The Added Value of Cardiac Magnetic Resonance Imaging in the Prevention of Sudden Cardiac Death in Athletes

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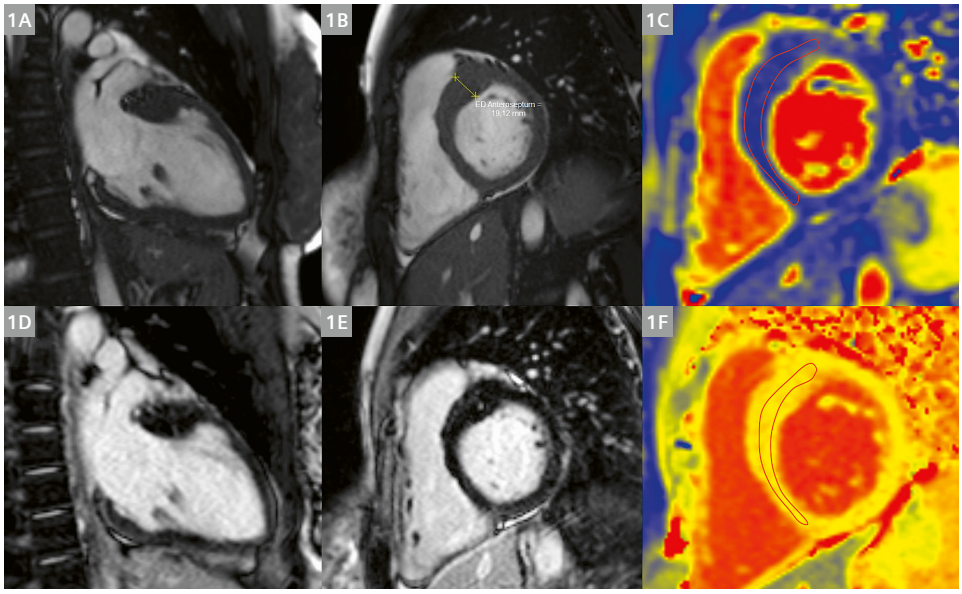
Introduction

Early differentiation of pathological alterations from physiological cardiac adaptation in highly trained athletes is key to preventing sudden cardiac death (SCD) [1]. Athletes engaging in high training loads develop a complex electrical, volumetric adaptation to sport, known as "the athlete's heart" [2, 3]. While not all aspects of physiological adaptation are fully understood today, many characteristics are well described. The imaging features of the athlete's heart include a balanced elevation in left and right cavity sizes, increased myocardial mass and wall thickness, and normal or low normal systolic function at rest compared to sedentary controls [4]. The main factors contributing to the extensiveness of the morphological attributes can be categorized as physiological and pathological. The physiological factors are age, gender, ethnicity, body size, and sports discipline. The pathological factors are illegal performance-enhancing drugs and underlying heart disease [3]. Cardiac magnetic resonance (CMR) imaging is increasingly recognized as an essential second-line imaging modality to diagnose, follow-up, and informed risk stratification in several myocardial diseases such as hypertrophic or arrhythmogenic cardiomyopathy, or acute myocarditis. Recent efforts using novel techniques such as strain analysis or mapping enable more sophisticated differentiation, even in so-called "gray-zone" cases [3]. As CMR is becoming more broadly available, some initiatives have used it as a screening method in collegiate athletes after COVID-19 infection [5]. While this might not be suitable in all cases and could even lead to overdiagnosis of several entities [6], the application of CMR is undoubtedly widening. We summarize the primary applications of CMR in the context of highly trained athletes, and illustrate some real-life CMR cases using a MAGNETOM Area 1.5T MRI scanner from Siemens Healthineers.



1 Young female basketball player with mild phenotypic hypertrophic cardiomyopathy. Images 1A and 1B show cine images in 2-chamber **(1A)** and short-axis **(1B)** views using the CINE segmented (b-SSFP/TrueFISP) sequences. The maximum end-diastolic wall thickness is 19 mm in the basal anteroseptal segment. The T2 mapping **(1C)** is normal (47 ms). Images **(1D, E)** show a very small late gadolinium enhancement in the hypertrophic segment. Image (1F) shows a slightly elevated native T1 mapping value (1012 ms) in the basal septum compared to our in-house normal female athletic values.

