ESC European Heart Journal - Cardiovascular Imaging (2026) **00**, 1–29
https://doi.org/10.1093/ehjci/jeag060

EACVI DOCUMENT

Indications, protocols, and interpretation of cardiovascular imaging for the evaluation and management of athletes. A Clinical Consensus Statement of the European Association of Preventive Cardiology (EAPC) and the European Association of Cardiovascular Imaging (EACVI) of the European Society of Cardiology (ESC). Part 2—Cardiovascular Magnetic Resonance, Cardiac CT and Nuclear Imaging

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Received 10 December 2025; revised 15 February 2026; accepted 16 February 2026; online publish-ahead-of-print 23 June 2026

The number of individuals engaging in sports continues to rise, and identifying those with cardiac substrates associated with increased risk of exercise-related adverse events is crucial. Athlete evaluation requires a refined diagnostic strategy to distinguish physiological cardiac remodelling from pathology. This joint European Association of Preventive Cardiology/European Association of Cardiovascular Imaging consensus provides a multimodality approach for advanced cardiovascular imaging in sports cardiology. Cardiovascular magnetic resonance, cardiac computed tomography, and nuclear imaging each offer complementary insights into cardiac structure, function, coronary anatomy, tissue characterization, perfusion, and inflammation. When integrated with clinical data and first-line tests, they improve diagnostic precision and risk stratification in scenarios frequently encountered in athletes, including ventricular arrhythmias, cardiomyopathies, congenital coronary anomalies, inflammatory myocardial disease, and coronary artery disease. Standardized protocols tailored to age, training, and clinical indication are essential to ensure reliability and avoid misinterpreting physiological adaptation as disease. The consensus emphasizes responsible reporting, considering performance and legal implications of diagnoses, and recommends second-line imaging when justified. Functional imaging, for ischaemia or inflammation, is central in guiding return-to-play decisions. Persistent evidence gaps include limited normative datasets across athletic subgroups and uncertain significance of subtle tissue abnormalities. Overall, this consensus supports harmonized, safe, and judicious multimodality imaging to protect athletes while preventing unnecessary sport restriction.

Keywords sport cardiology • cardiovascular magnetic resonance (RMC) • cardiac computed tomography (CCT) • nuclear imaging • athletes' heart

Introduction

Background and aims

Participation in sports continues to increase, highlighting the need to identify athletes with cardiac substrates that increase the risk of exercise-related adverse events. Accurate evaluation requires a refined diagnostic approach to distinguish physiological cardiac remodelling from underlying pathology. Cardiovascular magnetic resonance (CMR), cardiac computed tomography (CCT), and nuclear imaging provide complementary information on cardiac structure, function, coronary anatomy, tissue characterization, perfusion, and inflammation. When integrated with clinical assessment and first-line testing, these techniques improve diagnostic accuracy and risk stratification in common athletic scenarios, including ventricular arrhythmias (VAs), cardiomyopathies, congenital coronary anomalies (CCA), inflammatory myocardial disease, and coronary artery disease (CAD) in master athletes (MA).

Although recommendations for cardiovascular imaging in athletes have previously been published,[1] primarily emphasizing echocardiography, specific advice on the indications, protocols, and interpretation of CMR, CT and nuclear imaging in this population are missing and timely.

Accordingly, this clinical consensus statement was developed through collaboration between experts from European Association of Preventive Cardiology (EAPC) and European Association of Cardiovascular Imaging (EACVI) both constituent association of the European Association of Cardiology (ESC), focusing on the role of three imaging modalities for the diagnosis and prognostic characterizations of relevant cardiac conditions for both competitive or amateurs' athletes.

The objectives are:

(1) to describe the appropriate clinical indication for CMR, cardiac CT, and nuclear images in athletes;
(2) to advice how to perform CMR, cardiac CT, and nuclear imaging in athletes, focusing on major challenge encountered in clinical practice.
(3) To describe how to interpret cardiac CMR, CT, and nuclear imaging in athletes;

This document is part of a series of two manuscripts, which will address several modalities within cardiovascular imaging in athletes. For this reason, the introduction on cardiovascular adaptations to exercise and training and definition of athlete is included in the first document[2] and are not duplicated here.

CMR in sport cardiology

The ability of CMR to evaluate morphology and characterize myocardial tissue composition sets it apart from other imaging modalities complementing information obtained through echocardiography. Abnormalities in tissue characterization warrant restrictions on competitive sport in some cases.

However, CMR in athletes can present with certain challenges. Technical issues may arise during scanning, and accurate data interpretation requires a detailed understanding of exercise-induced cardiac remodelling. In addition, the clinical significance of subtle morphological abnormalities of myocardial scarring also requires careful evaluation.

Beyond these technical considerations, CMR reports for elite and professional athletes can have serious consequences,

including the potential impact on their career, as well as potential implications for physicians' insurance coverage, and legal matters. For this reason, it is crucial to uphold a strong sense of responsibility in all aspects of the process: from requesting investigations when clinically indicated, to ensuring their execution meets the highest standards and interpreting findings with due diligence, particularly when dealing with potentially subclinical or equivocal findings.

Cardiac CT and nuclear imaging in sport cardiology

Approximately 60% of cardiac diseases associated with sudden cardiac death (SCD) in young athletes have a structural basis and over 80% of fatalities in older (i.e. MA >35-years-old) athletes are linked to atherosclerotic coronary artery disease.[3] These statistics underscore the need for comprehensive screenings and assessments to promptly detect these potential pathologies and implement tailored risk stratification as basis for advice to ensure the safety of athletes during physical exertion. Pragmatic screening strategies, such as 12-lead electrocardiography (ECG), are effective for identifying various electrical diseases and raising suspicion about an underlying cardiomyopathy. A recent study of Malhotra et al. showed that cardiac diseases associated with SCD were identified in 0.38% of adolescent soccer players in a cohort that underwent cardiovascular screening.[4] However, they are inadequate for detecting CAD and aortopathy.

Coronary computed tomography angiography (CCTA) is the most sensitive method for detecting CAA and atherosclerotic CAD.[5,6] Despite concerns regarding radiation exposure, modern scanners equipped with prospective ECG gating enable image acquisition in a single heartbeat, significantly reducing radiation exposure to less than 1 millisievert (mSv). Besides visualizing coronary anatomy in young athletes, CCTA has emerged as a powerful diagnostic and prognostic tool for assessing the presence and severity of any atherosclerotic CAD, providing information beyond the coronary artery calcium score (CACS). With the addition of intravenous contrast, CCTA permits qualitative, semi-quantitative and quantitative plaque analysis and is effective for detecting high-risk plaque markers.[7,8] This technology has revolutionized the diagnosis and management of CAD in MA, providing detailed and accurate morphologic insights that can impact their care and outcomes, especially when combined with functional evaluation.

Moreover, CT exhibits superior spatial resolution than echocardiography in assessing athletes with aortopathy. It may also help investigate claustrophobic athletes and athletes with implantable devices in whom CMR imaging may be contraindicated or associated with undesirable artefacts.

Nuclear imaging uses various radioactive tracers to visualize and evaluate organ and tissue functionality.[9] Within the domain of sports cardiology, nuclear imaging assumes a role in the assessment of individuals with known CAD, complementing functional information to the anatomical data. Additionally, nuclear imaging is helpful in detecting inflammatory conditions such as myocarditis and sarcoidosis, although its application in sport cardiology is uncommon.

Clinical indication

CMR

Given its strengths on morpho-functional evaluation and tissue characterization, the indications for CMR have recently expanded,[10] necessitating an update of its appropriateness and effectiveness in the field of sports cardiology. From a practical clinical perspective, several major conditions represent the most common reasons for CMR referral in athletes:

(1) Abnormal ECG findings (e.g. infero-lateral inverted T-waves)
(2) VAs with morphologies other than fascicular or outflow tract origins,[11] polymorphic, repetitive or exercise-induced
(3) A sub-optimal acoustic window on echocardiography, preventing complete visualization of the left ventricle (LV) apex, lateral free wall or right ventricle (RV)
(4) Borderline echocardiographic findings, such as suspected pathological LV hypertrophy (LVH) or mildly depressed LV function.

In a large cohort of well-trained recreational and elite athletes (n = 503, 82% male, mean age 33 ± 15 years), CMR findings were positive in (i) 23% of cases referred due to abnormalities on history and physical examination; (ii) 34% of cases with abnormal T-wave patterns; (iii) 37% of cases with VAs with morphologies other than fascicular or outflow tract origins; and (iv) 58% of cases with borderline echocardiographic findings.[12] VAs were the most common indication for CMR, which revealed late gadolinium enhancement (LGE) in 54% of cases, usually associated with VAs morphologies other than fascicular or outflow-tract origins, particularly repetitive and polymorphic patterns. Other CMR-detected abnormalities included hypertrophic cardiomyopathy (24%) and dilated cardiomyopathy (14%). Although these data originate from a single-centre study, they highlight the importance of carefully selecting the clinical indication to CMR minimize the number of normal testing.

CCT and nuclear imaging

Clinical abnormalities

There are three main reasons why cardiovascular CT imaging may be appropriate in an athlete:

(1) Risk stratification of CAD
(2) Evaluation of symptoms suggestive of myocardial ischaemia
(3) Evaluation of suspected CCA.

CACS evaluation utilizes low-dose CT without the need for contrast and might be appropriate in middle-age or older athlete in whom a positive result may influence the choice of preventive therapies[13] such as statin therapy. It is noteworthy that although an athletic lifestyle is associated with a better cardiovascular (CV) risk profile and possible lower incidence of CV events and improved longevity,[14,15] several studies have consistently showed increased calcium in endurance athletes.[16–19] Thus, there is something of a mismatch between the higher burden of CAD and a lower risk of coronary adverse events that need to be considered.

CCTA requires administration of iodine-based contrast and ECG gating with the quality of imaging being dependent on low heart rates. The main indications for CCTA are symptoms suggestive of myocardial ischaemia. Typical symptoms include exertional chest, arm, or jaw tightness although other symptoms (anginal equivalents) include breathlessness or fatigue with exercise that is new and increases with exercise intensity. Ischaemic symptoms generally resolve with a few minutes of exercise cessation. Sometimes athletes will provide a good description of ischaemic preconditioning in which an athlete may experience chest pain early during exercise but then, after relative rest, can return to similar or even more intense exercise without symptoms. CCTA is an accurate, non-invasive imaging technique that characterizes coronary anatomy, atherosclerotic

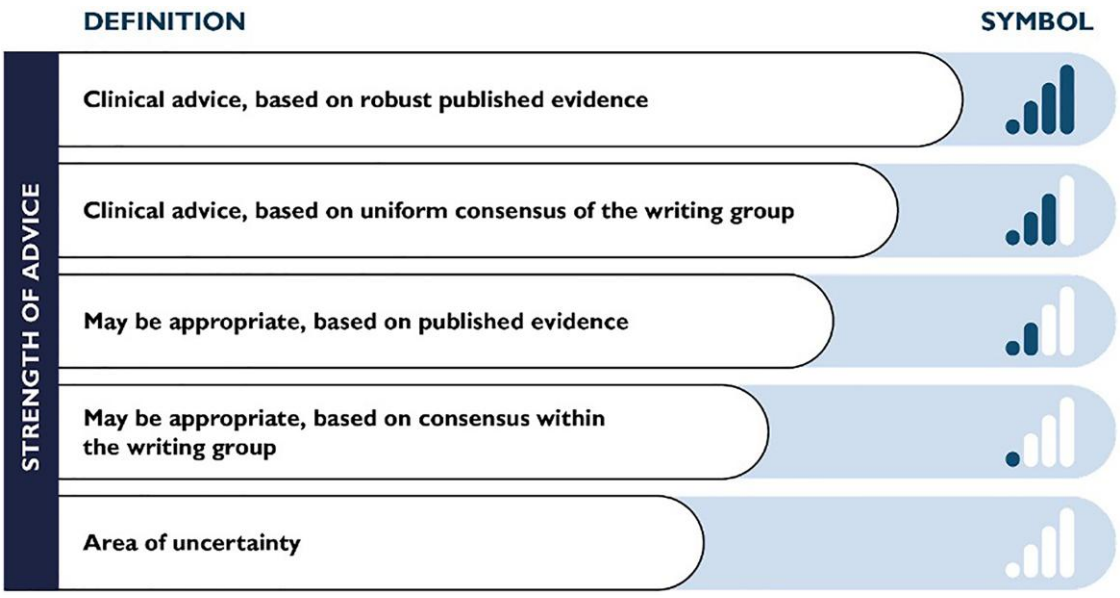

Figure 1 Strength of advice for clinical management considerations.

plaque burden, and lumen stenoses enabling their identification and quantitative assessment, thereby supporting accurate diagnosis and management, including antianginal therapy or revascularization.

Sudden onset of breathlessness with or without pleuritic chest pain should raise suspicion of a pulmonary embolus, particularly if accompanied by risk factors such as recent travel, an elevated D-dimer test, or a known deep venous thrombosis. CT pulmonary angiography is the test of choice for assessing whether a defect compromises the pulmonary vasculature filing.

Nuclear imaging is generally not indicated in asymptomatic athletes. Stress nuclear imaging and other stress imaging tests, such as stress echocardiography or stress CMR, are commonly used in athletes with low probability of obstructive CAD, if clinically indicated, where they have comparable diagnostic accuracy. After CCTA, nuclear imaging (or stress CMR/echocardiography) may be required to complement anatomical information by assessing the functional significance of coronary lesions to guide management.

ECG abnormalities and ventricular arrhythmias

ECG evaluation in athletes is primarily intended to identify subclinical cardiomyopathies or channelopathies. Echocardiography is generally the first-line evaluation when cardiomyopathy is suspected. In contrast, the resting 12-lead ECG is typically normal in the athlete with symptoms suggestive of exertional angina.[20] ECG findings such as Q-waves or T-wave abnormalities in the athlete with CAD resulting in a previous myocardial infarction are found in a minority of athletes and have relatively poor sensitivity for coronary vessel disease.[21] Thus, ECG abnormalities are an uncommon reason to refer an athlete for CCTA or nuclear imaging. However, an exception may be represented by the detection of VAs in the 12-lead ECG. In this case, the morphology of the ectopic beats and the age of the athletes will guide the indication for additional imaging testing, specifically to CCTA, as specified below.[22] The same indication originates from the occurrence of repolarization abnormalities (i.e. ST segment depression with negative T-wave) during exercise, potentially compatible with the presence of a myocardial bridge or coronary artery lesions in MA.

