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Role of cardiac magnetic resonance imaging in troponinemia syndromes

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Abstract

Cardiac magnetic resonance imaging (MRI) is an evolving technology, proving to be a highly accurate tool for quantitative assessment. Most recently, it has been increasingly used in the diagnostic and prognostic evaluation of conditions involving an elevation in troponin or troponinemia. Although an elevation in troponin is a nonspecific marker of myocardial tissue damage, it is a frequently ordered investigation leaving many patients without a specific diagnosis. Fortunately, the advent of newer cardiac MRI protocols can provide additional information. In this review, we discuss several conditions associated with an elevation in troponin such as myocardial infarction, myocarditis, Takotsubo cardiomyopathy, coronavirus disease 2019 related cardiac dysfunction and athlete's heart syndrome.

Key Words: Cardiac magnetic resonance imaging; Troponin; Myocardial infarction; Myocarditis; Takotsubo cardiomyopathy; COVID-19; Athlete's heart

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Core tip: Cardiac magnetic resonance has excellent spatial resolution to assess ventricular volumes and function. It is also continuing to evolve to provide key diagnostic and prognostic information particularly through the use of gadolinium contrast agent for the conditions presented in this review.

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INTRODUCTION

Troponinemia describes an elevation in serum troponin levels that can result from a myriad of conditions such as acute myocardial infarction (AMI), takotsubo cardiomyopathy (TTS), myocarditis and athlete's heart syndrome (AHS). More recently, severe acute respiratory syndrome coronavirus 2 has been linked to cardiac disease and elevated troponin levels. Given that troponinemia is nonspecific, establishing a definitive diagnosis can be difficult. Fortunately, cardiac magnetic resonance imaging (CMRI) has the ability to characterise myocardial tissue and identify unique pathological features of cardiac disease. As a diagnostic tool for these conditions, CMRI imaging may be promising.

This narrative review gives an overview of the diagnostic features and potential role of CMRI in conditions associated with troponinemia such as myocardial infarction (MI), TTS, myocarditis, coronavirus disease 2019 (COVID-19) related cardiovascular disease and AHS (Table 1).

MI

AMI is one of the most common causes of elevated troponin levels. It is defined by the presence of acute myocardial injury in conjunction with dynamic changes in troponin levels, and evidence of myocardial ischaemia[1]. Electrocardiography (ECG), transthoracic echocardiography (TTE) and invasive coronary angiography are the standard of care in the evaluation of MI. Adjunctive use of CMRI can be useful to confirm the diagnosis, assess chronicity, guide management and aid prognosis. It can be utilised to distinguish the changes seen between an acute and an established, or also called chronic MI. CMRI is also highly accurate at assessing ventricular volumes and function with superior spatial resolution, contrast-to-noise ratio and tissue characterisation compared to TTE.

On CMRI, AMI can demonstrate ventricular regional wall motion abnormalities (RWMAs) corresponding with the affected vascular territory on cine images. Intramyocardial haemorrhage may occur, and is represented by a hypointense zone in the infarcted area on T2-imaging or mapping[2]. Following ischaemic insult from coronary artery obstruction, myocardial cellular injury begins in the subendocardial region, and continues to extend towards the subepicardium if there is ongoing oxygen deprivation. This process is known as the "wavefront phenomenon of myocardial death", named by Reimer *et al*[3]. Late gadolinium enhancement images characteristically demonstrate a hypodense core surrounded by an area of hyperenhancement (Figure 1), and is found in a subendocardial or transmural distribution depending on the extent of the MI[4-6]. In chronic MI (CMI), cine images will typically show wall thinning, RWMA, and a lack of oedema on T2-weighted images[7]. In a study by Rehwald *et al*[8], the authors used rabbit models to demonstrate that gadolinium contrast agent uptake was greater in infarcted myocardial tissue. In another study, Kim *et al*[9] demonstrated that the extent of transmural hyperenhancement reflected the degree of irreversible injury. Native T1-sequences have also been explored by Kali *et al*[10], who demonstrated CMRI may be useful in diagnosing CMI, and determining likely irreversible injury. It is particularly useful in some situations, such as in renal failure patients, where gadolinium contrast is contraindicated.

