increased dramatically, particularly among older adults and women. An analysis of more than 696,000 marathon finishes in the Berlin Marathon between 1974 and 2019 demonstrated a continuous rise in participation across all age groups. As the number of individuals exposed to long-term endurance training continues to grow, interest has also increased regarding its potential long-term cardiovascular effects. [6]

In recent years, increasing emphasis has been placed on the importance of physical activity for maintaining health. Numerous scientific societies, including the World Health Organization (WHO), recommend at least 150–300 minutes of moderate-intensity aerobic physical activity per week or 75–150 minutes of vigorous-intensity aerobic activity. However, available

evidence indicates that an increasing number of individuals engage in substantially higher volumes of exercise.[8]

The Copenhagen City Heart Study initiated the discussion on the existence of an upper limit of beneficial exercise, commonly referred to as the “exercise paradox”. The study suggested that the health benefits associated with running may not increase linearly with training volume. The lowest mortality rates were observed among recreational runners who jogged 1–2.4 hours per week, whereas strenuous runners - defined as individuals running 4–6 times per week, at a fast pace, or with high training volumes - did not demonstrate a significantly lower risk of death compared with sedentary individuals. Owing to the small number of participants classified as strenuous joggers, the study does not provide conclusive evidence that high-volume endurance exercise is harmful to cardiovascular health. Nevertheless, it stimulated further research into the potential adverse consequences of long-term endurance training, including myocardial fibrosis, atrial fibrillation, ventricular arrhythmias, and other forms of exercise-related cardiac remodeling.[5,7]

Long-term endurance training induces a range of structural and functional cardiovascular adaptations collectively referred to as the “athlete’s heart”. These adaptations include enlargement of the cardiac chambers, increased ventricular wall thickness, enhanced diastolic function, and autonomic nervous system remodeling. In most athletes, these changes are considered physiological and reversible. However, growing evidence suggests that in a subset of individuals exposed to many years of intensive endurance exercise, cardiac remodeling may extend beyond physiological adaptation and involve potentially pathological changes.[1-3,16]

2. Myocardial Fibrosis: Definition, Types, and Potential Mechanisms in Athletes

2.1 Definition and pathophysiology of Myocardial fibrosis

Myocardial fibrosis (MF) refers to the excessive deposition of extracellular matrix within the cardiac muscle. MF is a multifactorial process in which prolonged and repetitive hemodynamic overload plays a central role. During intense exercise, substantial increases in stroke volume, intracardiac pressure, and ventricular wall stress result in mechanical stimulation of cardiac fibroblasts and enhanced synthesis of collagen types I and III.[9]

Oxidative and inflammatory stress have also been implicated in exercise-induced myocardial remodeling. Prolonged exercise may increase the production of reactive oxygen species and

stimulate the release of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α). These factors further activate cardiac fibroblasts and promote the accumulation of extracellular matrix components.[2,9]

Evidence suggests that repeated episodes of intense exercise may induce microinjury to cardiomyocytes. Recurrent microdamage can lead to apoptosis or necrosis of myocardial cells, which are subsequently replaced by fibrotic tissue, a process known as replacement fibrosis (Allwood et al., 2024).[9,10]

Under normal training conditions, a transient remodeling of the extracellular matrix (ECM) occurs, leading to improved myocardial stiffness and functional efficiency. This process is regulated by a balance between matrix metalloproteinases (MMPs) and their tissue inhibitors (TIMPs). In such circumstances, the changes are reversible and represent a physiological adaptation to exercise.[9]

However, when the training load is extremely high, sustained over many years, or when an individual has a genetic predisposition, this balance may become disrupted. The activity of profibrotic mediators, including transforming growth factor-beta (TGF-β), angiotensin II, and connective tissue growth factor (CTGF), increases. As a result, excessive collagen deposition, collagen cross-linking, and reduced myocardial compliance occur.[9]

Regression of myocardial fibrosis after a reduction in training load would favour the hypothesis of a physiological adaptation to exercise. Conversely, persistent or progressive fibrosis despite detraining may be indicative of pathological myocardial remodeling.

