

# The Evolving Role of Cardiovascular MRI in Sports Cardiology: From Athletic Adaptation to Cardiac Pathology

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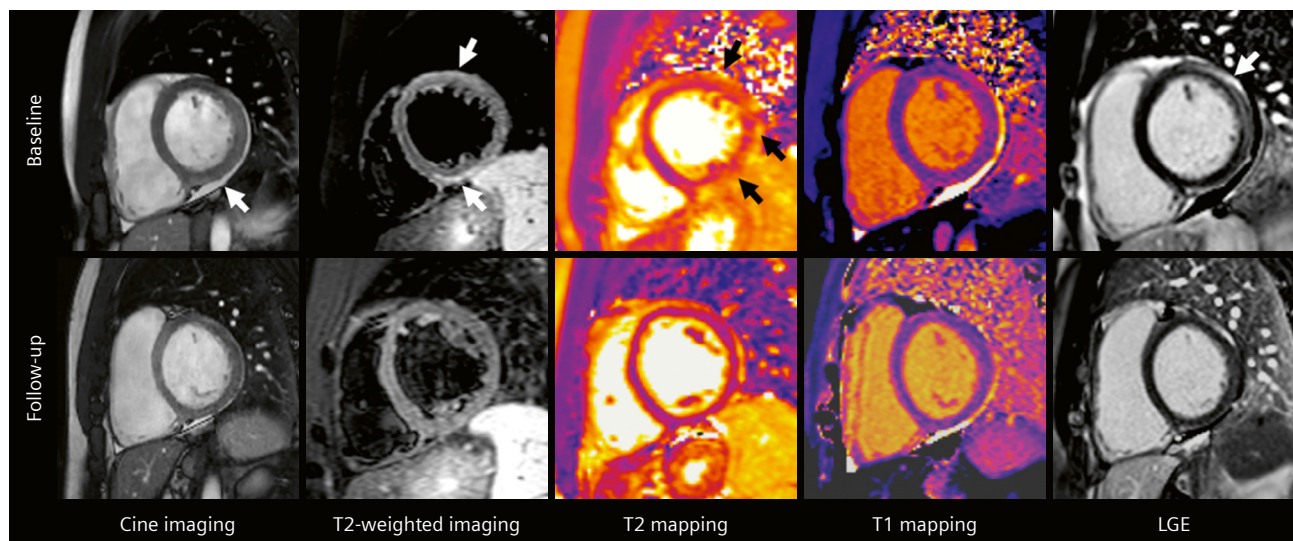
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## Introduction

Sports cardiology has evolved into a distinct subspecialty of cardiology dedicated to the prevention, diagnosis, and management of cardiovascular disease in athletes, highly active individuals, and patients wishing to participate safely in physical activity. Originally developed to reduce the incidence of sudden cardiac death (SCD) in young athletes, the field now encompasses pre-participation screening, evaluation of cardiovascular symptoms, risk stratification, and return-to-play (RTP) decision-making [1].

Concurrently, the demographic landscape of athletic participation has shifted significantly; an exponential rise in middle-aged and older master athletes engaging in extreme, high-volume endurance events (such as marathons and ultramarathons) has introduced a new set of age- and volume-specific cardiovascular challenges.

At the same time, physical activity is increasingly regarded as a therapeutic intervention. Exercise is a cornerstone of cardiovascular prevention, rehabilitation, and



- 1** Cardiovascular magnetic resonance (CMR) findings in a professional soccer player with acute myocarditis (IMPS). From left to right: Cine imaging, T2-weighted black-blood imaging, T2 mapping, T1 mapping, and late gadolinium enhancement (LGE) imaging. The baseline CMR study (top row, October 2024) demonstrates inferolateral myocardial edema with associated subepicardial LGE. Follow-up CMR (bottom row, December 2024) shows complete resolution of edema on T2-weighted and mapping sequences, with persistent limited residual inferolateral fibrosis on LGE imaging, consistent with healed myocarditis.

chronic disease management. Consequently, sports cardiologists are frequently confronted with a fundamental challenge: distinguishing physiological adaptations of the athlete's heart from pathological cardiovascular abnormalities [2]. In this context, cardiac magnetic resonance imaging (CMR) has emerged as an important diagnostic tool in modern sports cardiology. Its unique ability to characterize myocardial structure, function, tissue composition, fibrosis, and inflammation, and the possibility of anatomical evaluation of the coronary arteries make CMR indispensable for the assessment of athletes with suspected cardiovascular disease [3].