General principles

CMR

Protocols and reporting

CMR protocols for athletes are largely similar to disease-specific protocols for non-athletes,[10] but may require adjustments based on the clinical indication (*Figures 1* and *2*, *Table 1*). The presence of shunts should be carefully evaluated, particularly if disproportionate dilatation is present. Dedicated sequences can be acquired to detect and evaluate the severity of valve disease. Additionally, evaluating the potential for CCA is critical. In every scan, especially in young athletes and those with chest pain or exertional syncope, imaging sequences should be incorporated to visualize coronary artery origins. This can be achieved using 3D whole heart coronary magnetic resonance angiography. Additional sequences may be appropriate when cardiomyopathy or myocarditis are suspected. In MA, the focus shifts towards detecting acquired cardiovascular diseases, such as myocardial scarring and ischaemia. Athletes may present unique challenges during scans, particularly due to sinus bradycardia, which can complicate image acquisition. Given the potential clinical impact of LGE, post-contrast imaging should be of the highest quality, minimizing artefacts. *Table 2* outlines common technical challenges encountered during CMR in athletes, and potential solutions.

A CMR report for an athlete, like any other patient, should primarily address the clinical question that prompted the scan. While certain measurements and descriptions should be included in virtually all reports, the interpretation and conclusions must focus on the clinical indication and the pertinent findings relevant to answering that specific query (*Table 1*). CMR reporting is generally similar for athletes and non-athletes, except for

Figure 2 Indication for CMR scanning and reporting in athletes. CMR, cardiovascular Magnetic resonance; LGE, late gadolinium enhancement; LV, Left ventricle; Vas, ventricular arrhythmias.

the reference range limits. Ideally, the reporting physician should consider the athlete's ethnicity, sport type and level of conditioning to provide context and ensure accurate interpretation of the images and data. The best-evidenced technique for assessing the 'expected' cardiac remodelling in athletes is recognizing the strong correlation between VO2peak and ventricular volumes.[25,26] However, because cardiopulmonary exercise testing is not routinely performed, it is essential to obtain detailed information about the athlete's training history. Factors such as sports discipline and exercise intensity play a crucial role in determining the expected degree of cardiac remodelling and addressing specific clinical questions.[27] Therefore, both the clinical indication and the athlete's training history should be clearly stated at the beginning of the report.

The presence and distribution of LGE must be carefully evaluated, ensuring that all potential pitfalls (e.g. trabeculae, cleft,[28] anterior septal perforator arteries[29,30]) are excluded. If mapping sequences are available and included in the scan, their results should be incorporated into the report, following established guidelines.[31]

Table 1 Sequences, measures, and interpretation of cardiac structure while scanning and reporting in athletes

Structure	Sequence	Measures	Interpretation
Left ventricle			
Volumes, mass and wall thickness	SSFP Cine (short-axis stack and 3 long-axis views)	LV volumes, SV and mass should be presented with and without indexing to BSA. Wall thickness should be measured in multiple segments from a short-axis end-diastolic frame	• Although the report should describe structure relative to non-athletic norms, subsequent interpretation should place the results in relation to the degree of athletic conditioning. • Athletic remodelling (endurance and mixed disciplines) can lead to balanced chamber dilatation (volume ratio between LV/RV = 1), eccentric LVH, and a low-normal ejection fraction with high/normal SV • Note that LV mass is largely determined by the overall ventricular size and wall thickness, and it is frequently increased in athletes • In cases of extreme remodelling, other features should be consistent: look for sinus bradycardia, dilated IVC, and muscle composition in the localizers
Function	SSFP cine	Endocardial tracing to determine volumes, ejection fraction and SV	• Qualitative assessment of regional and global function with reference to the ejection fraction.
Oedema	T2-weighted and T2 mapping	Qualitative assessment of focal changes and region of interest quantification	• Focal increases in signal or increases in T2 mapping times are suggestive of oedema.
Fibrosis	LGE imaging, T1 mapping (native and post contrast with ECV calculation)	Qualitative assessment of focal scar and quantification of diffuse fibrosis with mapping	• Awareness of low-risk or benign patterns (e.g. inferior hinge-point) prevalent in athletes and sedentary controls. • Mid-myocardial and sub-epicardial patterns have been associated with arrhythmias in athletes. • Native T1 Mapping values and ECV should be similar to (or slightly lower than) those in a non-athletic population
Right ventricle			
Volumes, mass and wall thickness	SSFP cine (short-axis stack, long-axis and focused outflow tract views)	RV volumes and SV should be presented with and without indexing to BSA.	• Although the report should describe structure relative to non-athletic norms subsequent interpretation should place the results relative to the degree of athletic conditioning
Function	SSFP cine Axial stack can be acquired for RWMA of the RV free wall.	Endocardial tracing to determine volumes, ejection fraction and SV	• Qualitative of regional and global function with reference to the ejection fraction.
Oedema and fat suppression	Inversion recovery	May enable assessment of fat infiltration	• The accuracy and importance of fat suppression for RV has been questioned.
Fibrosis	LGE imaging	Qualitative assessment of post-gadolinium enhancement	• This can be a difficult qualitative assessment and can only be stated confidently when there is adequate wall thickness
Atria			
Size	Cine long-axis	Volume derived from 4 and 2-Chamber	• Generally less accurate than echo unless carefully planned. Note the susceptibility to foreshortening
Vascular			
Coronary ostia	Navigated cine +/− 3D	Ostia location	• Anomalous coronaries should be assessed in young athletes, in case of symptoms during effort, abnormal exercise test or evidence of endocardial injury.

Continued

Table 1 Continued

Structure	Sequence	Measures	Interpretation
Aortic root	Cine +/− 3D angiography	Carefully planned short-axis or angio sequences can be used to accurately assess aortic diameter	• Slightly larger vascular dimensions may be expected but prognostic cut-offs unchanged.
Valves			
Structure and function	Cine +/− velocity-encoding	Qualitative assessment in most instances looking for flow acceleration	• Similar evaluation as non-athletes
Other			
Pericardium	T1, T2 and post-contrast	Assess for effusions, oedema, LGE	

BSA, body surface area; ECV, extracellular volume; IVC, inferior vena cava; LGE, late gadolinium enhancement; LV, left ventricle; LVH, left ventricle hypertrophy; RV, right ventricle; RWMA, regional wall motion abnormalities; SV, Stroke volume.

Table 2 Common issues encountered when scanning athletes by CMR and possible suggested solutions to solve them

	Issue	Possible solutions
During scanning		
Body composition	Low body fat is related to higher volume of distribution for gadolinium-based contrast agents. This may mean faster apparent washout	• Image late gadolinium enhancement earlier than usual can be an option or additional LGE may be required in cases of fast wash-out rates
Bradycardia	Conventional cines designed around HR 70 bpm may take a very long acquisition time	• Increase segmentation if the athlete has difficulty maintaining the apnoea
Late gadolinium enhancement	More complete longitudinal recovery (bradycardia), faster gadolinium washout	• Longer T1 times may be needed • Alternatively switch to trigger 1 (where may be normal or even shorter) • Consider increasing segmentation
Coronary origins	Bradycardia may make navigated imaging very prolonged	• Prolonged diastole and large coronaries may make cines for coronary origins easy: a stack of short axis aortic valve views just above the valve, a coronal LVOT for left main stem and the basal SA stack for right coronary origin. • Check for an anomalous circumflex on the 4chamber cine ('RAC sign'[23] and 'crossed aorta'[24])
T1 and ECV mapping	Bradycardia may confound; tissue characterization alterations from athleticism	• Sequences with robust heart rate independence should be used (e.g. 5 s(3 s)3 s MOLLI rather than heart beat based sampling schemes.
Oedema	Bradycardia may length significantly the T-weighted sequences	• In case of extreme bradycardia resulting in a prolonged apnoea that the athlete is unable to sustain, T2 Mapping can be more advantageous

ECV, extracellular volume; HR, heart rate; LVOT, left ventricle outflow tract; RAC, retroaortic anomalous coronary artery.

CMR normal reference range for chamber size and function and tissue characterization in athletes

Defining normal reference ranges for chamber size and function is essential for evaluating both healthy individuals and athletes, considering impact of exercise-induced adaptation can be considered. Several factors influence this adaptation, including age, sex, ethnicity, sport type, training volume and intensity. Accurate interpretation is crucial to differentiate physiological remodelling from pathological changes that may indicate an inherited or acquired cardiac condition. Endurance training is generally associated with eccentric hypertrophy, whereas strength training leads to concentric hypertrophy. Although this framework is widely recognized, not all athletes exhibit these distinct patterns of cardiac remodelling. Recent studies of large cohorts of elite athletes across various sports disciplines show considerable variability in LV geometry, with normal LV geometry being common and eccentric hypertrophy present in some athletes.[32,33] Data on sedentary subjects cannot be used

Table 3 Selected studies on CMR reference values in athletes

Study	Study type	*N* of athletes	Age range (years)	Ethnicity	Male sex	Sport type	Training regimen	Years of training
D'Ascenzi et al 2018[35]	meta-analysis (27 studies)	983	18–55	Caucasian	100% males	Endurance, mixed, combined	>6 h/week	—
Csecs et al. 2019[36]	observational prospective study	327 (215 adults; 112 adolescents)	adults >18; adolescents 14–18	Caucasian	67% male adults; 87% male adolescents	skill, power, mixed, endurance	22.5 ± 4 h/w in male adults 20 ± 6 in female athletes; 13 ± 5 in male 16 ± 6 in female adolescent athletes.	—
Maceira et al. 2023[37]	observational prospective study	148	18–50	Caucasian	64.8% males	Low, medium, high intensity	16.4 ± 7.9 h/week	10.6 ± 5.3
De Bosscher et al, 2023[38]	Observational prospective study	201	17–20	Caucasian	79% males	Endurance: 58% cycling, 23% running, 11% triathlon, 5% rowing	>3 h/week and competing at national and international level	—

The table includes a meta-analysis, which collected data from 27 studies, and three other studies published after the meta-analysis.
h/w, hours per week.

to interpret athletes data.[34,35] Among athletes, available studies are heterogeneous in terms of population, training regimes and sport type. The only meta-analysis dedicated to athletes included 983 male Caucasian athletes and provided mean values, 1st and 95th percentile for LV and RV volumes and function in athletes practicing endurance, mixed or combined sports (*Tables 3* and *4*).[35] However, this study excluded several important subgroups, specifically adolescent athletes, female athletes, non-Caucasian athletes or athletes engaging in purely isometric sports due to the paucity of published data.[39] More recently, additional studies have provided CMR reference values in both adolescent[36] and female athletes.[36–38,40] *Tables 3* and *4* present the most recent reference values from the wider cohort currently available. However, non-Caucasian athletes remain underrepresented, which may limit the interpretation of their measurements. It is important to note that, while these documents aim to provide reference values, they remain somewhat imprecise because they lack sport-specific data that account for the intensity and volume of habitual training regimens. Similarly, balanced atrial dilatation is recognized as part of physiological cardiac remodelling in athletes, yet CMR-derived reference ranges for atrial dimensions remain limited to small cohorts.[41] Further multicentre studies are timely and necessary to address these limitations.

Emerging data on tissue characterization, including minor insertion-point fibrosis and extracellular volume (ECV) assessment, highlight the need for larger, more diverse studies to establish robust reference values. Currently, LGE is considered an abnormal finding in athletes except at the inferior RV insertion point (hinge point) where it is commonly observed. (*Figure 3*). Mapping techniques provide additional insights into tissue characterization. Athletes with the most significant LV remodelling, primarily those in endurance and mixed, exhibited the lowest T1 mapping values compared to sedentary controls and other sports.[42,43] Additionally, male athletes have lower T1 mapping values than female athletes.[42] However, no significant difference in T2 mapping was observed across sporting disciplines and between genders.[42,43]

CCT

Protocols and reporting

In the last decade, impressive technological developments in both scanners and post-processing tools have improved the imaging quality of CCTA, even under challenging situations, such as high heart rate or heart rate variability, or high body mass index.[44] Several multicentre trials and registries, such as the large PROTECTION VI, have shown a significant reduction in average radiation exposure from CCTA.[45] In sport cardiology, it is crucial to obtain artefact-free images, especially for case of CCA in young competitive athletes and atherosclerotic plaque assessment in MA. For these reasons, the use of a recent scanner generation, equipped with wide Z-axis coverage and fast gantry rotation time is suggested and nowadays offered by the four vendors (*Table 5*). Special attention to radiation exposure must be reserved for young athletes. Approaches such as prospective ECG-gating, low tube voltage, and iterative reconstruction, which are available on the most of scanners in clinical use, are highly suggested. Wide coverage detector, fast gantry rotation and low-concentration contrast agents may be considered to reduce Iodine load to the young athletes.[46]

As for CMR, CCTA reporting is generally similar for athletes and non-athletes. The components of the report include indication for procedure, patient clinical data, technical procedure information, image quality, clinical scan findings, and interpretation. In particular, the specific reason for ordering the test

Table 4 Basic CMR LV and RV indexed parameters in different athletic populations

Study	LVEDVi mL/m^2	LVESVi, mL/m^2	LVSVi mL/m^2	LVEF, %	LVMi, g/m^2	RVEDVi, mL/m^2	RVESVi, mL/m^2	RVEF, %
D'Ascenzi, male endurance athletes[a]	111 ± 4	49 ± 2	63 ± 6	59 ± 1	72 ± 2	120 ± 3	55 ± 2	54 ± 1
	95% CI	95% CI	95% CI	95% CI	95% CI	95% CI	95% CI	95% CI
	104–121	45–55	45–79	58–61	68–76	113–126	49–61	52–56
Csecs et al, male adult	122 ± 15	53 ± 10	69 ± 8	54 ± 4	86 ± 16	127 ± 17	57 ± 11	55 ± 4
Csecs et al, female adult	106 ± 12	44 ± 7	61 ± 8	58 ± 5	63 ± 10	106 ± 14	44 ± 7	58 ± 5
Csecs et al, male adolescent	112 ± 13	46 ± 8	66 ± 9	59 ± 5	72 ± 11	113 ± 14	48 ± 9	58 ± 5
Csecs et al, female adolescent	103 ± 13	42 ± 8	62 ± 9	60 ± 5	62 ± 10	106 ± 14	42 ± 8	59 ± 4
Maceira, high intensity sport	106 ± 19	40 ± 10	na	63 ± 6	83 ± 16	105 ± 17	41 ± 11	61 ± 7
Maceira, medium intensity sport	103 ± 16	37 ± 9	na	64 ± 6	80 ± 19	104 ± 19	41 ± 10	61 ± 6
Maceira, low intensity sport	79 ± 10	28 ± 7	na	65 ± 7	65 ± 14	81 ± 12	31 ± 9	61 ± 6
Maceira, male endurance athletes	109 ± 17	42 ± 11	na	63 ± 6	91 ± 14	107 ± 17	43 ± 11	61 ± 6
Maceira, female endurance athletes	93 ± 13	37 ± 6	na	63 ± 7	73 ± 11	95 ± 15	38 ± 7	60 ± 5
De Bosscher, endurance athletes	117 ± 18	50 ± 11	na	57 ± 5	79 ± 14	131 ± 23	64 ± 15	52 ± 6

All values are mean ± standard deviation except D'Ascenzi *et al*., which is a meta-analysis therefore values are mean ± standard error and 95% confidence intervals. LVEDVi, indexed left ventricle end-diastolic volume; LVEF, left ventricle ejection fraction; LVESVi, indexed left ventricle end-diastolic volume; LVMassi, indexed left ventricle mass; RVEDVi, indexed right ventricle end-diastolic volume; RVEF, right ventricle ejection fraction; RVESVi, indexed right ventricle end-diastolic volume.
[a]Mean ± Standard Error; 95% CI: confidence interval.