Besides determining left ventricular ejection fraction (LVEF) on cine imaging, a detailed assessment of LV deformation measurements can also take place using CMRI[11]. Left ventricular global radial strain, circumferential strain and global longitudinal strain (GLS) have all shown association with increased major adverse cardiac events (MACEs)[12], and GLS has been demonstrated to be an independent predictor of post-MI clinical outcome. Impairment of left atrial strain on CMRI post-MI has also been demonstrated to be an independent predictor for increase in MACEs, as well as improving prognostic value when combined with LVEF[13,14].

It has been demonstrated that in patients with coronary artery disease, those treated with revascularisation have a significantly lower annual mortality rate compared to those treated with medical therapy [15,16]. In these patients, CMRI can be used to assess the viability of a coronary artery territory. The most important parameters are LV end-diastolic wall thickness, quantitative LV systolic or diastolic performance during low-dose dobutamine stress testing, and late gadolinium enhancement (LGE)[17].

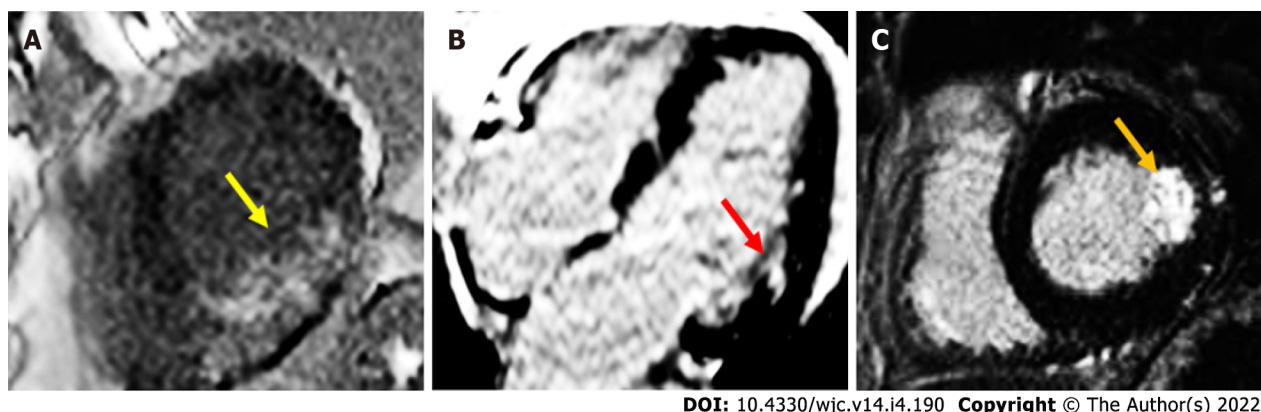
Table 1 Cardiac magnetic resonance imaging features

Condition	Cardiac magnetic resonance imaging features
Myocardial infarction	< 1 mo Myocardial oedema present on T2-weighted images, T2 mapping and T1 mapping Microvascular obstruction revealed as a hypointense core within hyperintense infarct zone in area of LGE Infarct size can be calculated using pre and post-contrast T1-weighted mapping and ECV assessment Myocardial necrosis/scar by LGE in a subendocardial or full-thickness pattern within a coronary artery territory Additionally at < 6 mo T2-weighted hyperintensity on double inversion recovery turbo spin echo
Takotsubo syndrome	Can help distinguish coexisting CAD or acute myocarditis LGE typically absent Myocardial oedema present on T2-weighted images, T2 mapping and T1 mapping Accurate assessment of WMAs on cine imaging Can be useful to identify ventricular thrombus
Myocarditis	Inflammatory hyperaemia demonstrated on T1-weighted images Myocardial oedema on T2-weighted images Myocardial necrosis/scar by LGE in a subepicardial or mid-wall pattern Greater T1 and T2 increases with acute inflammation Pericardial effusion
COVID-19 related cardiac dysfunction	Features similar to that of acute myocarditis Myocardial oedema on T2-weighted images Myocardial necrosis/scar by LGE in a subepicardial or mid-wall pattern Myocardial fibrosis using T1-weighted mapping and ECV assessment Can be useful to identify ventricular thrombus and pericardial effusion
Athlete's heart	LVH typically < 12 mm Lower ECV with LVH compared to HCM RV dilatation seen on cine imaging LGE focal and generally at the RV insertion points

LGE: Late gadolinium enhancement; ECV: Extra-cellular volume; CAD: Coronary artery disease; WMAs: Wall motion abnormalities; LVH: Left ventricular hypertrophy; HCM: Hypertrophic cardiomyopathy; RV: Right ventricular; COVID-19: Coronavirus disease 2019.