### **CMR and inflammatory myopericardial syndrome**

The recently introduced concept of inflammatory myopericardial syndrome (IMPS), which encompasses myocarditis and pericarditis, reflects the frequent clinical and pathophysiological overlap between these inflammatory cardiac disorders [4]. Although many athletes present with mild symptoms or even subclinical disease, inflammatory heart disease remains a major concern in sports cardiology because of its association with ventricular arrhythmias, SCD, and adverse ventricular remodeling, particularly in individuals exposed to high-intensity exercise during the active inflammatory phase [1, 4].

Determining when an athlete can safely return to training and competition following myocarditis remains one of the most challenging clinical scenarios in contemporary sports cardiology. European and North American recommendations have previously advocated temporary exercise restriction for at least 3–6 months after clinically overt myocarditis, followed by comprehensive reassessment including electrocardiography, ambulatory rhythm monitoring, exercise testing, biomarkers, and multimodality imaging before return-to-play was considered [1]. Among all diagnostic modalities, CMR has now emerged as the cornerstone of both diagnosis and follow-up as outlined in current IMPS guidelines [4]. The updated Lake Louise criteria have substantially improved the diagnostic accuracy of CMR by combining tissue characterization techniques such as native T1 mapping, T2 mapping, extracellular volume assessment, and late gadolinium enhancement (LGE). These techniques permit non-invasive detection of active myocardial inflammation, edema, and irreversible myocardial injury, providing information that cannot be obtained through electrocardiography or echocardiography alone [5]. Importantly, while myocardial edema and elevated T1/T2 values may resolve over time, LGE frequently persists as a marker of residual myocardial fibrosis and

scar formation. Growing evidence indicates that the prognostic significance of CMR extends far beyond diagnosis. Among all CMR findings, the persisting presence of LGE has consistently emerged as one of the strongest predictors of adverse cardiovascular outcomes in myocarditis [6]. Several observational studies and meta-analyses have demonstrated that residual myocardial scar detected by LGE is associated with an increased risk of ventricular arrhythmias, recurrent myocarditis, heart failure events, and cardiovascular mortality, even in patients with preserved left ventricular ejection fraction [6, 7]. Consequently, the absence of active inflammation alone may not be sufficient to permit unrestricted return to competitive sports when significant residual fibrosis remains. Particularly relevant for sports cardiology is the extent of myocardial scar burden. Current recommendations recognize that not all LGE findings carry the same prognostic implications. Athletes with persisting extensive myocardial fibrosis, especially when exceeding 20% of left ventricular mass, appear to have a substantially increased arrhythmic risk and are generally considered unsuitable for high-intensity competitive sports participation [1]. Serial CMR assessment therefore plays a pivotal role in RTP decision-making. Recent IMPS recommendations emphasize that CMR should not be limited to the initial diagnosis but should be incorporated into longitudinal patient management [4]. Repeat CMR examinations may be considered as early as 1–3 months after the initial diagnosis to document resolution of edema and inflammatory activity, particularly in athletes seeking expedited return to training. It is important to emphasize that these investigations may be performed without the use of contrast media in less severe myocarditis. However, persistence of elevated T1/T2 mapping values, ongoing myocardial edema, increasing scar burden, or newly detected ventricular dysfunction should prompt continued exercise restriction and further surveillance [4]. Even after clinical recovery, follow-up CMR remains valuable for monitoring the evolution of residual fibrosis and refining long-term arrhythmic risk stratification.

Accordingly, CMR has evolved from a purely diagnostic tool into an essential component of individualized athlete management. By simultaneously assessing active inflammation, ventricular function, and residual myocardial scar, serial CMR provides the most comprehensive framework currently available for guiding safe RTP decisions after myocarditis. As evidence continues to accumulate regarding the prognostic implications of residual LGE and scar burden, CMR-guided follow-up is likely to become increasingly central to future sports cardiology and IMPS management algorithms.