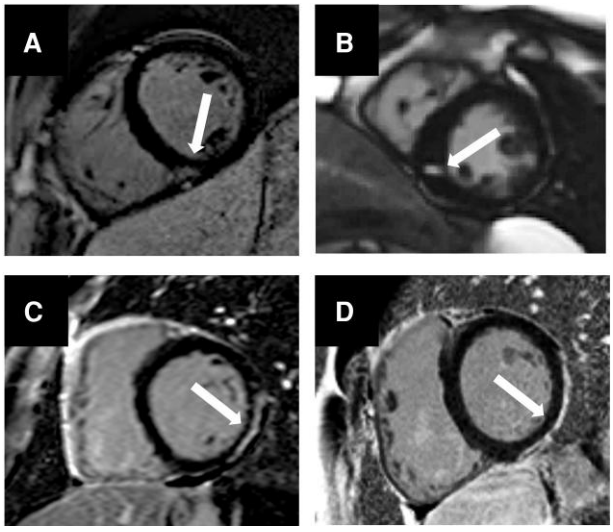

Figure 3 Example of late gadolinium enhancement. *(A)* LGE at the inferior hinge-point is observed in healthy athletes (white arrow). *(B)* Mid-myocardial LGE observed at the inferior RV insertion presenting with focal hypertrophy in an athlete with HCM (white arrow). *C* and *D* show two athletes with pathological non-ischaemic LGE, sub-epicardial, at the lateral wall (white arrows).

should be identified and documented (e.g. symptoms, positive stress test results, risk factors, others). Major aspects of image acquisition should be documented in the report, including the type of study, equipment, technical acquisition protocol type, amount and timing of contrast or other medications, and some measure of the radiation dose. Current studies include CACs, CCTA, the pulmonary veins, the cardiac veins, the left ventricular outflow tract, congenital heart disease (CHD), and other cardiac morphologies, with late Iodine enhancement of detecting myocardial scar. Three broad categories, such as coronary findings, non-coronary cardiac findings, and non-cardiac findings are important to include in the report. Coronary anatomy should be described in terms of left or right dominance and includes potentially relevant variance (e.g. absent left main). If coronary disease is present, stenosis severity, plaque morphology, and extent should be described. Stenosis severity may be described qualitatively (e.g. mild, moderate, severe, or occluded) or preferably with an estimated percentage of diameter obstruction in the following categories: 0) Normal: absence of plaque and no luminal stenosis; 1) Minimal: plaque with <25% stenosis; 2)

Table 5 Major technical parameters of the premium scanners from the different vendors

	Canon	GE Healthcare	Philips	Siemens
Model name	Aquilion One Prism	Revolution Apex Elite	Spectral CT 7500	Somatom Force
Z Detector size	160 mm	160 mm	80 mm	57.6 mm × 2
Cell dimension	0.5 mm	0.625 mm	0.625 mm	0.6 mm
Max Rotation speed	0.275 s	0.23 s	0.27 s	0.25 s
kVp selection	80–135 kVp	70–140 kVp	80–140 kVp	70–150 kVp
mA selection	10–900 mA	10–1300 mA	10–1000 mA	20–1300 mA
Deep learning recon	AiCE, PIQE	TrueFidelity	Precise Image	n/a

Mild: 25% to 49% stenosis; 3) Moderate: 50% to 69% stenosis; 4) Severe: 70% to 99% stenosis; 5) Occluded. Plaque type should be described as calcified, mixed or noncalcified. CACS quantification should be included when available. Other morphologic descriptors of a stenotic lesion, such as extensive length, bifurcation or ostial involvement, location in a tortuous segment, eccentric position, apparent dissection or ulceration may also be appropriate.[47] When CCTA is performed in MA for suspected CAD, a detailed description of plaque features that have been associated with high-risk of developing acute coronary syndrome at follow-up (i.e. positive remodelling, small spotty calcification, low-attenuation plaque, napkin-ring sign) is highly suggested, which may help to define the eligibility of the athlete to competitive sports.[48] When CCTA is performed in young athletes for suspected or known congenital coronary anomaly, a detailed description of the high-risk anatomical features, such as inter-arterial course and intra-mural tract, slit-like ostial morphology, acute takeoff angle, >5 mm length of lumen narrowing, >50% diameter stenosis in the narrowed portion, have to be mandatory included in the reporting[49] (*Figure 4*).

Nuclear imaging

To avoid repetition the section 'protocol' and 'reporting' for nuclear imaging are included in the document on stress imaging.

Differential diagnosis between athlete's heart and cardiomyopathy

CMR for the differential diagnosis of hypertrophic cardiomyopathy

It is known that 2–3% of White athletes and up to 13–18% of Black athletes exhibit increased LV thickness in a range of 13–16 mm, which may overlap with hypertrophic cardiomyopathy (HCM).[50,51] This typically occurs in highly trained endurance male athletes, while in females, the grey zone is defined by a wall thickness between 11 and 13 mm.[52,53] In physiological remodelling, wall thickening is typically symmetric and occurs with combined LV dilatation, resulting in eccentric LVH (*Figure 5A–C*). In contrast, HCM is characterized by regional wall thickening with a non-dilated LV (asymmetric hypertrophy with abnormal LV shape) and abnormal diastolic function in 80% of non-athletic cases[54] and (*Figure 5G–I* and *L-N*). Athletes with HCM often present with a distinct subset of characteristics features compared with sedentary HCM patients. These athletes may have relatively mild wall thickening (15–16 mm), a relatively larger LV cavity, and normal diastolic function.[55] However, most still exhibit asymmetric wall thickening, with one-third showing hypertrophy confined to the LV apex. A small proportion had concentric LVH with wall thickness between 13 and 15 mm. Applying current echocardiographic criteria to competitive athletes with HCM may fail to identify certain cases, especially those with apical HCM, relatively large LV cavities, or normal diastolic function. Many athletes with HCM, however, present with an abnormal ECG, which may be useful to suspect/confirm the diagnosis.

Although echocardiographic algorithms may aid in differentiating physiological LVH from HCM, CMR imaging provides high spatial resolution, enabling more accurate assessment of LV wall and cavity, especially when echocardiographic images are unclear or the hypertrophy is subtle or focal—such as apical, inferior, and lateral wall hypertrophy. In athletes with T-wave inversion, echocardiography diagnosed HCM in only 20% of cases, while CMR significantly improved detection (by identifying other 35% of cases), particularly in those with marked repolarization changes and apparently normal echocardiograms.[56] Therefore, in any case where the suspicion for HCM is high, such as in case of deep infero-lateral T-wave inversion, investigations should include CMR.

Approximately 65% of HCM patients show macroscopic myocardial fibrosis in the hypertrophied area, though the absence of LGE does not definitively rule out the disease, especially in adolescent and young individuals.[57,58] In HCM, LGE at the RV inferior insertion point is mid-myocardial (*Figure 3B*), whereas, in healthy athletes, fibrosis is typically found at the hinge point (*Figure 3A*). T1 Mapping may provide additional information useful to distinguish between HCM and exercise-related cardiac remodelling.[42,59–61] Physiological LVH is primarily due to myocyte enlargement rather than increased extracellular matrix, resulting to lower native T1[42] and ECV values than in controls and HCM patients.[59] In HCM, pathological LVH correlates with increased ECV, indicating that a significant portion of the LV mass is attributable to extracellular matrix expansion (i.e. fibrosis).[62] However, low ECV values may be detected in certain hypertrophied areas, reflecting a mixture of cellular hypertrophy and fibrotic myocardium (disarray).[63] Although T1 mapping shows promise, the data are limited and require validation in a larger cohort of athletes to establish its clinical utility (*Figure 6*).

CMR for the differential diagnosis of dilated cardiomyopathy

Dilated cardiomyopathy (DCM) is a rare but recognized cause of death in athletes, contributing to approximately 1% of all SCD.[64] It is characterized by LV dilation and impaired systolic function

Figure 4 Indication for CT scanning and reporting in athletes.

unrelated to abnormal loading conditions or coronary artery disease.[58] DCM can also affect the RV in 20–60% of cases.[65] On the other hand, the athlete's heart is also associated with biventricular dilation, and in highly trained endurance athletes, occasionally, with low-normal LV ejection fraction (EF).[66] Moreover, early DCM may also present with normal or mildly reduced LVEF in the range observed in athletes, creating a 'grey-zone' where physiological adaptations and pathology overlap. CMR is particularly helpful in distinguishing these conditions by accurately assessing ventricular volumes, function, and tissue characterization.

An unbalanced LV dilatation, along with low EF and stroke volume (SV) suggests DCM, whereas a low-normal EF with balanced ventricle dilation (LV/RV ratio = 1) and high SV is more indicative of an athletes' physiological remodelling (*Figure 3D–F*). Secondly, a LV end diastolic diameter >60 mm occurred in approximately 15% of elite athletes who participated in vigorous training programmes, (<1%)[67,68] while a diameter >70 mm is uncommon in athletes.[68,69] If accompanied by wall thinning, or a reduced LVEF or SV, particularly in sports less commonly associated with dilation (e.g. strength or skill athletes), this raises suspicion of DCM. If balanced biventricular dilation is associated with mild LVH, a low heart rate and high SV, this would support athletic remodelling. In addition, the presence of mid-wall LGE (particularly septal) is seen in about a third of DCM patients[58] and supports the diagnosis, although its absence does not exclude DCM. Furthermore, athletes can favourably augment cardiac output,[70] and LVEF in response to exercise, whereas DCM patients have a blunted response. Exercise imaging tests

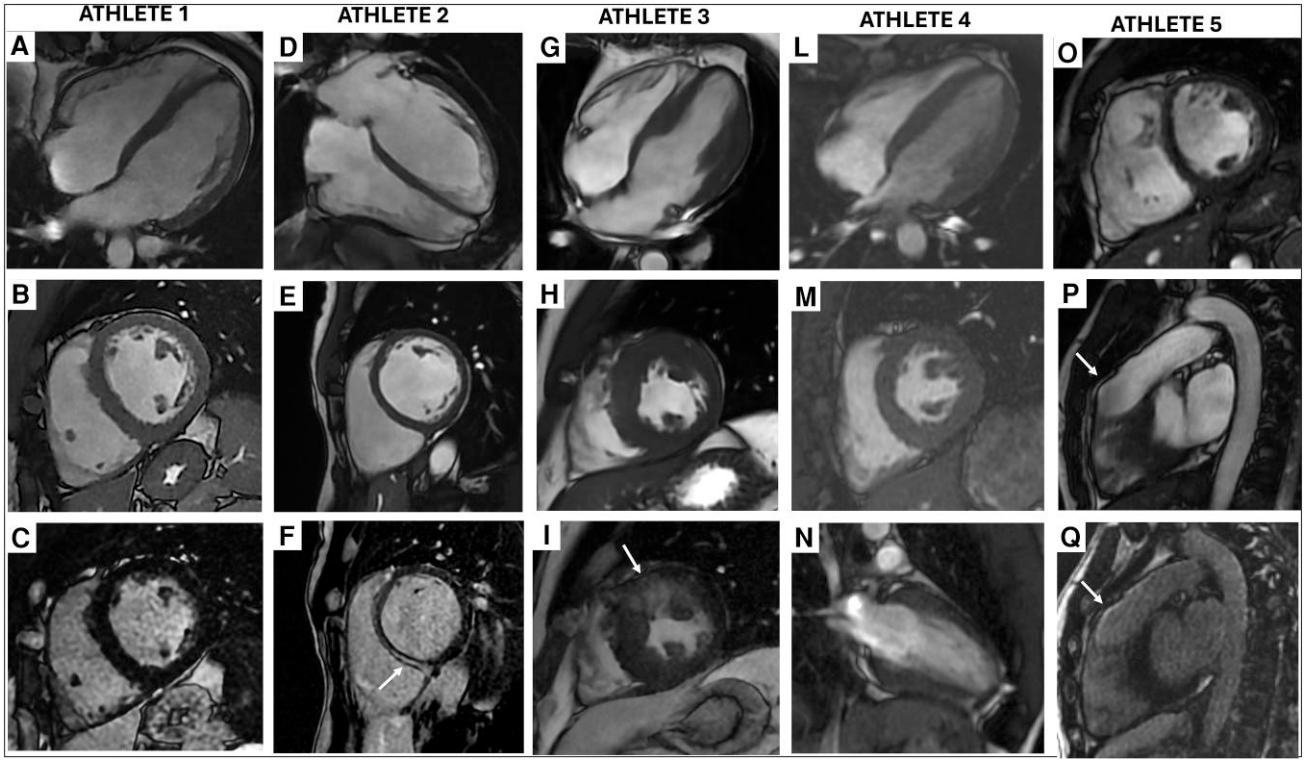

Figure 5 Examples of athletes with physiological remodelling and with cardiomyopathies. **Athlete 1** (*A–C*) shows physiological adaptation, with balanced dilatation of both ventricles and concomitant left ventricular hypertrophy (maximal wall thickness 13 mm) without LGE. **Athlete 2** has dilated cardiomyopathy: the four-chamber view and short-axis images (*D–E*) show unbalanced LV dilatation without concomitant LVH. Post-contrast images demonstrate LGE at the inferior septum (*F*, white arrow). **Athlete 3** has hypertrophic cardiomyopathy with predominant septal involvement (*G–H*), with LGE in the region of maximal hypertrophy (*I*, white arrow). **Athlete 4** presents mild LVH (13 mm) without concomitant LV dilatation (*L–N*) suggesting an early stage of HCM considering the T negative waves in the ECG. **Athlete 5** has arrhythmogenic cardiomyopathy, with RV dilatation (*O*), budging and LGE at the site of RV bulging (*P–Q*, white arrow).