For example, in a LV segment with $\leq 50\%$ transmural LGE, a normal dobutamine response is correlated with greater functional recovery after revascularisation[18,19]. In contrast, the presence of $\geq 50\%$ transmural LGE indicates nonviable infarcted tissue[20,21]. This technique is comparable to fluorodeoxyglucose positron emission tomography, which is considered the gold standard in the assessment of myocardial viability[22]. Unfortunately, the role of CMRI can be limited in many healthcare settings, considering factors including machine access, availability of imaging experts, cost and time.

The use of CMRI in assessing prognosis following MI has shown promising results. Assessment for microvascular obstruction (MVO) through the use of first pass perfusion studies during and following gadolinium contrast administration is one of the prognostic features that has been studied. It is implicated in adverse ventricular remodelling, larger infarct size (IS) and poorer clinical outcome[23-25]. van Kranenburg *et al*[26] have also demonstrated that MVO is an independent predictor for major adverse clinical outcomes at 2 years. Infarct size on CMRI has also been shown to be strongly associated with heart failure hospitalisation and all-cause mortality[27]. More recently, postcontrast T1 mapping has been shown to accurately quantify IS in a small study[28]. CMRI has also demonstrated some correlation between IS and peak troponin I[29], however this has not been a consistent or reliable finding. Intramyocardial haemorrhage has been linked to adverse LV remodelling and increased MACEs, but heterogeneity in imaging techniques mean further study is required[23]. The presence of



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Figure 1 Cardiac magnetic resonance imaging of acute myocardial infarction. A: Short axis mid-ventricular image demonstrating almost full-thickness transmural late gadolinium enhancement (LGE) in posterolateral wall (yellow arrow); B: Four-chamber image demonstrating focal LGE in lateral wall (red arrow); C: Short axis image demonstrating > 75% transmural LGE in lateral wall (orange arrow).

LGE in patients with symptoms suggestive of MI conferred worse MACEs compared to those without LGE[30]. The prognostic importance of LVEF has been demonstrated in a number of studies[31,32]. Study delay for at least 1 wk following AMI should be considered, to allow for myocardial functional recovery as found by Mather *et al*[33], and further imaging at up to 6 mo may be required to assess stabilised LVEF[34]. LVEF $\leq$ 35% and LGE were independently associated with MACEs, with better predictive value than TTE[35]. The extent of LV scarring has also been clearly associated with risk of spontaneous ventricular arrhythmias[36–38]. Assessment of the peri-infarct, or “grey-zone”, surrounding the infarcted core may also play a role in risk stratifying post-MI patients, with increased size posing potential heightened ventricular arrhythmic risk[39]. Yan *et al*[40] used a semiautomatic software detection system to quantify percentage of abnormal myocardial delayed enhancement of tissue surrounding the infarct core, and noted that it was an independent predictor of post-MI all-cause and cardiovascular mortality.

Incorporation of artificial intelligence-based analyses will likely have a role to play in the future in cardiac outcome and prognosis prediction. It has already been demonstrated that fully automated volumetric and myocardial segmentation assessment are equally effective as manual efforts in predicting MACEs[41].

As it stands, without the availability of randomised controlled data or larger studies, the actions to be taken if high risk CMRI features are seen are not completely clear[42,43].

TTS

TTS, which is also known as Takotsubo cardiomyopathy, transient apical ballooning syndrome, broken heart syndrome and stress-induced cardiomyopathy, is a condition of transient LV dysfunction that is typically triggered by physical or emotional stress[44]. TTS mimics MI with often indistinguishable clinical presentation, ECG changes and cardiac enzyme elevation, but without angiographic evidence of acute obstructive coronary artery disease or plaque rupture[44,45]. Given the transient nature of TTS, traditionally it was thought of as a benign condition however more recent data suggests this is misguided, with complications comparable to those seen in patients with the acute coronary syndrome [46,47]. CMRI is increasingly used to diagnose and evaluate complications of TTS in both the acute and subacute setting, particularly in those with atypical features, or bystander coronary artery disease[48].