## Clinical example

A 24-year-old professional soccer player presents with chest discomfort following a viral illness. Cardiac troponin levels are mildly elevated, while echocardiography demonstrates preserved ventricular function. CMR reveals subepicardial LGE in the inferolateral wall with associated myocardial edema, consistent with acute IMPS (Fig. 1).

Competitive sports participation is suspended. Follow-up CMR after six weeks demonstrates complete resolution of myocardial edema, indicating healed myocarditis, with only limited residual fibrosis. Return-to-play decisions are subsequently guided by repeat imaging, exercise testing, rhythm monitoring, and shared decision-making.

Furthermore, CMR provides valuable differential diagnostic information beyond tissue characterization. In a single examination, it enables comprehensive visualization of the coronary anatomy, which is particularly important in young athletes presenting with exertional chest pain. High-resolution, navigator-gated, ECG-triggered 3D whole-heart imaging allows radiation-free reconstruction of the coronary tree with sufficient spatial resolution to exclude high-risk coronary anomalies, such as a left main coronary artery originating from the right sinus of Valsalva with an interarterial course. Confirmation of normal coronary anatomy is pivotal for risk stratification and RTP decision-making, as undetected anomalies may lead to exercise-induced coronary compression, ischemia, myocardial infarction, or fatal ventricular arrhythmias during peak exertion.

## Athlete's heart versus cardiomyopathy: The value of MRI

Cardiomyopathies remain one of the leading causes of sudden cardiac death in young athletes. Distinguishing physiological remodeling from early cardiomyopathic disease is therefore one of the most critical tasks in sports cardiology. Endurance athletes frequently develop increased ventricular volumes, mild chamber enlargement, and lower resting ejection fractions. These adaptations can overlap with features of dilated cardiomyopathy (DCM) or arrhythmogenic cardiomyopathy (ACM), creating significant diagnostic uncertainty [8]. CMR offers unparalleled accuracy for ventricular volumetry and myocardial tissue characterization. It enables clinicians to determine whether observed structural changes are proportional to an athlete's training exposure or represent pathological remodeling. Particularly important is the identification of myocardial fibrosis through LGE imaging. Extensive LGE, ventricular aneurysms, progressive chamber enlargement, or evidence of replacement fibrosis strongly