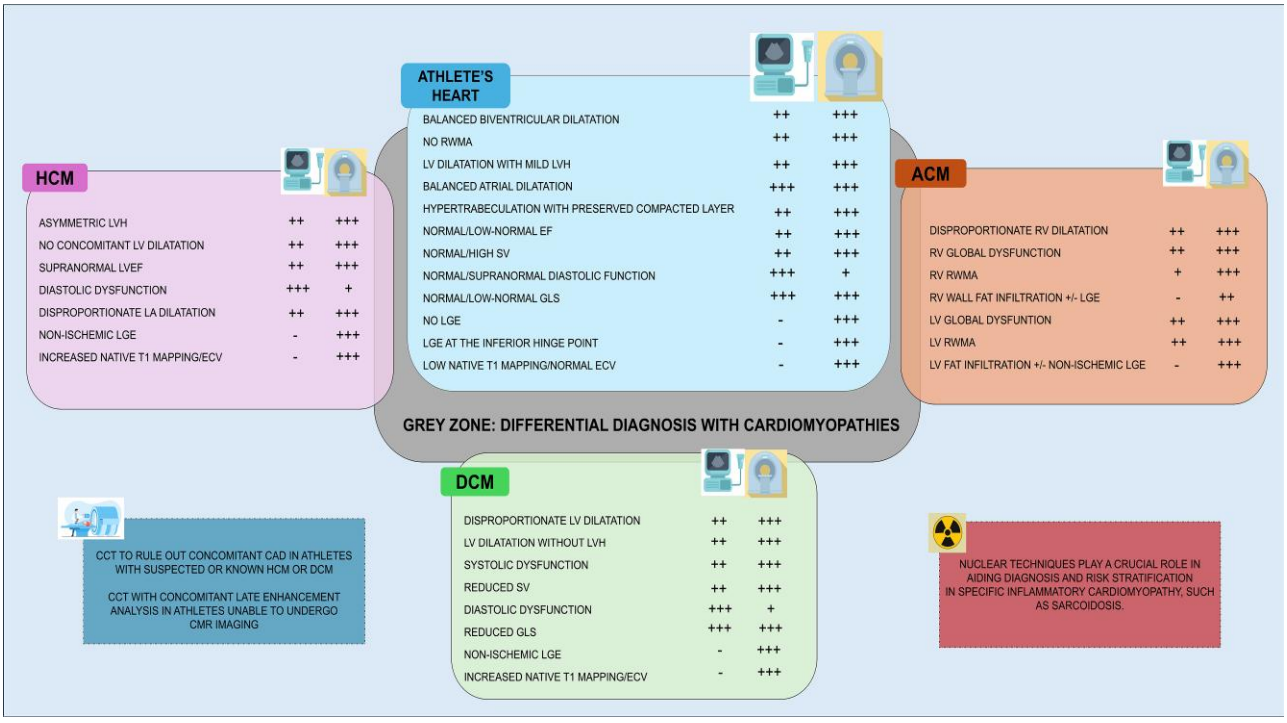

Figure 6 Imaging features of athlete's heart vs. cardiomyopathy assessed by echocardiography and CMR and the additional role of CT and nuclear imaging. ACM, arrhythmogenic cardiomyopathy; CCT, cardiac computed tomography; DCM, dilated cardiomyopathy; ECV, extracellular volume fraction; EF, ejection fraction; GLS, global longitudinal strain; LGE, late gadolinium enhancement; LV, left ventricle; LVH, left ventricular hypertrophy; HCM, hypertrophic cardiomyopathy; RV, right ventricle; RWMA, regional wall motion abnormalities; SV, stroke volume.

(routinely echocardiography, while stress CMR is currently limited to a select number of centres) can help distinguish the two entities[71] (*Figure 6*).

CMR results should be evaluated in conjunction with the clinical history, examination, and other available tests and in selected cases a re-evaluation after detraining may be helpful.[72]

CMR for the differential diagnosis of arrhythmogenic cardiomyopathy

Over time, the clinical paradigm of arrhythmogenic right ventricular cardiomyopathy has evolved from a primary focus on severe RV disease and malignant VAs to a more comprehensive concept that encompasses biventricular or left-dominant forms of the disease.[58] In contrast to the previous 2010 Task Force Criteria,[73] the updated European Task Force consensus report specifically refers to an arrhythmogenic cardiomyopathy (ACM), improving the detection of LV involvement.[74]

It is important to understand that the utility of morpho-functional abnormalities to distinguish subjects with ACM from healthy endurance athletes may be limited, as both groups can exhibit significant changes in cardiac volumes and EF.[35,66] In a subgroup of highly trained endurance athletes, up to 16% may present low EF. It has been reported that highly trained cyclists the RVEF of 44 ± 3% and/or LVEF 47% to 50%.[66] On the other hand, highly trained athletes presenting with life-threatening VAs[75–77] and underlying structural damage, as evidenced by the presence of LGE or low voltage areas on electro-anatomical mapping, often display only a mild degree of ventricular dysfunction, which is not distinguishable from athletic cardiac remodelling. Caution is needed when evaluating cardiac volume and function in athletes suspected of having ACM. In such cases, additional evaluation with exercise imaging (CMR or echocardiography)[70,78,79] and strain analysis[80] may be a useful diagnostic approach.

When evaluating the RV, both the presence of RWMA and global dilation or dysfunction is required to fulfil a major morpho-functional criterion. RWMA, even in the absence of global ventricular dilation or dysfunction, is highly specific to ARC and is not expected in healthy athletic cardiac remodelling[81,82] (*Figure 5O–Q*).

A key update in the latest task force criteria is the inclusion of LV non-invasive tissue characterization by CMR (i.e. LGE imaging), which is now considered equivalent in weight to invasive endomyocardial biopsy.[83] Importantly, LGE at the RV insertion points (hinge-point fibrosis) is a common finding in athletes and is regarded as benign. It is also essential to differentiate small mid-wall patches of LGE, which have been reported in up to 10–20% of MA,[84] from larger, striae (or band) pattern of myocardial LGE, as only the latter is considered indicative of left form of arrhythmogenic cardiomyopathy (*Figure 3C* and *D*).[83]

Although CMR has low diagnostic sensitivity for detecting of LGE in the RV due to the thin RV wall and suboptimal resolution, detecting RV LGE is a valuable sign for ACM diagnosis after ruling out other causes of scarring, including ischaemic heart disease or congenital conditions such as Tetralogy of Fallot. Consequently, the presence of LGE in at least one RV region,

as identified through CMR myocardial tissue characterization, has been established as a minor RV criterion[74] (*Figure 6*).

CMR for the differential diagnosis of left ventricular excessive trabeculation

The latest version of the guidelines considers excessive trabeculation as a phenotypic trait that can occur either in isolation or in association with other developmental abnormalities, LVH, dilatation, and/or systolic dysfunction.[58] A recent expert panel paper on excessive trabeculation of the left ventricle and the ESC guidelines for the management of cardiomyopathies[58,85] provide three key messages:

(1) Replace the term 'non-compaction' with 'hypertrabeculation' with or without associated cardiomyopathy;
(2) Excessive trabeculation may be present as a normal variant;
(3) The presence of a trabecular pattern should not determine, *per se*, a clinical management.

Investigations have documented a reversible phenotypic expression in athletes characterized by enhanced ventricular trabeculation. This is recognized as a morphological by-product of increased cardiac preload requirements induced by rigorous physical training. The incidence of cases meeting the criteria for excessive trabeculation, as determined through echocardiographic assessments, varies, with reported rates ranging from 1.4% to 8.1% among competitive athletes.[86–90] Such variability is influenced by differing diagnostic criteria, ethnic backgrounds, and the nature of the athletic disciplines considered.

Rather than being distracted by the presence or extent of excessive trabeculation in athletes, the focus for clinicians should be on interpreting the history, physical examination and information from clinical tests, including cardiac imaging, to determine whether ventricular function and the degree of cardiac remodelling, such as dilatation and hypertrophy, are consistent with an athlete's heart. The thickness of the compacted layer should also be considered. If there is any concern in the clinical information available that leads to a suspicion that findings are not entirely consistent with physiological cardiac remodelling, only then should athletes be managed according to the guidelines as likely DCM, with potential follow-up, advice to adapt exercise regimens or initiate specific medical management for DCM.

Genotype-positive phenotype-negative athletes and screening of first-degree relative of patients with cardiomyopathy

In individuals who are judged to be genotype-positive and phenotype-negative by echocardiography or family members (FMs) of patients with an inherited cardiomyopathy, CMR may identify subtle phenotypes of disease expression and should be considered in these individuals as part of their baseline assessment, as reported in the 2023 ESC Guidelines for the management of cardiomyopathies.[58] CMR is particularly valuable in clinical scenarios including athletes with localized hypertrophy in areas where echocardiography may struggle, such as the apex or anterolateral wall,[91,92] or identifying subtle regional wall motion abnormalities, particularly in the RV.[81] Although not diagnostic, subtle structural abnormalities (i.e. abnormal papillary muscle insertions,[93] septal convexity,[94] and mitral valve leaflets abnormalities[91,95]) should be documented. Another common scenario in which CMR can increase the diagnostic yield is athletes with isolated T-wave inversion. Among 69 athletes diagnosed with cardiomyopathy after comprehensive evaluation for deep T-wave inversion, echocardiography was diagnostic in 37 (54%) cases. CMR identified an additional 24 cases of cardiomyopathy or healed myocarditis, in whom the echocardiogram was reported as suspicious or normal.[56] CMR may be useful in younger athletes where disease phenotype may be subtle, but the risk of SCD from cardiomyopathy during sports is considered higher. CMR is crucial for detecting isolated non-ischaemic LGE when no other morphological features on echocardiography suggest structural heart disease, as in the context of non-dilated left ventricular cardiomyopathy (NDLVC), where the presence of non-ischaemic LGE is fundamental for the diagnosis. The evaluation of tissue characterization should include pre- and post-contrast T1 mapping. T1-mapping is advisable, particularly in mild disease and/or young FMs, in which LGE may be absent.[63,96,97] Based on both the initial assessment and clinical course, CMR scans should be repeated at follow-up to establish a definite diagnosis and monitor disease progression.[58]

The additional value of CCT for differential diagnosis between cardiomyopathies and athletes' heart

CCTA is primarily utilized to rule out concomitant CAD or myocardial bridging in individuals with HCM. This is because many of them exhibit confounding symptoms and similar ECG patterns. Additionally, it is employed to reasonably exclude ischaemic aetiology in individuals with a dilated left ventricle and impaired contraction. CCT is also important for evaluating individuals with cardiomyopathy when CMR is not an option due to claustrophobia or implantable devices that may cause artefacts and inadequate image quality. In this setting, CCT enables precise definitions of cardiac volume and function, accurate measurements of LV wall thickness, including its apical portion, and can also be used for tissue characterization to identify myocardial scar or fat. Delayed myocardial enhancement using iodine is the equivalent of LGE at CMR and is able to detect and characterize the pattern of myocardial scar in different forms of cardiomyopathy.[98] The scar pattern may help to determine the precise nature of cardiomyopathy; for example, ACM vs. sarcoid cardiomyopathy and CT contrast studies perform equally well compared with CMR-contrast ones,[99] although myocardial oedema assessment is poor. Overall, CCT can provide diagnostic and prognostic information comparable to CMR in patients unable to undergo CMR.[100]

The additional value of nuclear imaging for the differential diagnosis between athlete's heart and cardiomyopathies

In individuals with suspected or established DCM or NDLVC, the role of radionuclide imaging is limited. However, 18F-FDG can aid in identifying myocardial inflammation associated with active sarcoidosis and other atypical forms of myocarditis.[58] Isolated cardiac uptake has also been described in patients with ACM in the period of 'hot phases'. Moreover, PET using 15O-water, 13N-ammonia or 82-Rubidium, combined with dipyridamole, adenosine or regadenoson, evaluates microvascular dysfunction in HCM, DCM, or NDLVC phenotypes, offering predictive value for adverse outcomes irrespective of the cardiomyopathy subtype.

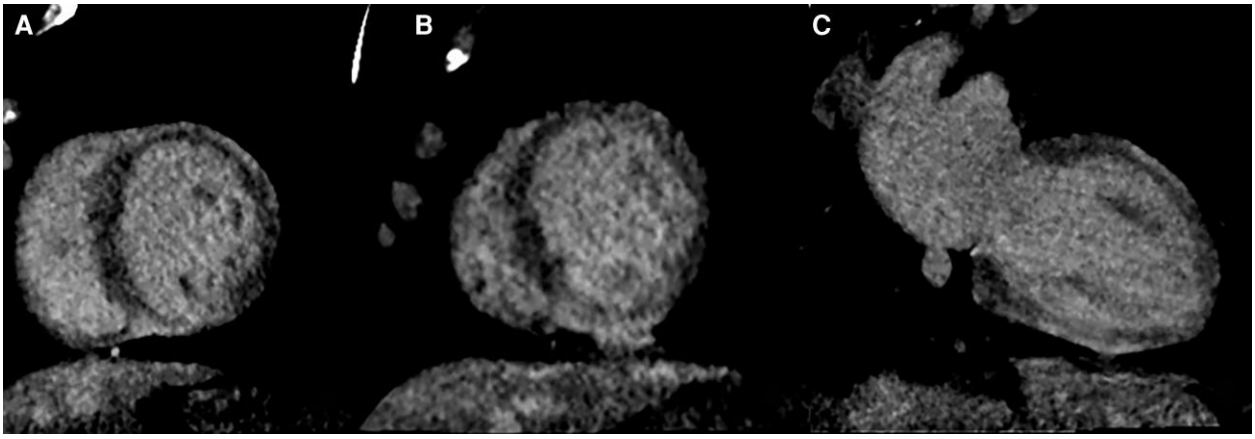

Figure 7 Myocardial late iodine enhancement at Cardiac CT. A case of 35-year-old male athletes with sustained ventricular tachycardia with contraindication to CMR (claustrophobia). Cardiac CT dedicated to late iodine enhancement well highlighted the presence of LIE with non-ischaemic pattern on both anterior and inferior wall of left ventricular (panel *A* and *B*, left ventricle short axis; panel *C* left ventricle long-axis view).