In the acute setting, CMRI can define TTS by excluding other aetiologies such as MI and myocarditis and identifying RWMA that extend beyond a single coronary artery distribution[49] (Figure 2). One of the hallmarks is reversible myocardial inflammation corresponding to RWMA[31,44]. CMRI can assess myocardial inflammation and oedema with T2-weighted images[44,50–52].

In the subacute phase, its strength in identifying subtle RWMA makes it the ideal modality to accurately assess for resolution of regional dysfunction, with full recovery being a criteria confirmation of diagnosis[44].

Late gadolinium-enhanced imaging is a valuable adjunct in confirming a diagnosis of TTS when there is coexisting coronary artery disease or suspicion for myocarditis. It is widely believed that in TTS, there is an absence of LGE on CMRI[31], however, there are studies challenging this notion, having demonstrated LGE in patients with TTS in the acute phase[52–54]. It should be noted that LGE in this setting is transient, resolving on serial imaging to confirm the diagnosis of TTS, and has been associated with increased incidence cardiogenic shock and a longer timeframe for resolution of wall motion abnormalities[55,56]. In contrast, patients with myocardial infarction will have focal subendocardial or

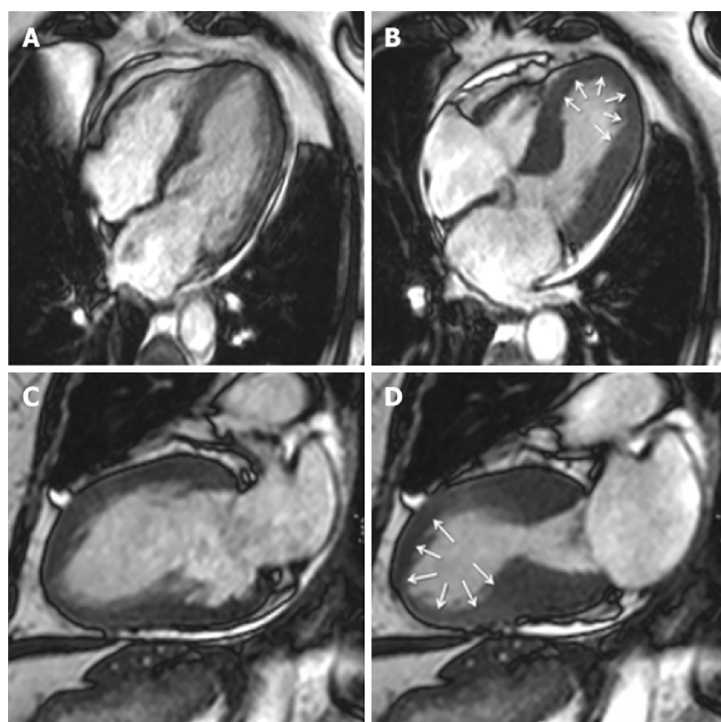


Figure 2 Cardiac magnetic resonance imaging of Takotsubo cardiomyopathy. Typical apical ballooning seen in takotsubo syndrome. A, B: Cine four-chamber in late diastole and systole respectively; C, D: Two-chamber view in late diastole and systole respectively. Modified from Plácido *et al* [123] and licensed under the Creative Commons Attribution 4.0 International License (<http://creativecommons.org/licenses/by/4.0/>).

transmural LGE evident, while those with myocarditis typically will have a mid-wall distribution of LGE[57].

In addition to confirming the diagnosis of TTS, CMRI is useful in identifying complications such as mitral regurgitation and LV outflow tract obstruction seen on blood flow imaging, pericardial effusion seen on black blood T1-weighted sequences, and ventricular (including apical) thrombi not visualised on TTE, during early gadolinium (EGE) sequences. Thrombi will appear as a low signal intensity without gadolinium uptake, in comparison to the high intensity signal from the blood pool[50,52].