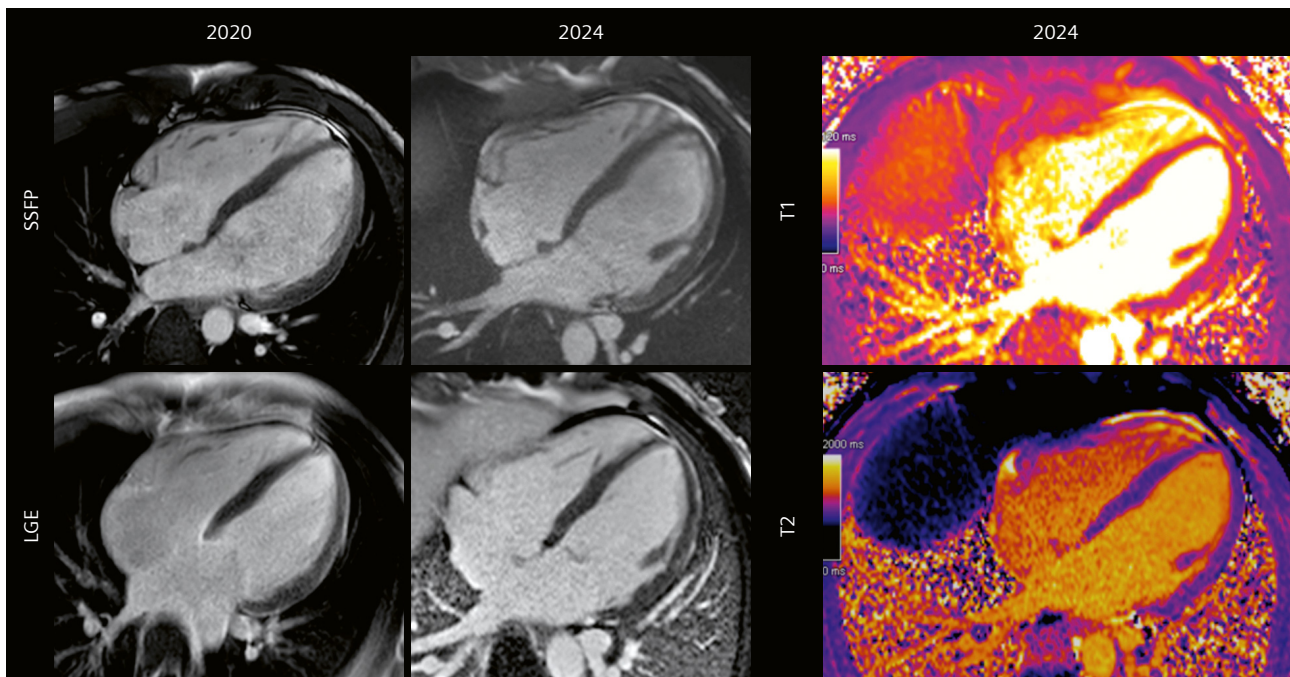
suggest underlying cardiomyopathy rather than physiological adaptation. In cases of persistent diagnostic uncertainty, exercise testing with stress echocardiography — or, in selected cases, bicycle exercise CMR — may further aid differentiation by assessing ventricular functional reserve during physiological stress [9].

## Clinical example

A 30-year-old elite handball player with mixed endurance and strength training underwent serial CMR evaluation after an abnormal echocardiographic finding. In 2020, initial CMR demonstrated marked but symmetrical left ventricular enlargement (LVEDVi 132 mL/m<sup>2</sup>) with mildly reduced systolic function (LVEF 51%) and increased stroke volume index (SVI 69 mL/m<sup>2</sup>), preserved wall motion, normal tissue characterization, and no LGE, consistent with physiological athlete's heart. At follow-up in 2025, left ventricular volumes remained markedly increased (LVEDVi 142 mL/m<sup>2</sup>) with a mildly reduced LVEF of 47% and increased stroke volume index (SVI 67 mL/m<sup>2</sup>), while myocardial mass remained within the expected range for athletic remodeling (78 g/m<sup>2</sup>). The absence of fibrosis, regional wall motion abnormalities, and progressive pathological features supported continued classification as exercise-induced cardiac remodeling rather than dilated cardiomyopathy (Fig. 2).

## MRI in arrhythmogenic and hypertrophic cardiomyopathy

Among cardiomyopathies, hypertrophic cardiomyopathy (HCM) and arrhythmogenic cardiomyopathy deserve special attention due to their association with exercise-related SCD [10]. CMR contributes substantially to risk stratification by detecting myocardial fibrosis through LGE imaging, apical aneurysms, regional ventricular dysfunction, right and left ventricular involvement, and subtle hypertrophy not evident on echocardiography. The extent of LGE has emerged as an important marker of arrhythmic risk and may influence recommendations regarding high-intensity training and competitive sports participation. Current evidence suggests that many athletes with cardiomyopathies can participate safely in sports when high-risk features are absent. However, individuals with HCM-associated obstruction, extensive fibrosis, or ACM generally require restrictions from intensive competitive activities. All such cases require comprehensive counseling by experienced sports cardiologists, thorough education of athletes regarding the associated risks, and a shared decision-making approach to arrive at an informed and individualized clinical recommendation.



**2** Serial cardiovascular magnetic resonance (CMR) findings in an elite handball player with physiological exercise-induced cardiac remodeling. The baseline CMR study (left column, 2020) and follow-up CMR study (middle and right columns, 2025) demonstrate persistent left ventricular enlargement. End-diastolic four-chamber cine images (left and middle columns) show stable chamber dilatation without focal structural abnormalities. Corresponding late gadolinium enhancement (LGE) images demonstrate no focal myocardial fibrosis. Native T1 mapping (top right) and T2 mapping (bottom right) at follow-up show normal tissue characteristics without evidence of diffuse myocardial fibrosis or myocardial edema, consistent with athlete's heart.