Inflammatory myocardial disease and pericarditis

The role of CMR

CMR is crucial for diagnosing inflammatory diseases of the myocardium and pericardium and for follow-up studies evaluating treatment response and residual effects.[101]

Myocarditis and pericarditis, like any other inflammatory process, are responsible for the appearance of oedema, hyperaemia, and myocardial necrosis with fibrosis. T2-weighted imaging detects myocardial and pericardial oedema as an increased signal intensity, while the presence of fibrosis may be detected by LGE.[102] The presence of myocardial LGE indicates increased extracellular space due to necrosis and, oedema in the acute phase (up to 3 months) and to myocardial scar in the chronic phase. Classical CMR criteria, the so-called Lake-Louise criteria for the diagnosis of myocarditis,[103] were recently updated to include T1 and T2 mapping as possible contributing techniques.[104]

In pericarditis, pericardial inflammation is associated with neovascularization and chronic inflammation. For pericarditis, the following features are those to be taken into account: (i) pericardial thickening (>2 mm), (ii) pericardial oedema on T2-weighted imaging, (iii) pericardial LGE, and (iv) pericardial effusion.[101]

Since myocarditis and pericarditis are both inflammatory conditions, the timing is important: when clinically suspected, CMR should ideally be performed within 2 weeks of symptom onset. Follow-up CMR should be performed at least 1 month after the acute phase, ideally 3–6 months, to detect residual LGE, which indicates myocardial fibrosis in myocarditis and chronic pericardial inflammation in pericarditis.

For athletes, evidence of myocardial and pericardial inflammatory findings on CMR is important for the diagnosis and for objectively assessing clinical remission and time to return to play, which should be associated with normalization of clinical, laboratory, and CMR findings (specifically the absence of oedema and the assessment of possible residual LGE).[3] The persistence of ongoing inflammation evolving into chronic myocarditis can lead to prolongation of the withdrawal from sports participation.

The role of CT

Recent data demonstrated the feasibility and diagnostic accuracy of myocardial fibrosis detection by cardiac CT vs. CMR.[98] Even if CMR remains the gold standard for myocardial tissue characterization, CT with a dedicated acquisition protocol to identify late iodine enhancement (LIE) may represent a good alternative, especially in specific settings[75,105] (*Figure* 7). Of interest, the radiation dose needed for myocardial/pericardial inflammation and fibrosis assessment is negligible and usually below 1 mSV.[98] These characteristics make CCT an appropriate alternative to CMR for identification or exclusion of the presence of myocardial fibrosis in young athletes, when CCT is performed for CCA evaluation. Similarly, LIE could be identified in acute or sub-acute pericarditis as with CMR, but this application is supported by very few data. On the contrary, CCT may provide important information regarding the presence of systemic disease in very selected cases of idiopathic pericardial effusion.[76] However, the routine use of CCT for diagnosis and prognosis evaluation in both myocarditis and pericarditis is still not supported by large clinical trials and should be limited to very selected cases, especially among young athletes.

The role of nuclear imaging

Nuclear imaging techniques, including SPECT and PET, provide crucial insights into myocardial inflammation, perfusion defects, and viability, supporting the early detection and precise characterization of myocarditis and pericarditis.

PET imaging with 18F-FDG may be useful in patients with suspected myocarditis who cannot undergo CMR.[106] In subjects with suspected cardiac sarcoidosis, PET imaging with 18F-FDG and perfusion tracers helps exclude alternative diagnoses (e.g. ischaemic heart disease) and provides patterns of inflammation/fibrosis that are helpful for detecting detect active myocardial inflammation.

Coronary disease

Athletes with cardiovascular risk factors

Regular physical activity plays a crucial role in managing several risk factors associated with CAD.[3] Given that CAD can often be subclinical or unrecognized, it's essential to assess CAD risk before participating in physical activities. The 2021 ESC Guidelines on cardiovascular disease prevention focus primarily on assessing the risk factors for atherosclerotic CAD, which remains a major cause of morbidity and mortality in Europe.[107] The guidelines recommend using an updated SCORE algorithm to estimate an individual's 10-year risk of fatal and non-fatal CAD events, such as myocardial infarction and stroke. This algorithm, known as SCORE2, categorizes individuals aged 40–69 years into low-to-moderate, high, or very high risk based on non-modifiable factors such as age, sex, and country, as well as modifiable risk factors such as smoking status, blood pressure, and non-HDL cholesterol. For individuals aged ≥70 years, a separate algorithm, SCORE2-OP, applies.

Asymptomatic athletes identified as having a high or very-high-risk profile according to the SCORE2 risk charts should undergo initial medical screening, including clinical examination, ECG, and an exercise test.[40] If the initial screening is negative, there is typically no need for further tests. However, it's crucial to remain vigilant for mild symptoms suggestive of chronic coronary syndrome and initiate appropriate investigation when necessary. Based on these premises, while regular physical activity confers numerous health benefits, it is essential to tailor exercise recommendations based on individual risk profiles, especially in CAD. Pre-participation assessment using risk algorithms such as SCORE2 can help identify potential risks and guide appropriate management strategies for athletes and individuals engaging in physical activity.[107]

CCT

CCT, including unenhanced CT for CACS and CCTA, has the highest negative predictive value for ruling out CAD among non-invasive investigations, in both obstructive (≥50% stenosis) and non-obstructive (<50% stenosis) disease.[108] Recent studies using CACS and CCTA have shown that active middle-aged male athletes with a history of high lifelong exercise exhibit a have prevalence of CAC and atherosclerotic plaques.[16] However, the most active group displayed a more benign coronary atherosclerosis phenotype, characterized by a higher prevalence of calcified plaques[16] except in the MASTER@HEART study.[18] In asymptomatic MA, CACS greater than 400 provides strong prognostic value compared with CACS of zero.[109] Therefore, CACS may be appropriate in MA (over of 35 years of age) as a useful marker to improve cardiovascular risk stratification.[110] CCTA may be particularly useful for evaluating MA with an abnormal or borderline stress test, or when clinical suspicion is raised by factors such as age, sex, symptoms, or risk-SCORE assessment.[111] The recent update to the Italian recommendations on sports cardiology[112] recommended CCTA in athletes when three or more elevated cardiovascular risk factors are present, regardless of stress ECG findings. CCTA is the only non-invasive imaging modality that can provide detailed plaque characterization and quantification. The application of the latest scanner generations, such as photon-counting CT, will further reduce the radiation dose. When combined with AI-based image post-processing, plaque assessment and prognostication will further improve.[113,114] The role of CCTA as a primary imaging test is expected to increase in younger asymptomatic athletes with abnormal stress tests and a higher risk profile of CAD. However, further investigations are warranted to clarify the role of CCTA in this context, and it is important to note that there is still no evidence that the use of CACS or CCTA in primary prevention reduces the risk of SCD or cardiac events in athletes.

Nuclear imaging

In general, nuclear imaging is rarely indicated in asymptomatic athletes. It can be performed in subjects with a high or very high-risk profile, particularly when CAD is highly suspected or documented after the initial evaluation, to re-stratify risk and guide management. As compared to other stress imaging tests, in populations with low prevalence of obstructive coronary disease, nuclear imaging is more sensitive for the recognition of inducible myocardial ischaemia.[105]

Suspected or known chronic coronary artery disease in master athletes

CMR

For the anatomical diagnosis of atherosclerotic coronary artery disease, CCTA is the gold standard. However, to evaluate whether CAD is causing myocardial ischaemia, myocardial perfusion imaging with CMR using adenosine or regadenoson is recommended in the 2024 ESC Guidelines for the management of chronic coronary syndrome[108] Following stress perfusion imaging, LGE images are obtained to assess the presence of previous myocardial infarcts. Findings are reported as 'ischaemia' in case of a stress perfusion defect without, or with limited, subendocardial myocardial infarction, or as 'no ischaemia' in case of a stress perfusion with transmural myocardial infarction. Visual assessment of the stress perfusion images is well established for both diagnosis and prognosis, and quantitative analysis may offer more precise information in multi-vessel disease, being less observer dependent.[115] Dobutamine stress perfusion and cine imaging are preferred in young with CCA to detect myocardial ischaemia.[116] In the diagnostic work-up for athletes with acute coronary syndromes, CMR cine, T2-weighted and LGE imaging can be of clinical value, especially in patients with a working diagnosis of myocardial infarction with non-obstructive coronary arteries (MINOCA).[117] In the evaluation of suspected MINOCA, CMR reclassifies the majority of patients to the correct diagnosis,[118] and can rule-out true MINOCA, which is important since a normal scan carries a good prognosis.[119]

CT

CCTA plays a role on in diagnosis and prognosis in MA with suspected or established CAD[3] (*Figure 8*). Both CAC and CAD are more common in long-term endurance MA than in non-athletes matched for age and risk profile[16–18,120–122] (*Table 6*), but the prognostic significance of these findings remains unclear. Regarding diagnosis, CCTA is today the most accurate non-invasive test for identifying significant CAD (sensitivity in diagnosing >50% stenosis vs. invasive angiography of 97%).[6] Based-on prospective trials, CCTA is integrated in the routine clinical management of patients with suspected CAD as a first-line diagnostic and prognostic method. The addition of CCTA to the standard clinical care markedly clarifies the diagnosis of chest pain due to CAD and results in a better management, exemplified by a reduction >40% in the incidence of myocardial infarction, according to the SCOT-HEART trial.[123,124] For athletes, the 2020 ESC Guidelines on sport cardiology do not routinely recommend CCTA for first-line screening of those at

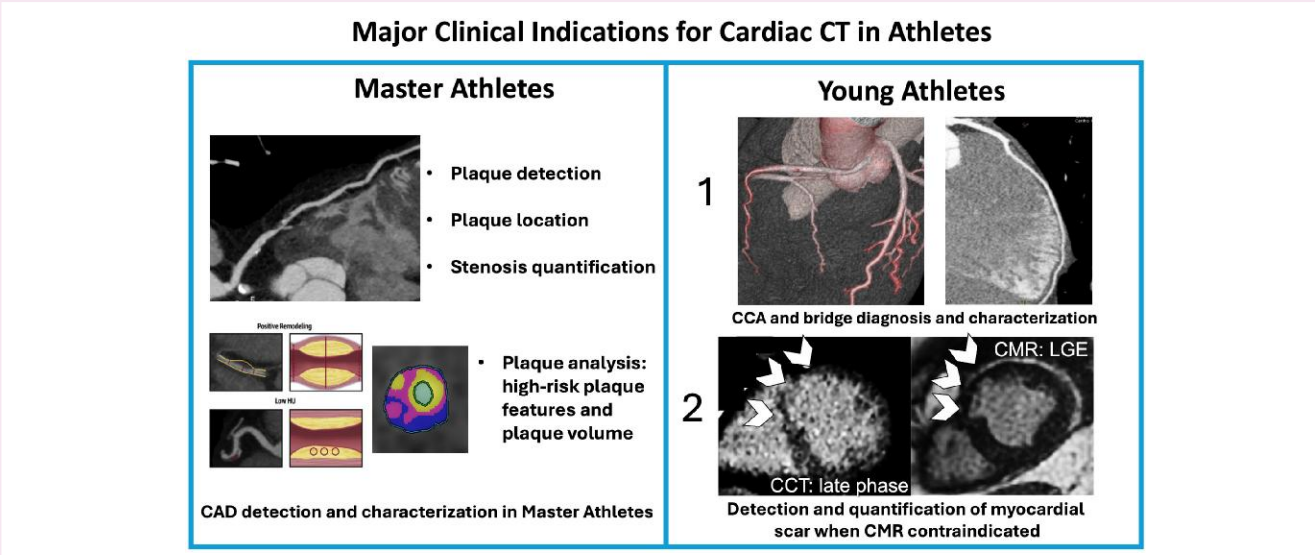

Figure 8 Major clinical indications for Cardiac CT in young and master athletes.

low risk. However, CCTA is recommended if the exercise stress test is equivocal or the ECG is uninterpretable.[3,108]

In athletes with known CAD, ESC recommends for the risk stratification additional functional testing (maximal stress testing) to anatomical imaging (CCTA, invasive angiography).[3] Moreover, CCTA may offer prognostic information in athletes with CAD. In some multicentre studies, identification of high-risk coronary features, including positive remodelling (PR) and low-attenuation plaques by CCTA, improved the prognostic stratification, compared to that derived from the degree of stenosis alone[7,8,125] (*Figure 9*). Recently, the ESC Guidelines on chronic coronary syndrome[108] recognized that the assessment of fractional flow reserve CT-derived (FFRCT) may be considered in patients with a known intermediate coronary artery stenosis in a proximal or mid-coronary segment on CCTA (Class IIb) and for identifying patients at high risk of adverse events in case of one vessel disease of the proximal LAD ≥70% stenosis on CCTA (Class I). Two recent studies have shown that FFRCT is feasible and effective for evaluating coronary physiology in endurance athletes[126,127] and may be particularly useful when moderate or heavily calcified stenoses have been detected at CCTA, allowing deferral of additional functional tests such as nuclear imaging.