It has been hypothesised that the elevated catecholamines observed in TTS have a role in the microvascular dysfunction noted in patients with TTS, correlating with improvement in myocardial function[58]. While not established in TTS, there is emerging evidence in the utility of quantitative perfusion CMRI to more objectively assess the role that microvascular dysfunction plays in this syndrome, and is subject to further research[59].

ACUTE MYOCARDITIS

Acute myocarditis is an inflammatory cardiomyopathy secondary to infectious and noninfectious conditions, sometimes associated with symptoms of heart failure developing over ≤ 3 mo. The clinical presentation can be nonspecific and may include chest pain, heart failure, cardiogenic shock, arrhythmias and/or sudden cardiac death. Early investigations may demonstrate elevated troponin levels, elevated acute phase reactants such as C-reactive protein, erythrocyte sedimentation rate and eosinophil count. An ECG may be normal, show nonspecific abnormalities or be similar to the pattern of acute pericarditis and AMI. Most importantly, it is important to exclude alternative causes such as MI. Due to the variable clinical presentation, the gold standard for diagnosis remains an endomyocardial biopsy (EMB), which is an invasive procedure that carries risk of life-threatening complications. CMRI may provide a noninvasive alternative for the assessment of myocarditis[60].

CMRI has become an important tool in the assessment of myocardial inflammation in patients with suspected myocarditis. Assessment of gross abnormalities includes changes in ventricular size and geometry, regional and global wall motion abnormalities and identification of pericardial effusion. In addition, there are techniques to assess microscopic markers of myocardial inflammation such as T1-weighted sequences for detection of myocardial hyperaemia, LGE for myocardial necrosis, fibrosis or scars and T2-weighted imaging to identify oedema[61-63]. Following early CMRI data, consensus diagnostic criteria were released and incorporated into the Lake Louise criteria[60] (LLC) (Table 2). The use of newer mapping techniques such as for native T1 and T2, and quantification of extracellular volume (ECV), in comparison to the LLC, appear superior in the diagnosis of acute myocarditis, with

Table 2 Lake Louise consensus criteria for myocarditis on cardiac magnetic resonance imaging

Two out of three criteria must be met to be consistent with myocardial inflammation:

Regional or global myocardial signal intensity increase in T2-weighted images

Increased global myocardial early gadolinium enhancement ratio between myocardium and skeletal muscle in gadolinium-enhanced T1-weighted images

At least one focal lesion with nonischaemic regional distribution in inversion recovery-prepared gadolinium enhanced T1-weighted images (late gadolinium enhancement)

positive predictive value of 90% *versus* 71%[64].

LGE has been shown to be highly accurate in the diagnosis of myocarditis with a high correlation with EMB[65]. Acute myocarditis is associated with subepicardial or mid-wall late gadolinium enhancement most commonly in the lateral, inferolateral or inferior wall[66-68] (Figure 3). In particular, small studies have suggested specific patterns for certain viruses: parvovirus B19 is associated with the lateral wall while human herpes virus 6 is linked to the septal wall[69]. The presence of LGE on follow-up studies denotes areas of irreversible myocardial injury[62,65].

CMRI diagnostic accuracy in the workup for chronic myocarditis (> 14 d) is not as well established compared to acute myocarditis, with T2-mapping providing the only discernible additional diagnostic benefit together with the LLC[66,70].

Although CMRI has demonstrated prognostic guidance in acute myocarditis, cardiac enzyme markers do not reflect the degree of myocardial injury or permanent scarring as demonstrated on CMRI LGE[70]. There are insufficient data available to relate CMRI features to independent risk of ventricular arrhythmias, although this is clearly raised in the context of impaired LV function[67,71]. In a meta-analysis, the presence of LGE, particularly anteroseptal location, has been found to be an independent risk factors for adverse cardiac outcomes, including all-cause mortality, cardiac mortality including sudden cardiac death, and MACEs[72].

COVID-19 RELATED CARDIAC DYSFUNCTION

COVID-19 infection has variable presentations, most commonly involving respiratory symptoms. However, since the declaration of the pandemic in March 2020, there have been increasing reports of cardiovascular disease. The incidence has been reported to be $\geq 40\%$, depending on the definition or population sampled[73-76]. Postmortem studies of confirmed COVID-19 cases have demonstrated the presence of the virus in the myocardium, but not necessarily with consistent expression of cardiac sequelae[77]. There are numerous mechanisms involved in myocardial injury, which include direct viral invasion and host innate immunity response, hypoxia, micro- and macrovascular thrombosis, inflammatory injury and stress-induced cardiomyopathy[78].