Applications of CMR imaging in athletes

Cine sequences are well suited to assessing the athlete's heart in terms of biventricular volumes, function, and myocardial mass. CMR measures these parameters very accurately, showing minimal intra- and interobserver variability [7]. It is also worth mentioning that echocardiography and CMR measurements are not directly comparable. CMR systematically shows larger volumes and smaller wall thickness than echocardiography [8]. Volumetric evaluation of athletes requires athletic controls to prevent false diagnoses and unnecessary restrictions on participating in the competitive sport activity. The cardiac adaptation varies according to gender: Male athletes generally show more pronounced ventricular volumes and myocardial mass than female athletes [9, 10]. Different sports disciplines also lead to slightly altered morphological features: Endurance athletes tend to present with robust cardiac adaptation primarily due to the volume load of the heart, while power athletes such as weightlifters experience states of extreme pressure overload that lead to increased LV wall thickness with virtually unchanged LV volumes [11]. Myocardial deformation imaging has been shown to detect early dysfunction in a number of cardiovascular diseases. Various imaging techniques (e.g., feature tracking, SENC, or DENSE) help to assess myocardial deformation in CMR [12]. Nowadays, thanks to their ease of use, feature-tracking applications using cine images are also gaining popularity in the post-processing analysis of strain imaging. However, there is little data about the typical strain pattern of the athlete's heart.

In addition to precise volumetric measurements and exact morphological evaluation in all myocardial segments, CMR is also capable of non-invasive tissue characterization [7]. Edema is visualized qualitatively with T2-weighted and early gadolinium-enhanced images, and T2 mapping sequences can be used for quantitative assessments. In young athletes, edema-specific sequences are used in acute settings such as acute myocarditis or contusion. Necrosis and fibrosis of the myocardium are visualized on the late gadolinium-enhanced (LGE) images. We acquire LGE images after administering extracellular, gadolinium-based contrast media. At the same time, native T1 mapping and extracellular volume (ECV) provide invaluable information regarding the diffuse extracellular fibrosis or necrosis of the heart. In athletes aged < 35 years, LGE is often caused by myocarditis or different types of cardiomyopathies [3, 7].

It is worth noting that novel evidence also supports the use of normal values for athletic T1 mapping, rather than just site-specific normal values, because native T1 might decrease slightly in cases of physiological hypertrophy [13, 14].

Differential diagnosis and risk stratification in the athlete's heart

Due to the phenotypic overlap between the athlete's heart and early or mild forms of cardiomyopathies, highly trained athletes must be evaluated in cardiovascular centers that deal with a high volume of athletic patients. The use of specific novel CMR techniques is pivotal in some gray-zone cases.

Hypertrophic cardiomyopathy

According to a sizeable forensic registry of competitive athletes in the USA, hypertrophic cardiomyopathy (HCM) is the single most common cause of SCD and it affects male minority athletes even more seriously than other demographic groups [15]. Not surprisingly, diagnosing HCM in highly trained athletes is a considerable challenge, especially in the case of gray-zone left ventricular (LV) hypertrophy with a wall thickness of 13–16 mm [16, 17]. There are, however, some distinctive characteristics that might aid the diagnosis. In a CMR imaging study, the following clues are of importance: focal areas of hypertrophy, where typically asymmetric septal and apical forms are suspicious of pathological alterations, as well as the application of sport indices, showing the ratio between maximum end-diastolic wall width or mass and the end-diastolic volume index for the identification of pathological hypertrophy. Tissue-specific information is invaluable in HCM: LGE shows a midmyocardial pattern in the hypertrophic areas or elevated T1/ECV values, suggesting diffuse fibrosis in the affected segments. In a small set of HCM patients, athletes, and controls, elevated T2 mapping and global longitudinal strain values (absolute value) helped to distinguish HCM from the athlete's heart [18]. In the differential diagnosis of hypertrophy among highly trained athletes, it is also worth mentioning that decreased native T1 values as assessed by T1 mapping help to distinguish Fabry disease, a rare storage disease that causes LV hypertrophy [13]. Finally, it is of the utmost importance that while CMR might provide important clues for the differential diagnosis of HCM, the final decision *must only* be made with the clinicians' assessment of symptoms, family history, and 12-lead ECG [3, 19].

For demonstration purposes, we present the case of a young female elite athlete (Fig. 1). On CMR, asymmetric hypertrophy (19 mm) with slight T1 elevation and a small LGE was present. She was diagnosed with a mild phenotype of HCM. After a comprehensive risk assessment as per the ESC Guideline on sports cardiology and exercise in patients with cardiovascular disease, she was cleared to return to high levels of sporting activity with close supervision [1].