2.2 Classification of Myocardial fibrosis

Myocardial fibrosis encompasses several distinct forms that differ in their histological characteristics and underlying pathophysiology. The table below summarizes the major types of myocardial fibrosis and the mechanisms involved in their development.

Table 1. Characteristics of the Main Types of Myocardial Fibrosis

Feature	Reactive Interstitial Fibrosis	Infiltrative Interstitial Fibrosis	Replacement Fibrosis
Mechanism	Excessive collagen synthesis by activated fibroblasts without cardiomyocyte loss	Deposition of abnormal substances within the myocardial interstitium	Replacement of dead cardiomyocytes with fibrotic tissue
Cause	Chronic pressure or volume overload, RAAS activation, oxidative stress, and inflammation	Deposition of amyloid proteins or glycosphingolipids	Cardiomyocyte necrosis, apoptosis, or injury
Distribution	Diffuse, often perivascular	Diffuse	Focal or extensive scar formation
Reversibility	Potentially reversible	Partially reversible following treatment of the underlying disease	Irreversible
Detection by CMR	Best assessed using T1 mapping and extracellular volume (ECV) quantification	Best assessed using T1 mapping and ECV quantification	Primarily detected by late gadolinium enhancement (LGE)
Clinical Significance	Early marker of myocardial remodeling	Marker of infiltrative/storage diseases	Advanced myocardial damage associated with adverse prognosis

Source: based on Mewton N et. al 2011[11]; Abbreviations: CMR – cardiac magnetic resonance; ECV – extracellular volume; LGE – late gadolinium enhancement; RAAS – renin–angiotensin–aldosterone system.

A key consideration in the study of myocardial fibrosis in athletes is the distinction between diffuse, potentially reversible interstitial fibrosis and focal, irreversible replacement fibrosis.

3. Diagnostic Approaches to Myocardial Fibrosis

3.1 Cardiac Magnetic Resonance Imaging

Cardiac magnetic resonance (CMR) is widely regarded as the reference standard for the non-invasive assessment of myocardial fibrosis. Its high spatial resolution enables detailed characterization of myocardial tissue and facilitates the detection of even small fibrotic lesions that may not be identified using other imaging modalities.[11]

3.2 Late Gadolinium Enhancement (LGE)

Late gadolinium enhancement (LGE) imaging is based on the preferential accumulation of gadolinium-based contrast agents within regions characterized by expansion of the extracellular space, including post-infarction scars, replacement fibrosis, and areas of chronic myocardial injury. Differences in contrast uptake and washout between normal and diseased myocardium enable the visualization and quantification of fibrotic tissue. LGE has demonstrated high sensitivity for the detection of focal myocardial fibrosis, particularly replacement fibrosis. However, its ability to identify diffuse interstitial fibrosis is limited, as homogeneous distribution of fibrosis throughout the myocardium may reduce the contrast differences required for lesion detection.[11,21]

3.3 T1 Mapping

T1 mapping has emerged as a quantitative CMR technique for myocardial tissue characterization. By measuring native myocardial T1 relaxation times and, when combined with contrast-enhanced imaging, calculating the extracellular volume fraction (ECV), this method provides an indirect assessment of diffuse interstitial fibrosis.[11]

In contrast to LGE, T1 mapping is capable of detecting subtle and diffuse alterations in myocardial tissue composition before the development of focal fibrotic lesions. Consequently, it has become an important tool for the evaluation of early myocardial remodeling and diffuse extracellular matrix expansion.