Considering the multifaceted components of COVID-19-induced myocardial injury, it should not be expected that the imaging findings of this infection would duplicate that of a viral myocarditis syndrome alone. Cardiac involvement in COVID-19 infection may not be present with clinically severe cardiac symptoms[75,79], and even though echocardiography is a sensitive tool to identify gross cardiac dysfunction, LVEF may be normal[77]. There have been reports of primary cardiac involvement in COVID-19[80], where CMRI can be useful in identifying acute viral myocarditis features, as well as evidence of thrombosis such as LV apical thrombus.

Studies using CMRI have demonstrated the severity of cardiovascular involvement following acute infection. In a report by Huang *et al*[81], the authors noted that 57% of patients had myocardial oedema or LGE on CMRI performed > 1 mo after development of infection (Figure 4). This suggests an ongoing pathological process affecting the myocardium. Historically, the LGE distribution in acute viral myocarditis involved the lateral and inferior walls. However with COVID-19, LGE patterns have been reported as subepicardial, mid-wall or subendocardial mimicking AMI[75]. Of note, there are no studies available where participants have baseline cardiac MRI data prior to COVID-19 infection. In addition, T2-signal hyperintensity tended to favour the interventricular septum, anterior and anterolateral walls, as well as basal inferior and mid-chamber. T1-mapping and ECV also demonstrated increased values, suggestive of myocardial fibrosis[75,81].

A developing use for CMRI is in the diagnosis suspected COVID-19-vaccine-associated myocarditis. These events tend to occur more frequently in young male patients after the second dose of mRNA vaccine[82,83]. Patients typically present with chest pain, troponin elevations, and abnormal CMRI findings[84]. CMRI abnormalities include myocardial oedema, hyperaemia and LGE, which are expected findings in acute myocarditis[85-89]. To date, there are no specific features for COVID-19-vaccine-associated myocarditis.

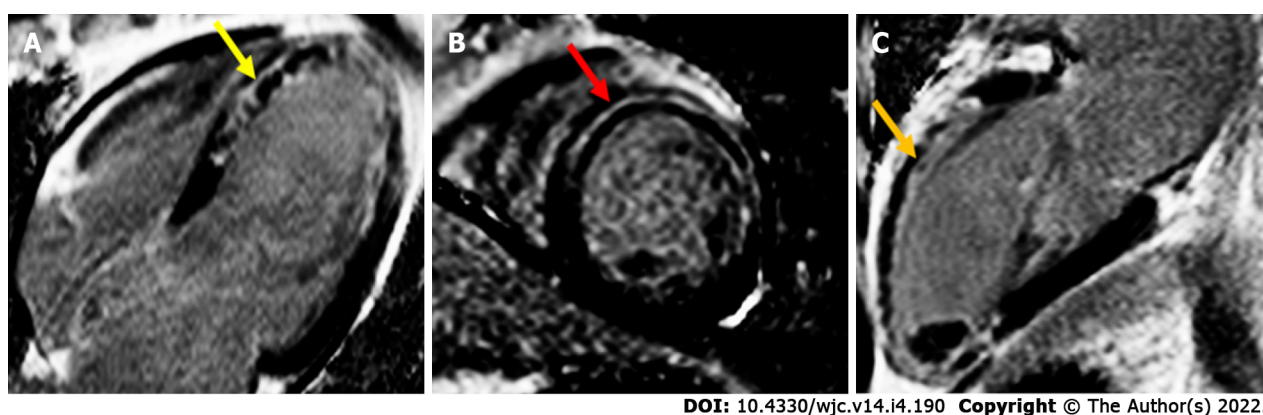


Figure 3 Cardiac magnetic resonance imaging of acute myocarditis. A: Four-chamber image demonstrating LGE in septal wall in a mid-wall pattern (yellow arrow); B: Short axis mid-left ventricular image demonstrating LGE in anteroseptal wall in a mid-wall pattern (red arrow); C: Two-chamber image demonstrating LGE in the anterior wall in a mid-wall pattern (orange yellow).