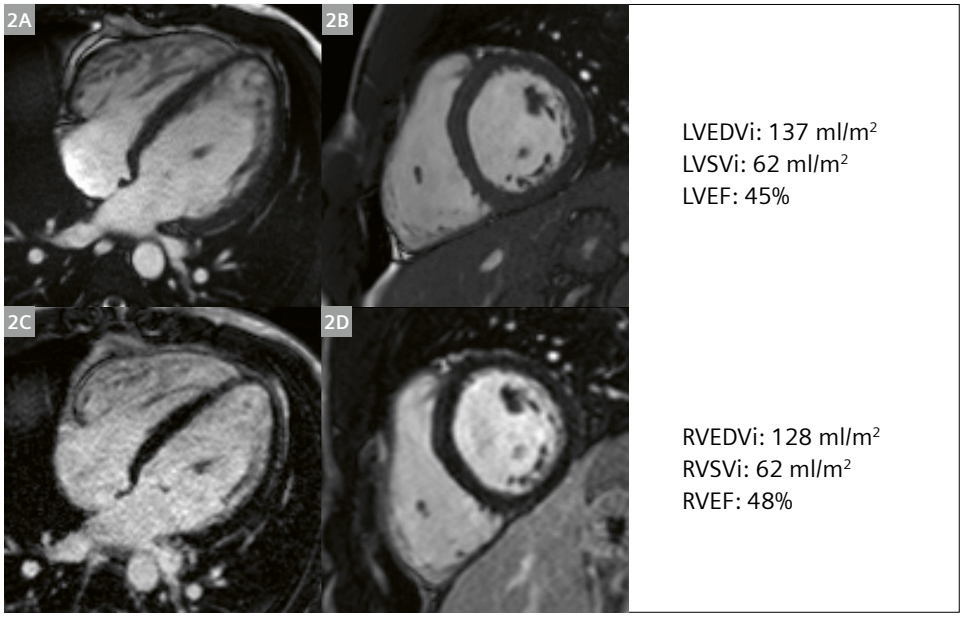
Arrhythmogenic cardiomyopathy

Arrhythmogenic cardiomyopathy (AC) was first described as a fatty-fibrotic replacement predominantly affecting the right ventricle (RV), although LV involvement also occurs [3]. It is often associated with ventricular arrhythmias and SCD – so much so that, according to Italian data, AC accounted for 23% of SCD among young athletes. The diagnosis of AC is quite complex and is currently based on the modified Task Force Criteria that include anamnestic, electrophysiological, and imaging data [20]. Overall, CMR plays a prominent role in the detailed evaluation of RV volumes, function, and regional wall motion abnormalities compared to echocardiography. As the dimensional criteria for AC were described based on the comparison between sedentary controls and AC patients, the European Association of Cardiovascular Imaging (EACVI) suggested applying only "major" volumetric criteria to elite athletes [3]. Nevertheless, elevated volumes (men: RVEDVi > 110 ml/m^2; women: > 100 ml/m^2) only fulfil AC criteria in combination with decreased systolic function and regional akinesia, dyskinesia, or aneurysmal deformation of the RV. While LGE is not currently part of the Task Force Criteria, it can be present in up to 40% of cases [3, 20]. Regional RV feature-tracking strain may also help identify AC in athletes with preserved RV ejection fraction [21].

Dilated cardiomyopathy

Endurance and mixed sports are often associated with biventricular dilatation at rest. They can sometimes be associated with mildly reduced systolic function, which raises the suspicion of dilated cardiomyopathy (DCM) [3]. This presents an important diagnostic challenge and requires a series of examinations, ideally including CMR. DCM also provides some vital imaging clues that can help with the final decision. In athletic adaptation, biventricular cavity enlargement is usually associated with normal systolic function and increased wall thickness [9]. In mild functional impairment (LVEF approximately 50–45%), it might be beneficial to assess the improvement during exercise. Stress CMR is a valuable tool for detecting reduced cardiac functional reserve and early pathological alterations that are not present at rest. This might be beneficial to recognizing DCM in the early stages of the disease [22]. The presence and pattern of LGE are paramount for the differential diagnosis and risk stratification of DCM, though the absence of LGE does not exclude the disease. Among other forms of LV scar, septal midmyocardial fibrosis was linked to ventricular arrhythmias [3, 23, 24]. While there is currently little data on the subject, T1 mapping could potentially play a role, as the physiological hypertrophy in athletes causes a slight decrease in native T1 values, and diffuse fibrosis that is not visible on LGE images can elevate T1 and ECV values [25].