3.4 Complementary Role of LGE and T1 Mapping in the Assessment of Myocardial Fibrosis

LGE and T1 mapping provide complementary information regarding myocardial structure and tissue composition in athletes. LGE remains the preferred technique for the identification of

focal replacement fibrosis and the assessment of arrhythmic risk, whereas T1 mapping and ECV quantification facilitate the evaluation of diffuse interstitial fibrosis. Importantly, diffuse fibrotic changes detected by T1 mapping may reflect either early pathological remodeling or physiological adaptation to long-term endurance training. Therefore, a comprehensive assessment of myocardial fibrosis in athletes increasingly relies on the combined application of both CMR techniques.

4. Frequency of Myocardial Fibrosis in Long-Distance Runners

4.1 Prevalence of Myocardial Fibrosis: Evidence from recent studies

Available evidence suggests that myocardial fibrosis is identified in approximately 10–25% of endurance athletes, depending on the studied population and imaging criteria used. Table 2 summarizes the findings of selected studies and meta-analyses published between 2009 and 2024. The included investigations primarily assessed competitive endurance athletes participating in sports characterized by a predominance of aerobic exercise, including marathon running, ultramarathon running, triathlon, road cycling, and cross-country skiing. Most participants had engaged in regular endurance training for at least 10–20 years. A common characteristic of the included cohorts was the predominance of male athletes, with several studies enrolling exclusively men. The mean age of participants generally ranged between 40 and 60 years. Individuals with known coronary artery disease, cardiomyopathy, heart failure, or significant cardiovascular symptoms were typically excluded from participation.

Comparison of athlete and control groups demonstrated a consistently higher prevalence of myocardial fibrosis among endurance athletes. These findings may indicate an association between long-term exposure to high-volume endurance exercise and myocardial remodeling. However, whether such remodeling represents a physiological adaptation or a pathological process remains a matter of ongoing debate.

Table 2. Prevalence of Myocardial Fibrosis Detected by Late Gadolinium Enhancement in Endurance Athletes: Summary of Studies Published Between 2009 and 2024

Study	Study population	Athlete type	Athletes with LGE +	Control Group with LGE +	Prevalence of LGE+ (%)
Breuckmann et al., 2009	102	Marathon runners	12/102	4/102	11,80
Wilson et al., 2011	12	Veteran endurance athletes	6/12	0 cases detected	50,00
Zhang et al., 2020	772	Endurance athletes	163/772	19/587	21,1
Ragab et al., 2023	74	Marathon runners	sie.74	0/36	11
Allwood et al., 2024	1595	Endurance athletes	378/1595	20/602	23,7

Source: Own elaboration based on Breuckmann et al. (2009), Wilson et al. (2011), Zhang et al. (2020), Ragab et al. (2023), and Allwood et al. (2024)[10,12-15]

Several limitations should be considered when interpreting these findings. Most studies included relatively small sample sizes and heterogeneous populations comprising veteran marathon runners, Ironman triathletes, and older endurance athletes. Consequently, the results may not be generalizable to all long-distance runners, particularly recreational athletes with lower cumulative training exposure. Furthermore, the higher prevalence of fibrosis reported in these cohorts may overestimate the true prevalence within the broader endurance-running population.

4.2 Patterns and Location of Late Gadolinium Enhancement (LGE)

The studies presented in the table 2. also examined the most common locations of myocardial fibrosis detected by late gadolinium enhancement (LGE) imaging.

4.2.1 Right Ventricular Insertion Points

The most frequently reported location of LGE was at the right ventricular insertion points. Fibrotic lesions were predominantly located in the basal and mid-ventricular segments of the interventricular septum. These findings are thought to be associated with chronic hemodynamic overload of the right ventricle resulting from prolonged endurance exercise. Allwood et al. (2024) classified this pattern as minor fibrosis, as the extent of LGE was generally small, and similar findings have also been observed in healthy older individuals and patients with pulmonary hypertension. Therefore, this pattern is generally considered to have a relatively benign clinical significance.[10,12,14]