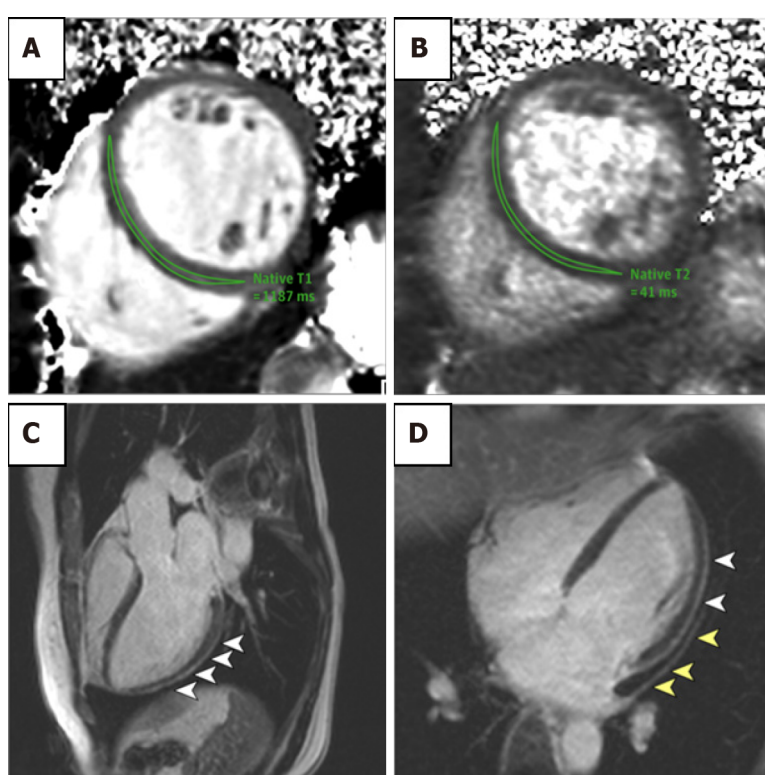


Figure 4 COVID-19 related cardiac dysfunction on cardiac magnetic resonance imaging. Cardiac magnetic resonance imaging of an adult woman with COVID-19-related perimyocarditis. A, B: Significantly raised native T1 and native T2 in myocardial mapping acquisitions; C, D: Pericardial effusion and enhancement (yellow arrowheads) and epicardial and intramyocardial enhancement (white arrowheads) using LGE acquisition. Modified from Puntmann *et al* [75] and licensed under CC BY 4.0 (<https://creativecommons.org/licenses/by/4.0/>).

Evidence for the outcome of COVID-19-induced myocardial injury continues to evolve. Studies have found that elevation in troponin T levels confer significantly increased risk of mortality [73,90,91]. However, whether this and other markers of myocardial injury are byproducts of disease severity, or directly contribute to morbidity and mortality, remains to be elucidated. Currently, there is no long-term data on COVID-19 effects on the cardiovascular system, but considering its global impact, the identification, monitoring and study of outcomes is critical, with CMRI likely to play an essential role [92,93].

HIGH-ENDURANCE ATHLETES AND AHS

Competitive sports level training can lead to a condition known as AHS, defined by complex cardiac

chamber remodelling, ventricular systolic impairment and abnormalities involving the electrical conduction system. Electrocardiogram changes can include first-degree atrioventricular block, incomplete right bundle branch block, early repolarisation and isolated increased QRS voltages that may meet criteria for LV hypertrophy (LVH)[94]. Transient troponin elevation occurs with moderate-to-high intensity exercise[95,96]. The role of CMRI is also continuing to evolve in helping distinguish AHS from conditions such as hypertrophic cardiomyopathy (HCM) and arrhythmogenic cardiomyopathy (ACM), which can have similar ECG and TTE features.