Figure 2 shows the CMR images of an asymptomatic young male kayaker who was referred for CMR due to elevated volumes on echocardiography. CMR found elevated LV volumes exceeding the 95th percentile of normal athletic values, and an LVEF lower than the normal athletic value with mild LV hypokinesis at rest. We did not find signs of regional or diffuse LV fibrosis. Examinations and risk stratification are currently underway for the athlete. His imaging will include stress CMR using an MR-conditional bicycle to establish his reaction to physiological exercise.



2 Young male kayaker with early signs of dilated cardiomyopathy. Cine images in 4-chamber **(2A)** and short-axis **(2B)** view show dilated left (LV) and right ventricles with slightly elevated LV trabeculation. Images **(2C, D)** show no pathological late gadolinium enhancement in the myocardium.

Left ventricular non-compaction

LV non-compaction is characterized by a distinctive double-layer appearance: compact myocardial wall and pronounced myocardial trabeculation with or without deep inter-trabecular recesses. Currently, the most widely used CMR criteria for diagnosis is a non-compacted-to-compacted layer ratio of > 2.3 in diastole [26]. A debate is still ongoing as to whether or not this morphologically and clinically heterogeneous group of individuals should be classified as pathological. High preload conditions such as pregnancy or athletic training are commonly associated with increased LV trabeculation. While LV trabeculation was found in 18–19% of asymptomatic athletic and adolescent populations [27, 28], cardiac pathology should be considered in cases of cardiac symptoms, decreased systolic function, or a family history of heart failure or SCD [1]. Novel proof-of-concept studies show abnormal strain patterns in LV non-compaction [29], and the presence of LGE suggests "cardiomyopathic process", though the overall prevalence of LGE is currently unclear [3].

Myocarditis

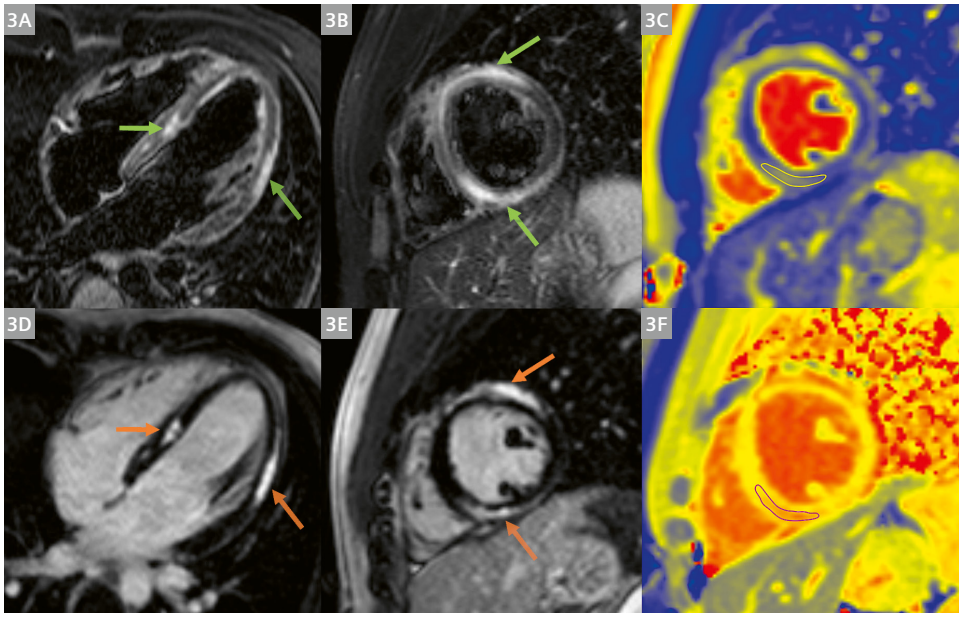
Acute myocarditis causing electrical instability of the heart is also linked to cases of sudden cardiac death in young athletes [3, 30]. A recent history of viral infection and indicative symptoms of chest pain, palpitation, and fever are key factors in the workup of acute myocarditis – though with young athletes, toxins should also be considered and excluded. From an imaging perspective, CMR is very well suited to confirm or exclude the diagnosis of myocarditis [31, 32]. The updated Lake Louise Criteria from 2018 require the following main findings at a regional or global level: myocardial edema on T2 mapping or T2-weighted images and non-ischemic myocardial injury presenting as an abnormal T1 mapping value, elevation of the extracellular volume, or LGE. The supportive features are as follows: pericarditis presenting as effusion in cine images; abnormal LGE, T2, or T1 mapping; and systolic LV dysfunction presenting with regional or global wall motion abnormality [31].