4.2.2 Mid-Myocardial and Subepicardial Left Ventricular Fibrosis

The most common location of non-ischaemic fibrosis was the lateral and inferolateral wall of the left ventricle. This pattern was described as major fibrosis or a non-ischaemic pattern, as the distribution of LGE did not correspond to the perfusion territory of a single coronary artery. The presence of this type of fibrosis may reflect a previous inflammatory process, the accumulation of exercise-induced myocardial microinjury, or more advanced fibrotic remodelling. Furthermore, it has been associated with an increased risk of ventricular arrhythmias.[12-14]

4.2.3 Interventricular Septum

LGE located within the interventricular septum was reported relatively infrequently. These lesions were observed primarily in older endurance athletes and most commonly demonstrated a mid-myocardial or subepicardial distribution. The clinical significance of this pattern remains uncertain; however, its presence may indicate more advanced myocardial remodelling processes.[12-15]

The clinical relevance of LGE in endurance athletes extends beyond its mere presence and anatomical distribution. Increasing evidence suggests that the prognostic significance of myocardial fibrosis may depend on its extent, pattern, and location within the myocardium.

Consequently, understanding the potential clinical implications of these findings is essential for assessing the long-term cardiovascular consequences of endurance training.

5. Clinical Consequences of Myocardial Fibrosis

Several studies and meta-analyses have demonstrated that ventricular fibrosis detected by late gadolinium enhancement (LGE) is a powerful predictor of ventricular tachyarrhythmic events in patients with ischaemic cardiomyopathy, non-ischaemic dilated cardiomyopathy, and hypertrophic cardiomyopathy [19].

5.1 Atrial fibrillation

An increasing body of evidence suggests that long-term high-intensity endurance exercise may promote atrial remodelling. The principal mechanisms implicated in this process include enlargement of both the left and right atria, an increased burden of supraventricular ectopic beats, enhanced parasympathetic activity (increased vagal tone), bradycardia, inflammatory changes, and atrial fibrosis [17,18].

A meta-analysis by Wilhelm (2013) demonstrated an approximately fivefold higher risk of atrial fibrillation (AF) among middle-aged endurance athletes compared with non-athletic controls. Although direct evidence of atrial fibrosis in endurance athletes remains limited, growing experimental and clinical evidence suggests that fibrosis plays a significant role in the pathogenesis of AF. Fibrotic remodelling may disrupt normal electrical conduction within the atrial myocardium, thereby creating a substrate that facilitates the initiation and maintenance of atrial fibrillation. Consequently, atrial fibrosis is increasingly recognised as one of the key mechanisms linking long-term endurance exercise to the elevated incidence of AF observed in endurance athletes. [18]

5.2 Ventricular Arrhythmias

Long-term endurance exercise has been associated with right ventricular remodelling and myocardial fibrosis, both of which may provide a substrate for ventricular arrhythmias. As a consequence, athletes may develop premature ventricular beats, non-sustained ventricular tachycardia, or sustained ventricular tachycardia.

5.2.1 Premature Ventricular Complexes (PVCs)

Premature Ventricular Complexes (PVCs) are relatively common among athletes and are generally considered to have limited clinical significance. Nevertheless, their presence often warrants a comprehensive evaluation, including assessment of symptoms, family history of cardiovascular disease, and the overall burden of ventricular ectopy. Particular attention should be paid when the number of PVCs exceeds 2,000 per 24 hours [16]

Frequent PVCs resulting from focal automaticity arising from the ventricular outflow tracts or from the fascicles of the left bundle branch system are relatively common and, in the absence of structural heart disease, are generally regarded as benign. In contrast, other morphologies, such as PVCs displaying a left bundle branch block pattern, a broad right bundle branch block pattern, or an intermediate or superior axis, are less common and should prompt further investigation to exclude underlying structural heart disease or cardiomyopathy [16]. Myocardial fibrosis may contribute to the development of ventricular ectopy by creating areas of heterogeneous electrical conduction and delayed activation, thereby facilitating the initiation of ventricular arrhythmias.