## Thoracic aortic disease and the "athlete's aorta"

Athletes frequently exhibit mild enlargement of the thoracic aorta, raising important questions regarding the distinction between physiological adaptation and pathological aortopathy. Beyond the well-recognized concept of the athlete's heart, recent data have introduced the notion of an "athlete's aorta." Although acute increases in blood pressure during resistance and endurance exercise transiently elevate thoracic aortic wall stress, these changes appear modest in healthy athletes and remain far below the estimated biomechanical thresholds associated with aortic rupture. Mild aortic enlargement is observed more frequently in athletes, yet current evidence does not suggest a corresponding increase in aortic dissection risk, supporting the view that such findings largely represent physiological vascular adaptation to long-term training rather than aortic pathology. However, a clear distinction must be made between training-induced aortic remodeling

and underlying pathological conditions; athletes with genetic aortopathies (such as Marfan, Loey-Dietz, or vascular Ehlers-Danlos syndromes, as well as bicuspid aortic valve (BAV)-associated aortopathy) require meticulous risk stratification, stringent serial monitoring, and optimized medical management, in particular aggressive blood pressure control, to minimize the risk of catastrophic aortic events. Further studies are needed to clarify the long-term effects of repetitive exercise-induced wall stress on aortic remodeling. While echocardiography remains the first-line imaging modality, CMR offers comprehensive visualization of the entire thoracic aorta without ionizing radiation. It provides highly reproducible measurements of aortic dimensions and allows serial monitoring over time. Emerging evidence suggests that moderate aortic enlargement in athletes may often represent adaptive remodeling rather than pathological disease [11].

## Clinical example

A 40-year-old male competitive weightlifter presented for routine sports cardiology screening, where echocardiography incidentally revealed a bicuspid aortic valve associated with mild dilation of the ascending aorta. Subsequent magnetic resonance angiography (MRA) confirmed a bicuspid aortic valve layout without significant valvular stenosis or regurgitation, and quantified a maximum ascending aortic diameter of 39 mm. In accordance with the ESC Guidelines on Sports Cardiology and Exercise in Patients with Cardiovascular Disease [1], competitive sports clearance — including high-intensity resistance training — can be safely granted for individuals with BAV when the aortic diameter is below 40 mm, provided there is no severe valvular dysfunction, systemic hypertension, or family history of aortic dissection. Consequently, the athlete was cleared to continue full-scale training and competition under a strategy of stringent clinical surveillance that incorporated annual echocardiographic or CMR reassessments to closely monitor the athlete for progressive aortopathy.

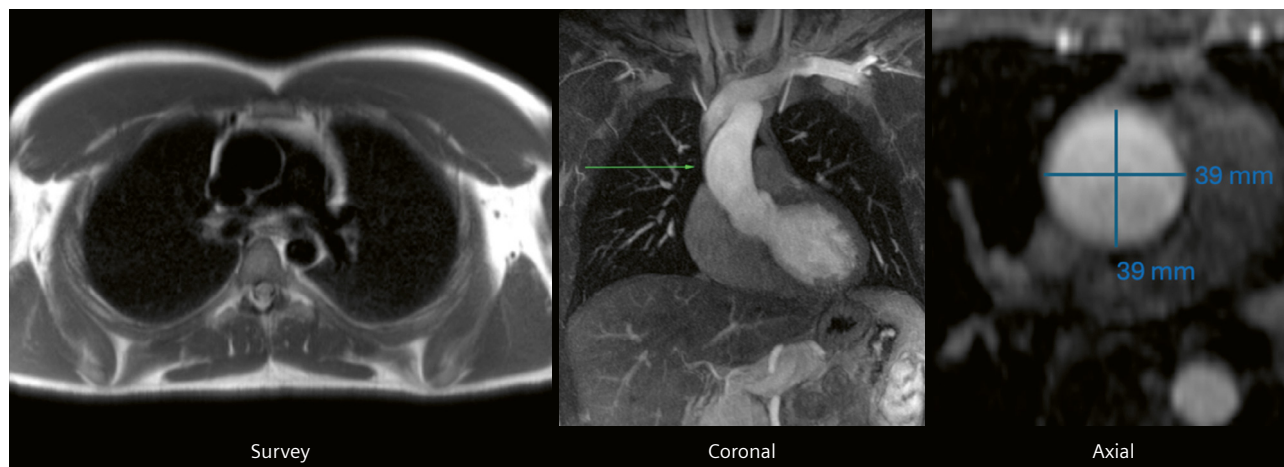
## Coronary artery disease, myocardial scar, and exercise