Nuclear imaging

In general, nuclear cardiology plays an important role in diagnosing and stratifying the risk of patients with suspected or known CAD,[108] as well as after coronary intervention. This also applies to athletes, with special consideration. Regarding diagnosing CAD in asymptomatic athletes, the recent ESC Guidelines on Sports Cardiology,[3] recommend a maximal exercise test or an equivalent stress test for athletes with a high-risk SCORE. Myocardial perfusion imaging serves this role by providing valuable insights into myocardial blood flow and perfusion, aiding early detection of ischaemia. For athletes, the preferred method of stress is exercise, which provided valuable functional information. A detailed description of the role of nuclear imaging in athletes with suspected or known CAD is reported in the part of this Consensus Document dedicated to stress imaging modalities.

Congenital coronary anomaly in young athletes

Adult (sports) cardiologists may come across very selected CHD, with CCA being the most commonly encountered.[128,129] CCA have an estimated prevalence of 0.21–5.79% in the general population.[1] Of note, they are the second most common cardiovascular cause (14%) of SCD in athletes under 35 years of age 90. Risk is particularly significant if a coronary artery originates from the opposite sinus of Valsalva and has an intraarterial course.[1]

CMR

ESC guidelines on adult CHD[130] indicates CCT as the preferred technique for the evaluation of CCA, while AHA considers either CT or CMR angiography. The latter is a good alternative because it does not use radiation, which is particularly important for younger patients.[131] Evaluation for CCA is indicated in symptomatic athletes or when CCA is suspected on echocardiography.

For CMR assessment, 3D whole-heart coronary CMR angiography is the reference technique. A recent study compared the use of respiratory self-navigated coronary artery CMR at 1.5 T with CT angiography and demonstrated that CAA could be detected with high accuracy.[132] The cine stack on the aortic valve can also help identify the coronary origins. Other abnormal appearances in the four-chamber view, including the 'RAC sign'[23] of the 'crossed aorta'[24] should also be noted and should raise suspicion.

Reports should always include the anatomic origin of the coronary artery, any coronary artery abnormalities, and their relationship with other cardiac structures. If available, the presence and severity of inducible ischaemia related to the anomalous coronary artery should be assessed and described.

CCT

CCA is classified as an abnormality of origin or course. CCT provides an accurate evaluation of coronary artery origin and course (i.e. inter-arterial vs. retro-aortic) with identification of

Table 6 Main studies correlating coronary atherosclerosis and endurance activities

Author	No. of athletes	Type of activity	Endpoints	Conclusion
Merghani (Circulation 2017)[17]	152	ran ≥10 miles or cycled ≥30 miles per week and have continued to do so for ≥10 years, and competed in ≥10 endurance events	Coronary artery calcium (CAC); Plaque composition.	Male athletes are more likely to have a CAC score >300 Agatston units or coronary plaques compared with sedentary males with a similar risk profile.
De Bosscher (Eur Heart J. 2023)[18]	382	cycling ≥8 h or running ≥6 h per week, or triathlon (combination of swimming, cycling, and running) ≥8 h per week for at least 6 months prior to baseline. Lifelong and late-onset athletes started regular endurance exercise training at <30 years and >30 years of age, respectively.	Prevalence of coronary plaque and type of plaque	Lifelong athletes had more coronary plaques and more coronary proximal plaques (calcific, noncalcific, mixed)
Aengevaeren (Circulation 2017)[16]	289	Running, Cycling, Tennis with different levels of intensity as light (<3 METs), moderate (3 to 6 METs), vigorous (6 to 9 METs), or very vigorous (≥9 METs)	CAC; Progression of plaques;	Exercise intensity but not volume was associated with progression of coronary atherosclerosis
Mohlenkamp (Eur. Heart J. 2008)[122]	108	Running at least 5 marathons during the previous three years	CAC; LGE	CAC distribution was similar in athletes and in age-matched controls; CAC and the number of number of marathons predict the presence of LGE
Schwarts (Mo. Med 2014)[120]	50	Male runners who had participated in 25 consecutive Twin Cities Marathon races	Association the correlation between CAC and risk factors	CAC score is related to CAD risk factors and not the number of marathons run or years of running.
Jafar (Mayo Clin. Proc. Innov. Qual. Outcomes 2019)[121]	56	Runners or triathletes who had run competitively for 10 or more years	CAC	Significantly higher rate of coronary artery calcification existed in long-term marathon, ultramarathon, and extreme runners than in sub-marathon runners

high-risk features like the slit-like ostial morphology, the acute take-off angle and the presence of orifice >2 cm above the sino-tubular junction, and length of lumen narrowing[133] (*Figure 8*). Similarly, CCT may provide accurate identification of myocardial bridges, whose length and depth can be quantified with high accuracy (*Figure 10*), parameters associated with risk stratification for adverse events at follow-up.[134] In particular, the bridge is considered 'deep' if its depth into the myocardium is ≥2 mm and 'long' if its length is ≥2.5 cm.[24] A follow-up in MA with a bridge may be recommended to assess plaque progression, in particular at the site of the bridge entrance. Taking into consideration that most of patients and athletes with CCA needing advanced cardiological evaluation are of young age, it should be underlined that CCT in these patients using a dedicated acquisition protocol and a last-generation scanner should be performed with less than 1–2 mSV. A major strength of CCT in athletes is the possibility of knowing the coronary and aorta anatomy from the same study as the exclusion of CAD. Results of cardiac imaging may then guide sports participation counselling for patient-athletes.[3] Sport cardiologists may encounter in their practice adult with CH where there is anomalous orientation of the coronary arteries as per pathology (i.e. tetralogy of Fallot) or where coronary arteries have been reimplanted (i.e. dextro-Transposition of the great arteries). CCT findings for such a peculiar population should always be discussed and interpreted together with CHD specialist and integrated with functional test like stress echocardiography. Beyond the CCA field, CCT may also be useful for assessing cardiac and vascular anatomy after surgical or interventional procedures in case of subjects with operated adult CHD, such as atrial or ventricular septal defect, patent foramen ovale or bicuspid aortic valve, to evaluate their eligibility to return to sport activities.

Nuclear imaging

In the ischaemic evaluation of CCA, SPECT-myocardial perfusion imaging (with physical rather than pharmacological stress) has been proposed as first-line imaging method. In several studies, SPECT has shown favourable diagnostic results. PET-myocardial perfusion imaging could alternatively be appropriate. Patients presenting with CCA and ischaemic symptoms may not necessarily

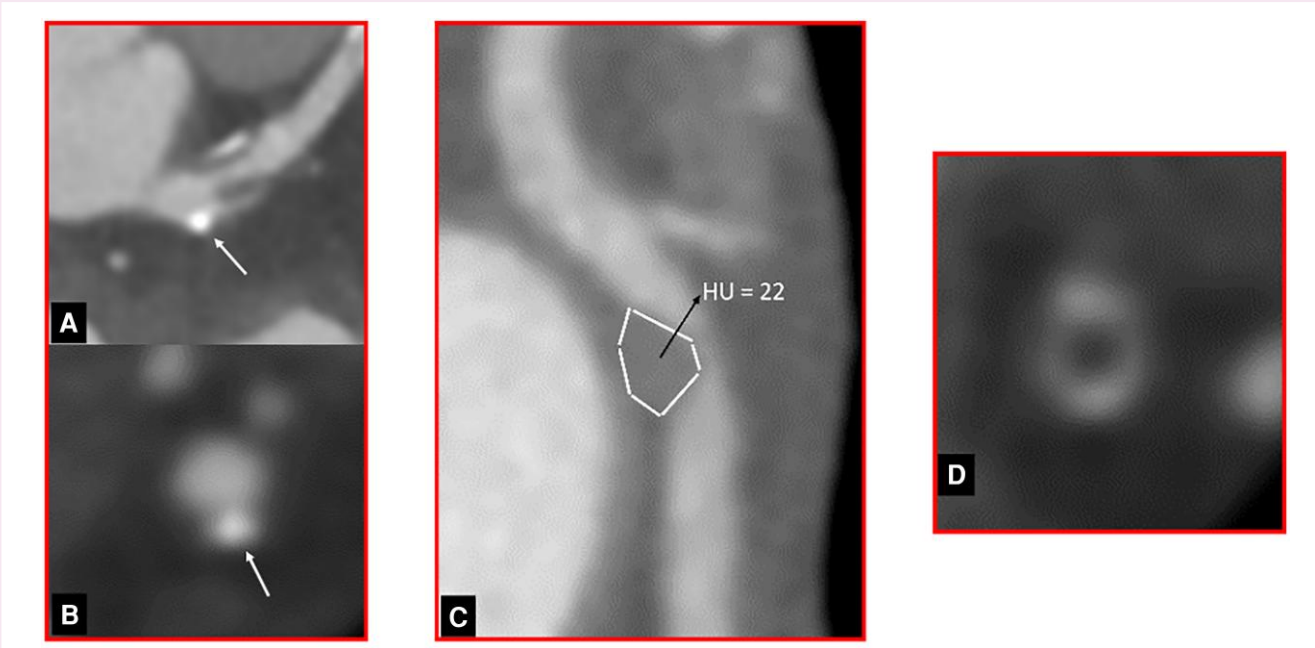

Figure 9 High-risk coronary plaque characteristics at CCTA. In (*A*—long axis) and (*B*—cross section) an example of spotty calcification is illustrated (white arrow). A plaque characterized by positive remodelling (white contour) and low attenuation value (HU = 22) is represented in panel (*C*). In (*D*) an example of Napkin-ring sing is depicted.

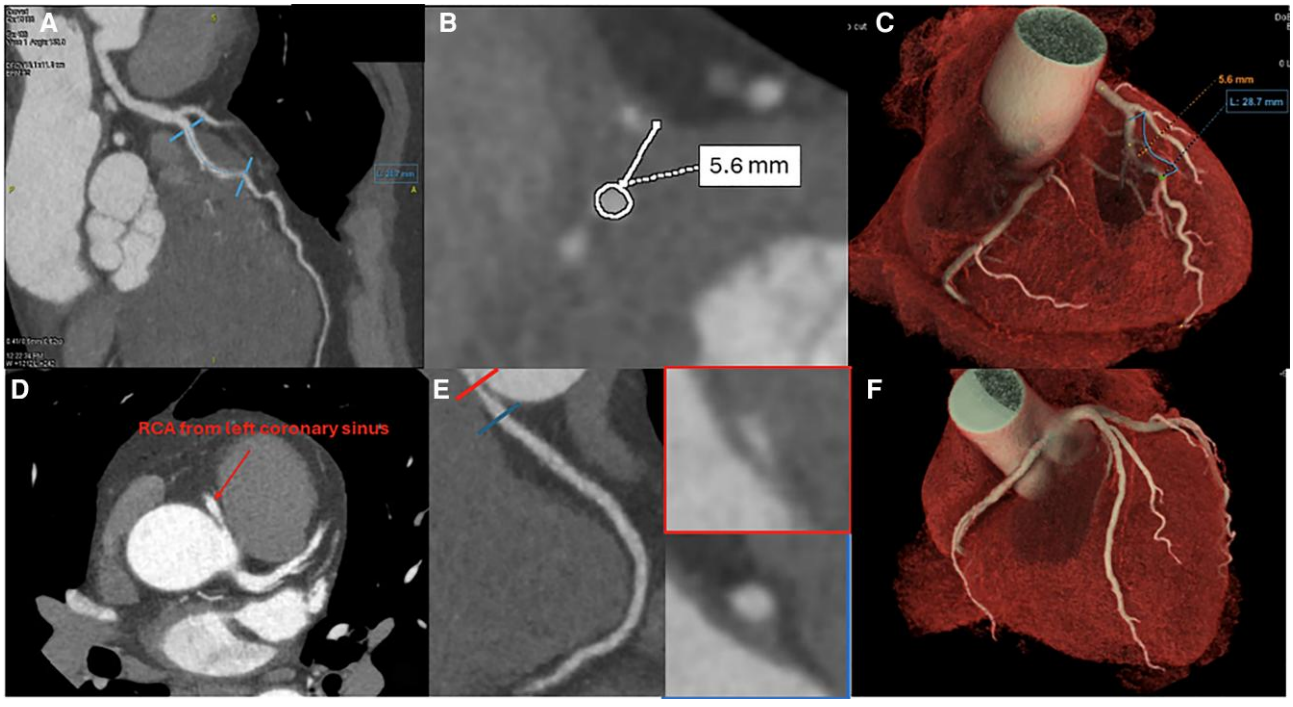

Figure 10 CCTA characterization of a myocardial bridging and a CCA. Deep and long myocardial bridge of left anterior descendent artery (mid segment) (panel *A*); cardiac CT enables accurate evaluation of depth (5.6 mm in panel *B*) and length (26.7 mm in panel *C*). In panel *D*, *E* and *F* a congenital coronary artery abnormality with right coronary artery from the left coronary sinus; cardiac CT highlights the presence of ostial slit-like morphology (panel *E*).