At the beginning of the COVID-19 pandemic, the first results among athletes suggested that myocarditis occurred at an alarmingly high rate, even without symptoms [33]. However, more than a year into the pandemic, we found that the prevalence of definitive acute myocarditis is as low as 1–2% [34–36].

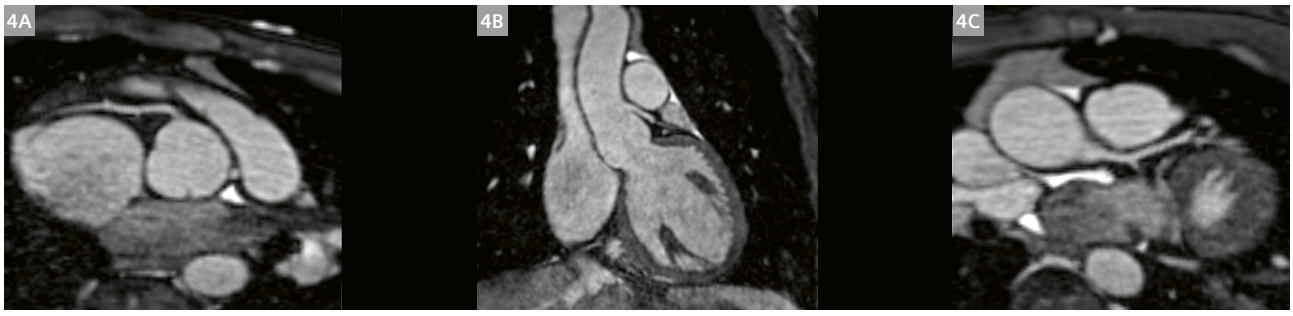
We present the case of a young male floorball player who presented at the emergency room after a short viral infection with considerable chest pain and ECG alteration. Coronary angiography showed no obstruction in the coronary arteries. Therefore, the patient was referred to CMR with the suspicion of myocarditis. CMR confirmed the diagnosis, showing an extensive non-ischemic pattern of myocardial edema and necrosis in the basal and midventricular septal segments and in the apical lateral segment (Fig. 3). The athlete was prohibited from participating in competitive sport for at least three months. After that, he will return to our clinic for a reevaluation [1].

Congenital coronary artery anomalies

While congenital coronary artery anomalies are rare and only affect approximately 0.4% of the adolescent population [28], they are present is as many as 19% of all cases of SCD in young athletes [11]. Two forms are



3 Young male floorball player presenting with acute myocarditis. Edema images using fat-suppressed T2-weighted TIRM sequences in 4-chamber **(3A)** and short-axis **(3B)** views show elevated signal intensity in midmyo/subepicardial pattern (green arrows). On image **(3C)**, T2 mapping values are elevated (56 ms) in the areas showing signal intensity elevation on T2-weighted images compared to our normal values. Late gadolinium enhancement in the same non-ischemic pattern is visible on images **(3D, E)** (orange arrows). Image **(3F)** shows native T1 mapping elevation (1151 ms).



4 Adolescent male basketball player's coronary origin. Images (4A–C) were acquired using a prototype 3D T2-prep non-contrast MR angiography[1] with isotropic spatial resolution [1.2 x 1.2 x 1.2 mm] in free breathing [38]. Image **(4A)** shows the origin of the right coronary artery from the right sinus, while **(4B, C)** depict the origin of the left main coronary artery from slightly above the left sinus.

associated with the most pronounced risk of SCD: an anomalous vessel coursing between the aorta and the pulmonary artery, and an anomalous vessel with an interseptal course that requires surgical repair [1]. In patients with suspected congenital coronary artery anomalies, CT or CMR angiography is recommended. CMR angiography is a non-invasive, radiation-free method of visualizing the coronary origins [37, 38].

Figure 4 shows the non-contrast CMR angiography[1] of a young basketball player who experienced fatigue and presented with ECG abnormalities on exertion. The CMR angiography revealed no coronary artery anomaly, but the left main coronary originates slightly higher up than the usual spot in the left coronary sinus.

Conclusions

CMR plays an essential role both in the assessment and risk stratification of cardiovascular diseases in highly trained athletes. The overlapping phenotypic features of the athlete's heart and mild or early characteristics of cardiomyopathies still constitute a considerable challenge, but these difficulties can be overcome by applying novel CMR techniques such as mapping, strain, or stress imaging.

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[1] *Work in progress. The application is currently under development and is not for sale in the U.S. and in other countries. Its future availability cannot be ensured.*

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