As coronary computed tomography angiography (CCTA) increasingly identifies subclinical coronary atherosclerosis in athletes, sports cardiologists are confronted with

growing numbers of individuals requiring exercise counseling despite otherwise excellent fitness. Notably, there is growing evidence that endurance athletes exhibit a higher prevalence and burden of coronary plaques compared to less active peers. The acknowledgment of increased coronary atheromatosis in highly active middle-aged master athletes represents a clinical paradox, given that such changes occur even in the absence of traditional cardiovascular risk factors [12]. Although CCTA remains the primary modality for evaluating coronary status, CMR contributes valuable information regarding myocardial viability, ischemia assessment through stress perfusion imaging, detection of silent myocardial infarction, and quantification of scar burden. In athletes with chronic coronary syndrome or previous acute coronary syndrome, CMR can help determine the extent of irreversible myocardial damage and guide exercise prescription [13–17].

## Clinical example

A 62-year-old female endurance athlete with a long-standing history of intensive running and road cycling and without traditional cardiovascular risk factors presented with recent, non-specific performance decline during training. During a routine exercise stress test, she demonstrated non-specific ST-segment depression and a significantly increased burden of exercise-induced ventricular premature complexes (VPCs). Subsequent stress perfusion CMR demonstrated preserved biventricular function, absence



- 3** Magnetic resonance angiography (MRA) findings in a male competitive weightlifter with a bicuspid aortic valve and mild ascending aortic dilatation. The left image (axial survey image) demonstrates enlargement of the ascending aorta relative to the descending thoracic aorta, with prominent pectoral musculature reflecting the athlete's strength-training phenotype. The middle image (coronal reconstruction) and right image (axial reconstruction) of the contrast-enhanced 3D MRA sequence confirm mild ascending aortic dilatation, with a maximum diameter of 39 mm and no additional aortic abnormalities.

of LGE, and an inducible perfusion defect in the inferior wall consistent with myocardial ischemia (Fig. 4). CCTA subsequently revealed a significant proximal right coronary artery (RCA) stenosis of approximately 80%–90%, which was confirmed by invasive coronary angiography (ICA). Successful percutaneous coronary intervention (PCI) with stent implantation of the proximal RCA was performed. Following recurrent chest pain several days after PCI, a follow-up stress perfusion CMR demonstrated resolution of the previously documented ischemia and no evidence of procedure-related myocardial injury. Mild residual impairment of myocardial perfusion reserve not attributed to a coronary territory was found consistent with microvascular dysfunction in the setting of newly developed arterial hypertension. After optimization of blood pressure control and resolution of symptoms over the following weeks, the athlete successfully returned to her usual training regimen. This case highlights the value of stress perfusion CMR for documenting the absence of ischemia in general and of residual inducible ischemia following coronary revascularization, a key requirement for exercise counseling and return-to-play assessment in athletes with coronary artery disease.