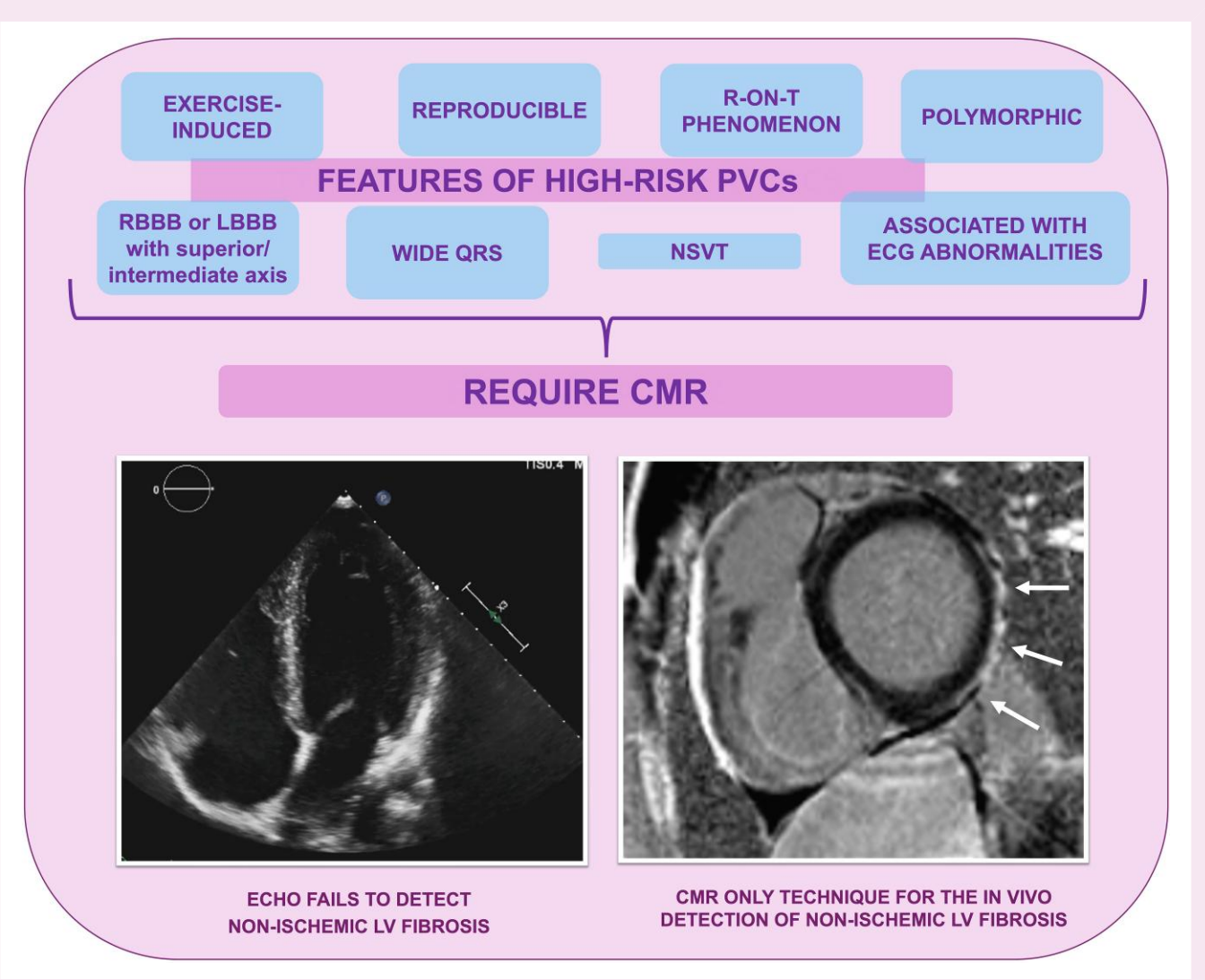

Figure 11 PVCs with features considered at high risk to be associated with non-ischaemic LGE. In this figure the high-risk features of PVS are reported and an example is provided. On the left there is the echocardiogram of an athlete with PVCs with high-risk features. The echocardiogram is normal, but the CMR reveals non-ischaemic LGE (sub-epicardial distribution). LBBB, left bundle branch block; LGE, late gadolinium enhancement; LV, left ventricle; NSVT, non-sustain ventricular tachycardia; RBBB, right bundle branch block.

have CCA as the cause of ischaemia, especially in the absence of high-risk features. Additional factors contributing to myocardial ischaemia could involve coexisting atherosclerotic coronary artery disease (especially in middle-aged or elderly patients), or in rare cases, microvascular disease, and coronary vasospasm.[135]

Hybrid imaging (that combines CCTA with SPECT or PET) may help to determine if ischaemia is anatomically related to the CCA.[136] Notably, only exercise-SPECT/PET must be performed, while drug testing with vasodilators cannot replicate the ischaemic mechanism in athletes with CCA.

Other common clinical indications

Ventricular arrhythmias in athletes

The role of CMR

VAs and specifically premature ventricular arrhythmias (PVCs) in competitive athletes may be a benign phenomenon, but they can also be a sign of a concealed life-threatening condition. The evaluation of athletes with VAs requires integrating several aspects derived from clinical history (age, family history of SCD, atherosclerotic cardiovascular disease risk factors, symptoms), physical examination, 12-lead ECG, and maximal exercise testing.[137] A qualitative analysis of PVCs is warranted for the appropriate management of competitive athletes.[3,11,129] Morphology and complexity of PVCs, rather than their frequency, can predict the probability of an underlying structural heart disease.[138] Indeed, in the presence of arrhythmias with high-risk (e.g. RBBB with a wide QRS or left bundle branch block, LBBB with intermediate or superior axis, couplets with short RR interval, non-sustained VT, short coupling interval with 'R on T' phenomenon), particularly when complex and with an increased burden during exercise, the indication for CMR is crucial, even if echocardiography is normal.[139] CMR is able to unmask life-threatening conditions such as NDLVC including LV non-ischaemic scar and arrhythmogenic left ventricular cardiomyopathy, all of which can present with normal

echocardiographic findings,[58,140,141] A careful analysis of the resting ECG is needed, as T-wave inversion, complex PVCs and exercise-induced PVCs with RBBB or polymorphic morphology have been identified among the predictors of abnormal findings on CMR.[142,143] More recently, the association of uncommon PVCs with low QRS voltages and the reproducibility of these findings at repeated exercise testing has been demonstrated to improve risk stratification and appropriate CMR referral of athletes with apparently idiopathic PVCs[144,145] (*Figure 11*).

The presence of PVCs with a low-risk pattern, in the absence of heart disease, is typically associated with a good prognosis. These PVCs are not associated with tachycardia-mediated cardiomyopathy even in the presence of a high arrhythmic burden. They are typical of young athletes and usually do not require CMR.[3,138,141]

Notably, when deciding on sports eligibility, the morphology of PVCs can also be extremely useful in interpreting LV non-ischaemic scars related to previous myocarditis or cardiomyopathies, in determining whether PVCs originate from the anatomic site where LGE is identified.

In conclusion, in selected cases of athletes with uncommon PVCs, CMR allows an early diagnosis of concealed, life-threatening cardiac conditions where echocardiography can be completely normal (e.g. LV non-ischaemic scar). CMR should not be mandatory for all athletes with PVCs, particularly when the PVCs are common and have a benign prognosis.

The role of CCT and nuclear imaging

While monomorphic infundibular or fascicular PVCs that are suppressed during exercise are generally benign in the absence of other risk features,[137] high-risk feature of PVCs and/or abnormal findings from history and/or physical examination warrant the prescription of second line tests.[146] Given its potential to reliably delineate coronary artery anatomy, rule out obstructive CAD, and accurately characterize the atherosclerotic plaque burden, CCTA is indicated in case of high-risk PVCs/VT especially in MA (>40 years old in both males and females); furthermore, CCTA is also indicated in younger athletes with high-risk VAs in case of suspicion of CCA.[49,147] Exercise-induced VAs may be associated with an increased risk of death and cardiovascular events during follow-up across the spectrum of pre-test probability of CAD, in both symptomatic[148] and asymptomatic adult/senior subjects.[149] Indeed, high-grade PVCs occurring during recovery were associated with long-term risk of cardiovascular mortality in asymptomatic individuals, whereas PVCs occurring only during exercise were not associated with such an increased risk.[150]

In nuclear imaging techniques, SPECT as PET can be used to identify and quantify myocardial ischaemia,[108] and to provide prognostic information in subjects with exercise-induced VAs at intermediate to high pre-test probability of CAD.[108] Furthermore, SPECT and PET may provide functional information that may can non-invasively complement the anatomical information provided by CCTA, when appropriate approaches are used to minimize radiation exposure.[108] In particular, there is a growing interest in stress PET which is able to provide information on absolute myocardial blood flow (MBF) and on MBF reserve[151] which increases the sensitivity in detecting myocardial ischaemia. Quantitative perfusion PET is also useful for identifying coronary microvascular dysfunction in patients with angina or ischaemia in the absence of epicardial obstructive CAD (ANOCA/INOCA) and may inform treatment.[108] SPECT and PET are not currently included in the suggested work-up for VAs secondary to CAD in most recent ESC guidelines on the management of VAs.[152] However, SPECT and PET can be considered in patients with high PVC and in those patients with high chest muscle mass, who might have CTA quality issues. In highly selected cases, i.e. patients with coexisting high-risk VAs, atrioventricular conduction disturbances (especially high-degree atrioventricular block), and/or left ventricular dysfunction/scarring (especially basal septal thinning) in the absence of CAD or another explanation, fluorine-18 fluorodeoxyglucose-positron emission tomography (18F-FDG PET) is indicated, and may allow the non-invasive diagnosis of cardiac and/or extracardiac sarcoidosis.[106,153] Finally, PET may also provide diagnostic and prognostic information in the highly selected group of patients with suspected arrhythmic myocarditis, or with active inflammatory phases of genetic cardiomyopathies, although athlete-specific data on this topic are currently lacking.

Aortopathy and bicuspid aortic valve

CMR

As in the general population, the bicuspid aortic valve (BAV) is the most common CHD in adult athletes. Echocardiography is the first-line imaging technique for the evaluation. Nevertheless, CMR may provide additional information that becomes essential in certain clinical scenarios.

Cine CMR sequence offers a precise evaluation of aortic valve morphology and motion, as well as a reliable quantification of aortic stenosis by direct orifice planimetry. CMR can be particularly useful in cases of a sub-optimal acoustic window. Additionally, CMR is the 'gold-standard' for assessing LV function and volumes, allowing an accurate assessment of LV remodelling over time and the distinction between a physiological response to exercise or the result of the volume/pressure overload induced by a dysfunctional aortic valve. Normal CMR values for LV function and volumes are available for adult endurance athletes and should be used in this population (refer to paragraph 2.1.3 and *Table 3*).

In addition, phase-contrast flow mapping sequences in CMR quantify *trans*-aortic flows with high accuracy and reproducibility, which is particularly useful for assessing complex regurgitant jets that are inadequately evaluated by echocardiography. The evaluation of the origin of coronary arteries should be integrated into the scan. CMR is also a reliable and reproducible imaging modality for evaluating BAV-associated aortopathy and other aortopathies. Electrocardiogram-gated contrast-enhanced 3D angiography or 3-dimensional SSP of the aorta offers a multiplanar visualization of the entire aorta and allows the identification of BAV-associated congenital defects such as aortic coarctation or patent ductus arteriosus, as well as an accurate measurement of aortic diameters without the use of ionizing radiation.[99] CMR is recommended for the monitoring of thoracic aortic dimensions over time as indicated in the 2024 ESC Guidelines for the management of peripheral arterial and aortic diseases,[100] in whom a long-term follow-up is warranted and limiting radiation exposure is of particular importance. No specific aortic dimensions normograms are available for the athletic population; thus, in the absence of dedicated data, normograms for the general population should be applied. Likewise, the use of Z-score is indicated in children and adolescent athletes, as well as in those with extremely small or large body sizes. Finally, in recent years, 3D-cine phase-contrast with three-directional velocity encoding (4D-flow) has emerged to study intravascular flow allowing the estimation of wall shear stress. These sequences have aided our understanding of the mechanisms underlying BAV-associated aortic dilation, though they currently remain a research tool.[58]

CCT

The extent of aortic remodelling due to increased parietal tension secondary to vigorous athletic exercise is usually mild. Data from young athletes <35 years have highlighted mildly larger aortic root diameters compared with control subjects, although absolute aortic diameters in young athletes rarely exceeded 40 mm in males and 34 mm in females.[154] Data in professional athletes occasionally showed higher frequency (up to 21%) of aortic enlargement (>40 mm) compared to controls with matched cardiovascular risk. It is unlikely that such mild anatomical remodelling is associated with an increased risk of aortic events.[155]

Actual dilatation of the thoracic aorta is usually asymptomatic and often remains unnoticed on physical examination and by ECG assessment. In any case, due to the large size of many athletes, it could be reasonable to index the aorta calibres to the body-surface area or better to height.[156] Presentation as an acute aortic syndrome is a very uncommon, well-recognized cause of SCD in athletes.[3] Confirmation of a dilated aorta mandates that subjects are assessed, case by case, for the risk of aortic rupture. Hereditary thoracic aortic conditions such as Marfan's syndrome, bicuspid aortic valve, positive family history of sudden cardiac death or of non-fatal acute aortic syndrome, large aortic diameters, arterial hypertension, as well as practice of vigorous strength exercise in predisposed individuals confer a higher risk of aortic rupture.[157] Echocardiography can be used routinely to monitor ascending aorta size and aortic valve function. Echocardiography, however, cannot provide a comprehensive evaluation of the entire aorta.[158] CCT angiography is a superior method for depicting the baseline anatomy of the entire aorta. These modalities define the size and length of aneurysmal foci throughout the aorta and can characterize aortic wall changes (dissection, atherosclerotic plaques). In CMR, CT may also be required when echocardiography provides discordant measurements. Compared with echocardiography, CT is typically less operator-dependent and less affected by patient anatomy/acoustic windows. However, CMR may be preferable to CT to minimize lifetime radiation exposure, particularly in young individuals undergoing long-term surveillance.[157,159] CT may be indicated if there are contraindications to CMR or for accurate planning of percutaneous or surgical intervention. Patients with a hereditary thoracic aortic condition should undergo regular imaging screening of the ascending aorta. The schedule may vary case by case and should generally take advantage of the mix of available imaging modalities. The general recommendation is to perform a baseline assessment by CT or CMR, then followed by echocardiography, if all segments of the dilated aorta can be comprehensively evaluated and there are no discordant measurements.[159]

In athletes with a non-dilated aorta (<40 mm), whether bicuspid or tricuspid, a follow-up interval of up 2–3 years is recommended in the ESC Guidelines on the diagnosis and treatment of aortic diseases, usually with echocardiography.[157,159] In athletes with moderately dilated aorta (40–45 mm), bicuspid or tricuspid valve, the follow-up interval may be ideally every year, depending on growth rate. In athletes with a dilated aorta (>45 mm), annual echocardiography, or more frequent echocardiography in cases of accelerated growth, is suggested.[159]

BAV is the most common congenital abnormality with a prevalence of 2–3% among young athletes and up to 6% of patients presenting for transcatheter aortic valve replacement (TAVR).[160] There have been a number of proposed classifications of BAV morphology, including the most accepted Sievers, which essentially focuses on the number of raphes, i.e. rudimentary soft tissue related to non-separation of two cusps.[160] More recently, a TAVR focused and simplified BAV classification has been proposed. It helps to differentiate BAV morphology regarding (i) the number of commissures (two vs. three) and (ii) the presence or absence of a raphe which has been explored extensively in the TAVR literature and recent CT guidelines.[161] There are known differences in the incidence of ascending aortic dilation and aneurysms, which are less common in non-raphe type Bicuspid Valves than traditional Sievers BAV.[162] CCT plays a fundamental role in distinguishing a raphe from non-separation of 2 cusps or a so-called tri-commissural bicuspid valve, by providing consistent anatomical clarity with raphe terminating below the sino-tubular junction whereas fused cusps with tri-commissural anatomy extends to the sino-tubular junction.[163] Characterization of the aortic root, assessment of calcification distribution and of the aortic annular geometry are key components in the evaluation of bicuspid aortic valve disease in athletes and non-athletes alike.[164]

Nuclear imaging

Nuclear imaging does not have a current clinical role in the assessment of athletes with suspected aortopathy. However, several novel techniques hold promise in refining the risk stratification of patients with aortic disease. In particular, PET imaging enables quantification of the activity of specific pathological processes within the aorta, using 18F-FDG and 68Ga-DOTATATE to assess inflammatory activity and 18F-fluoride to measure calcification activity.[165,166]

Mitral valve prolapse

CMR

Mitral valve prolapse (MVP) is a common valve abnormality[167] in Western countries with a similar prevalence (0.6–3.1%) among athletes compared to the general population. Generally, MVP is a benign condition without significant mitral regurgitation or haemodynamic impact on the LV. However, a small yet poorly defined subset of individuals remains at a higher risk of malignant VAs and SCD with an annual incidence of <1% in unselected individuals with MVP, though no definite data exists for athletes. Recent studies using CMR have described the morphological and tissue abnormalities associated with 'arrhythmic' MVP, which have been used for risk stratification (mitral annulus disjunction and the presence of non-ischaemic LGE at the basal infero-lateral wall of the LV).[168]

In athletes, the presence of MVP requires two different clinical workups. First, if MVP is associated with moderate-to-severe regurgitation, echocardiography remains the imaging modality of choice. However, if an arrhythmic burden is documented on echocardiography in athletes with MVP without significant regurgitation, CMR could be performed, particularly in athletes with VAs with a RBBB morphology and a wide QRS, or with symptoms, such as syncope or family history of SCD.