5.2.2 Exercise-Induced Arrhythmogenic Right Ventricular Cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy (ARVC), currently considered part of the broader spectrum of arrhythmogenic cardiomyopathies (ACM), is characterized by progressive replacement of the cardiomyocytes with fibrous and fatty tissue. The pathological process typically begins in the right ventricle and leads to focal abnormalities of contractility, eventually progressing to more diffuse right ventricular dysfunction as the disease advances [22,25].

Studies have shown that endurance athletes with ventricular tachycardia often exhibit right ventricular structural abnormalities resembling classical ARVC, despite the absence of disease-causing genetic mutations. This observation has contributed to the hypothesis of exercise-induced ARVC, whereby prolonged intensive endurance exercise may promote myocardial remodelling and increase the risk of ventricular arrhythmias. Such a mechanism may partly account for the higher prevalence of ventricular arrhythmias observed in some long-distance runners. Importantly, Casian et al. (2024) emphasized that distinguishing ARVC from physiological exercise-induced cardiac remodelling remains a significant diagnostic challenge.[22,25]

5.3 Sudden Cardiac Death (SCD)

Sudden cardiac death describes the unexpected natural death from a cardiac cause within a short time period, generally ≤1 hour from the onset of symptoms, in a person without any prior condition that would appear fatal [23].

In a large prospective study, Leyva et al. demonstrated that myocardial fibrosis was an independent predictor of ventricular arrhythmias and SCD, even after adjustment for age, disease etiology, and left ventricular ejection fraction (LVEF). Notably, all cases of sudden cardiac death occurred in patients with detectable myocardial fibrosis on cardiac magnetic resonance imaging (CMR) [21].

Particularly high risk was observed among individuals with extensive gray zone fibrosis, defined as regions of heterogeneous fibrotic tissue located between healthy myocardium and dense scar. Within these areas, surviving strands of cardiomyocytes are interspersed with fibrotic tissue, creating zones of slow and heterogeneous electrical conduction that facilitate the development of re-entry circuits. Consequently, gray zone fibrosis is regarded as a highly arrhythmogenic substrate capable of promoting malignant ventricular tachyarrhythmias and increasing the risk of sudden cardiac death.[21]

6. Conclusions

In summary, current evidence indicates that regular physical activity provides numerous health benefits, including improved quality of life, reduced cardiovascular risk, and increased life expectancy. It is well established that long-term endurance exercise induces a range of physiological cardiovascular adaptations, collectively referred to as the “athlete’s heart”. However, growing evidence suggests that, in a subset of individuals exposed to prolonged and intensive endurance training, cardiac remodeling may extend beyond physiological adaptation and involve potentially pathological changes. The exact threshold of exercise exposure associated with an increased risk of adverse cardiac remodeling remains unknown and requires further investigation. Based on the available literature, myocardial fibrosis appears to be more prevalent among endurance athletes than in the general population. It is most commonly detected as focal late gadolinium enhancement (LGE) on cardiac magnetic resonance imaging, typically of a non-ischemic pattern and frequently located at the right ventricular insertion points. Nevertheless, important uncertainties remain regarding the clinical significance of different LGE patterns. While some forms of fibrosis may represent a benign consequence of

exercise-induced remodeling, several studies suggest that subepicardial and mid-myocardial fibrosis of the left ventricle may serve as a substrate for ventricular arrhythmias. The relationship between exercise-associated myocardial fibrosis and sudden cardiac death also remains incompletely understood. Large-scale prospective studies with long-term follow-up are needed to determine the true prevalence of myocardial fibrosis, clarify its underlying mechanisms, and establish its clinical significance. Future research should include adequate representation of both sexes and evaluate the influence of age, training volume, and exercise intensity on the development of myocardial fibrosis and its potential cardiovascular consequences.

DISCLOSURES

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