### Shared decision-making and the future of imaging in sports cardiology

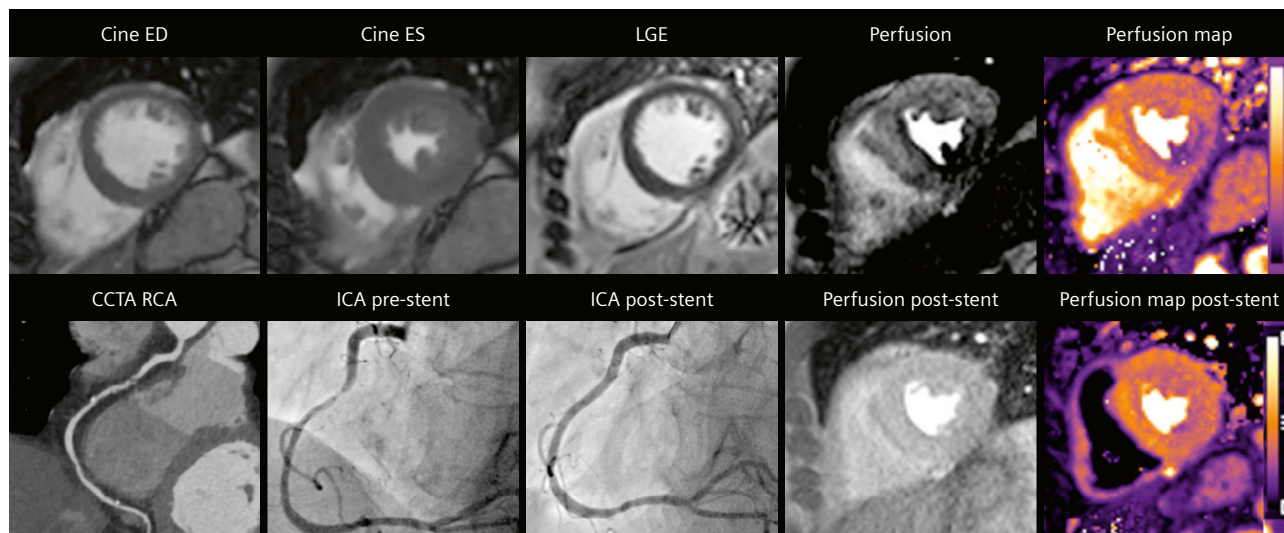
Modern sports cardiology has shifted away from blanket exercise prohibitions and toward individualized risk

assessment and shared decision-making. Athletes increasingly expect personalized recommendations rather than categorical restrictions. CMR is uniquely positioned to support this approach because it provides objective information regarding inflammation, fibrosis, ventricular remodeling, myocardial scar, and aortic pathology. In many cases, imaging findings become the central element around which discussions regarding risk, uncertainty, and athletic goals are conducted. Future developments including parametric mapping, artificial intelligence-assisted image analysis, and advanced tissue characterization are expected to further refine risk prediction and RTP recommendations.

### Conclusion

Cardiac magnetic resonance imaging has become an indispensable tool in contemporary sports cardiology. Whether evaluating inflammatory heart disease, differentiating athlete's heart from cardiomyopathy, assessing arrhythmic risk, monitoring aortic adaptation, or characterizing myocardial scar in coronary disease, CMR provides information that directly influences clinical decision-making.

As sports cardiology increasingly embraces individualized care and shared decision-making, CMR serves not merely as a diagnostic modality but as a central component of athlete-centered cardiovascular risk assessment. Its ability to combine anatomical, functional, and tissue-specific information makes it the cornerstone imaging technique for modern sports cardiology.



**4** Multimodality imaging findings in a 62-year-old female endurance athlete with obstructive right coronary artery disease. The top row shows pre-intervention CMR findings at the mid-ventricular short-axis level, including end-diastolic (ED) cine imaging, end-systolic (ES) cine imaging, late gadolinium enhancement (LGE) imaging, first-pass stress perfusion imaging, and quantitative perfusion mapping. Preserved ventricular function and absence of myocardial scar are demonstrated, while stress perfusion imaging and perfusion mapping reveal an inducible perfusion defect in the inferior wall consistent with ischemia. The bottom row shows coronary computed tomography angiography (CCTA), invasive coronary angiography (ICA) before and after stent implantation in the proximal right coronary artery (RCA), and follow-up stress perfusion CMR at the corresponding mid-ventricular level. Follow-up perfusion imaging and quantitative perfusion mapping demonstrate resolution of the territorial ischemia after revascularization, without evidence of residual inducible ischemia.

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