The acquisition protocol should include traditional long-axis views and a short-axis cine stack covering the entire ventricle (pay attention to include part of the left and right atria for full coverage since systolic-diastolic excursion may be exaggerated in MVP). Additional radial cine sequences for mitral valve segmentation can be useful to clarify valve anatomy. Phase contrast sequences may be useful for quantifying regurgitation and excluding an intracardiac shunt as a source of arrhythmias. Finally, post-contrast sequences are mandatory to evaluate the possible presence of LGE.

In addition to a standard assessment of the severity of any potential regurgitation, for MVP the following parameters should

Table 7 Key messages on the role of CMR, CCT and nuclear imaging in athletes

HCM	CMR provides superior spatial resolution compared with echocardiography, enabling accurate quantification of LVH distribution and detection of subtle or localized patterns (e.g. apical, inferior, lateral) that may be missed on ultrasound. In athletes with T-wave inversion and normal echocardiography, CMR significantly improves diagnostic yield. LGE identifies replacement myocardial fibrosis, present in ~65% of patients with HCM but not in athletes. Inferior RV insertion point differs between HCM and athletes: athletes present hinge-point LGE patterns (finding not correlated with prognosis) while HCM presents mid-myocardial LGE. T1 mapping and ECV quantification provide incremental tissue characterization: physiological LVH presents lower T1/ECV due to predominantly myocyte hypertrophy. CCT with concomitant late enhancement analysis can offer similar diagnostic and prognostic information for athletes unable to undergo CMR.
DCM	Balanced LV/RV dilation with mild LVH, high SV and low resting HR supports physiological adaptation, whereas isolated or predominant LV enlargement with low SV suggests DCM. Very large LV dimensions accompanied by wall thinning or reduced function—particularly in strength/skill athletes- raise suspicion for pathology. Mid-wall LGE, especially in the septum, supports a diagnosis of DCM when present, although its absence does not exclude it. CTT is primarily utilized to rule out concomitant CAD in athletes with suspected or known DCM. CCT with concomitant late enhancement analysis can offer similar diagnostic and prognostic information for athletes unable to undergo CMR.
ACM	RWMA—even without global dilation—are highly specific for ACM and are not seen in healthy athletes. Benign hinge-point fibrosis at RV insertion sites is common in athletes and should not be misinterpreted as ACM. A striae pattern of LV LGE is strongly suggestive of arrhythmogenic LV involvement. RV LGE has limited sensitivity due to technical challenges, but it is a pathological finding when present. CCT with concomitant late enhancement analysis can offer similar diagnostic and prognostic information for athletes unable to undergo CM.
Hypertrabeculation	Excessive trabeculation can be found as a normal physiological variant, particularly in athletes exposed to high preload condition and may be reversible if function, chamber enlargement and wall thickness remain appropriate for training load. CMR contributes to comprehensive assessment by evaluating ventricular volumes, systolic function, wall thickness and patterns of remodelling to determine whether findings align with physiological athlete's heart or suggest underlying pathology.
G+/P- or FMs	CMR can detect early localized structural abnormalities not visible on echocardiography (e.g. apical hypertrophy, subtle RV abnormalities, non-ischaemic scar). CMR is valuable for surveillance, helping monitor progression in younger genotype-positive athletes where disease phenotype may be subtle, but the risk of SCD during sports is nevertheless higher. CMR is part of their baseline assessment
Inflammatory diseases	CMR is essential for diagnosing myocardial and pericardial inflammation and for follow-up to assess treatment response and residual damage. Optimal diagnostic performance requires CMR early (within 2 weeks of symptom onset), especially myocarditis, with follow-up after recovery to document resolution or persistent fibrosis. CCT may represent an alternative to CMR for identification or exclusion of myocardial fibrosis even in young athletes who have an indication to CT for assessing a suspected CCA. A routine use of cardiac CT for diagnosis and prognosis evaluation in both myocarditis and pericarditis is not advised. Nuclear imaging techniques offer an alternative option for myocardial inflammation in case of CMR contraindication.
Master athletes	Asymptomatic MA at high or very high risk according to ESC Prevention Guidelines SCORE2 models may be considered for non-invasive imaging, if CAD is suspected after initial evaluation, aligning with current ESC Guidelines for chronic coronary syndromes/ESC Sports Cardiology guidelines. CACS might be considered in MA as marker to improve cardiovascular risk stratification. CCTA provides detailed plaque characterization and quantification and may be proposed in asymptomatic MA with abnormal stress tests or high clinical suspicion of CAD. MA with previously unrecognized anginal symptoms, abnormal or equivocal exercise tests, or moderate obstructive CAD on CCTA may benefit from functional stress tests, particularly stress nuclear imaging with SPECT or PET, which offer sensitive detection of myocardial ischemia.

Continued

Table 7 Continued

	CCTA might be advisable in asymptomatic lifelong MA with elevate CV risk profile or with ECG abnormalities at rest.
	CCTA is advised in all cases when exercise stress test is equivocal or the ECG is uninterpretable.
	CMR stress perfusion allows detection of myocardial ischaemia and guide management when coronary atherosclerosis is suspected.
CCA in young athletes	CMR angiography is a radiation-free alternative to CT and is particularly valuable in younger athletes when evaluating suspected CCA
	When present, inducible ischaemia related to the CCA can be assessed with stress CMR to guide risk stratification and sports eligibility.
	CCTA is the gold standard for identification and evaluation of origin an course of CCA.
	PET/CT and PET-myocardial perfusion imaging are the method of choice to detect ischaemia in the presence of CCA, however, concomitant other factors such as atherosclerotic coronary artery disease, microvascular disease, or coronary vasospasm should be appropriate.
	Hybrid imaging (i.e. SPECT/PET and CCTA) may be an option for assessing the anatomical situation of a CCA in relation to ischaemia
VAs	CMR can identity concealed structural heart disease (e.g. non-ischaemic LV scar, arrhythmogenic cardiomyopathy) in patients with PVCs with high-risk features: RBBB with a wide QRS or left bundle branch block, LBBB with intermediate or superior axis, complex, with an increased burden during exercise; R on T phenomenon, TVNS) especially when ECG shows additional abnormalities (e.g. T-wave inversion, low QRS voltages).
	CMR is not routinely required in athletes with low-risk arrhythmias, normal evaluation, normal ECG, where prognosis is generally benign.
	CCTA is indicated to rule out coronary artery disease in MA with high-risk PVCs/VT and to identify coronary artery anomalies in younger athletes when suspected.
	SPECT/PET can assess inducible ischaemia and provide prognostic information, complementing CCTA when anatomical and functional correlation is needed.
	FDG-PET is useful in highly selected athletes with ventricular arrhythmias and suspected inflammatory or infiltrative disease (e.g. cardiac sarcoidosis, arrhythmic myocarditis).
BAV and aortopathy	Mild aortic remodelling (with absolute dimensions still within normal limits) due to increased parietal tension of the aorta can be secondary to vigorous athletic exercise.
	CMR provides accurate assessment of aortic valve morphology, function, and severity of stenosis/regurgitation, especially when echocardiographic windows are suboptimal.
	CMR is the gold standard for quantifying LV volumes and function, helping distinguish athlete's remodelling from pathology related to pressure or volume overload.
	CMR enables comprehensive evaluation of BAV-associated aortopathy, including precise measurement of thoracic aorta dimensions and identification of associated congenital abnormalities—without radiation exposure.
	Athletes with moderately dilated aorta (40–45 mm), bicuspid or tricuspid valve, should have periodical follow-up with echocardiography. In athletes with dilated aorta (>45 mm), follow-up should be close (every year). In both cases, CCT may be the modality of choice for follow-up where echocardiography is difficult due to imaging windows or provides inconsistent data.
	Characterization of the aortic root and assessment of calcification distribution and annular geometry by CCT are key components in the evaluation of bicuspid aortic valve in athletes and non-athletes alike.
	Nuclear imaging does not have a current clinical role in the assessment of athletes with suspected aortopathy and heart valve disease.
MVP	CMR improves risk stratification by identifying specific arrhythmic MVP features: mitral annular disjunction (MAD) and non-ischaemic LGE in the basal inferolateral LV wall/papillary muscles.
	CMR is indicated in athletes with MVP and VAs or concerning symptoms (e.g. syncope), even when mitral regurgitation is not severe on echocardiography.
	CMR can provides accurate assessment of regurgitation severity, valve anatomy, and LV remodelling when ultrasound windows are inadequate.
	CCT plays a growing role for the diagnosis and characterization of mitral valve prolapse complementing echocardiography.

ACM, arrhythmogenic cardiomyopathy; BAV, bicuspid aortic valve; CCA, Coronary arteries anomalies; DCM, dilated cardiomyopathy; ECV, extracellular volume fraction; FMs, family members; G+/P–, genotype positive, phenotype negative; HCM, hypertrophic cardiomyopathy; HR, heart rate; LBB, left bundle branch block; LGE, late gadolinium enhancement; MA, master athletes; MVP, mitral valve prolapse; LV, left ventricle; LVH, left ventricular hypertrophy; PVCs, premature ventricular contractions; RBBB, right bundle branch block; RV, right ventricle; RWMA, regional wall motion abnormalities; SV, stroke volume; VAS, ventricular arrhythmias.

be included: leaflet involvement (bileaflet or only anterior/posterior); leaflet thickness, maximal systolic excursion specifying whether it appears on a 2- or 3- chamber view; presence of mitral annular disjunction (defined as the separation between left atrial (LA) wall at the level of MV junction and the LV free wall) and curling (defined as unusual systolic motion of the posterior mitral ring on the adjacent myocardium)[169]; the presence of a typical pattern of LGE in MVP (mid-myocardial stria on infero-lateral basal wall of the LV± at the level of the papillary muscles).[170]

CT

Cardiac CT plays a growing role in the diagnosis and characterization also of mitral valve prolapse, complementing echocardiography and CMR particularly as it relates to the confirmation of the presence and extent of mitral annular disjunction. While echocardiography remains the gold standard for the localization of the prolapsing scallop and the regurgitant jet, CT provides additional information on peri-valvular anatomy.[171]

Conclusion

Advanced cardiovascular imaging plays a central role in the evaluation and management of athletes, enabling accurate differentiation between physiological adaptations to exercise and potentially life-threatening cardiac disease. CMR is the reference technique for the comprehensive assessment of cardiac structure, function and tissue characterization, enabling early detection of cardiomyopathies and inflammatory diseases, particularly when echocardiography is inconclusive. CCT provides an unparalleled definition of coronary anatomy, making it the gold standard for identifying coronary anomalies in the younger athletes and characterizing atherosclerotic disease in MA, while also supporting assessment of aortic pathology and valvular disease when echocardiography or CMR is limited or contraindicated. Nuclear imaging complements these modalities by identifying inducible ischaemia and active inflammation in selected clinical scenarios, refining risk stratification and management decisions (*Table 7*).

Appropriate test selection, standardized protocols, and expert interpretation that integrate sport-specific cardiac adaptation—are essential to avoid misdiagnosis and unnecessary restriction from sport. Importantly, imaging findings must always be contextualized within the athlete's clinical presentation, training profile, and competitive environment.

Overall, CMR, CCT and nuclear imaging significantly enhance diagnostic precision and prognosis assessment in athletes, ultimately supporting safe participation in sports and optimizing long-term cardiovascular health. Developing standardized protocols for athletes will ensure consistency and reliability across different institutions, facilitating large-scale research and data assessment across populations. Longitudinal studies could provide insights into the long-term effects of high-intensity training on cardiac health and inform the development of preventive and therapeutic strategies. Progress in these research areas could significantly improve our understanding and treatment of cardiac conditions in athletes.

Acknowledgements

This document has been reviewed by Vincent Aengevaeren, Philippe Bertrand, Yohann Bohbot, Giovanni Di Salvo, Erwan Donal, Niall Keenan, Aniela Monica Petrescu, Ivan Stankovic, Valtteri Uusitalo, and Alessandro Zorzi.

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Conflict of interest: S.E.P. provides consultancy to Circle Cardiovascular Imaging, Inc., Calgary, Alberta, Canada. J.L. is a consultant to and holds stock options in Heart Flow. The rest of the authors declare that they have no conflicts of interest. The following co-authors are Associate Editors of the European Heart Journal Cardiovascular Imaging: Ana G. Almeida, Pal Maurovich-Horvat, Robin Nijveldt, and Danilo Neglia.

Data availability

There are no new data associated with this article.

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