Although ventricular hypertrophy and dilatation can occur in both ventricles, impairment of systolic function following prolonged exercise tends to be observed more frequently in the right ventricle (Figure 5A). In early forms of the disease, diastolic dysfunction is often observed, first defined by a reduction in mitral E:A ratio on echocardiographic Doppler imaging[97-100]. A hallmark of AHS is increased LV mass[101]. Unfortunately, this is not a discriminating feature and can overlap with other conditions such as HCM and ACM[102]. In particular, differentiating AHS from mild HCM, with LV wall thickness range 13–15 mm, is critical in preventing adverse outcomes for athletes. Despite early reports suggesting that different cardiac conditions can lead to particular patterns of LVH, this has not been demonstrated in subsequent studies[103,104]. Cessation of training usually leads to LVH regression and improvement in clinical outcomes.

The advancement of CMRI technology may help shed light on the potential long-term effects of competitive level exercise and help differentiate different cardiac conditions. LV cavity size (LV end-diastolic and end-systolic diameter) in AHS is usually larger than HCM, particularly if the end-diastolic diameter exceeds 54 mm[100]. CMRI can also provide accurate morphology assessment for excessive trabeculation and noncompaction cardiomyopathy, if it cannot be clearly delineated on echocardiography[105]. The use of more advanced CMRI tissue characterisation techniques such as T1 mapping and ECV assessment is also helpful. Athletes have been demonstrated to have lower ECV, likely as a result of myocyte enlargement, compared to nonathletes. Conversely, in HCM, there is increased ECV [106,107]. The role of LGE to distinguish AHS compared to HCM is not yet certain. Domenech-Ximenes *et al*[106] found that focal LGE was more prevalent in intensive endurance athletes compared to healthy subjects (37.6% *vs* 2.8%), with a typical pattern at the right ventricular (RV) insertion points. This may overlap with the LGE distribution in HCM[107].

Ventricular dilatation has been noted in athletes compared to healthy nonathletic individuals[108, 109]. LV dilatation is less severe as compared to the RV dilatation. Stress echocardiography observation of improvement in LVEF by > 11%, and presence of mid-wall LGE may help to discriminate between AHS and pathological dilated cardiomyopathy[95,96,110,111] but this has not yet been investigated in large studies. In a meta-analysis performed by D'Ascenzi *et al*[102], in high-performance athletes, RV end-diastolic volume (EDV) and end-systolic volume (ESV) exhibited the greatest relative increase in ventricular remodelling, compared to baseline parameters. This may be in response to increased venous return and other hemodynamic changes. The increase in RV size in athletes has led to situations where the dimensions meet part of the ACM criteria[112]. CMRI can be useful to improve spatial resolution in cases with poor echocardiographic windows. It can quantify function, identify RWMA, and determine the presence of myocardial fibrosis and fibrofatty infiltration, to evaluate ACM versus AHS. In one study, Zaidi *et al*[113] found that the presence of RV ejection fraction < 45%, the ratio of RV EDV to LV EDV > 1.1/1, RV RWMA and LGE found together in athletes was highly indicative of ACM.

Exercise CMRI may provide additional diagnostic information by comparing the difference between adaptive responses and ventricular pathology in AHS, as well as potential prognostic information. The development of in-scanner CMRI exercise protocols with excellent reproducibility has been important in facilitating studies on the difference in physiological and pathological responses of athletes[114,115]. During exercise, elite athletes with evidence of ventricular arrhythmias had an increase in RV EDV, decrease in RV ESV, and, as a result, had reduced RV ejection fraction compared to athletes with no evidence of ventricular arrhythmia and healthy controls[116]. Of note, stress TTE yielded similar sensitivity in identifying exercise induced RV dysfunction.

The use of CMRI has been explored in AHS adverse outcome prognostication. In the context of excellent spatial and temporal resolution of CMRI, there appears to be no difference in resting cardiac volumes of elite athletes with and without evidence of ventricular arrhythmias[116]. LGE at the junction of the right ventricle and interventricular septum has previously been noted in athletes, but is not related to any clinical sequelae. A pattern of myocardial wall fibrosis in AHS has otherwise not consistently been demonstrated, or has been affected by confounding factors such as veteran athletes with coronary artery atherosclerotic plaques[112,117-119] (Figure 5B–5D). Zorzi *et al*[120] did note that in athletes who presented with a history of ventricular arrhythmias and subsequently found to have LV LGE, a striae subepicardial – midmyocardial lateral LV wall distribution pattern was more prevalent. There are no long-term data on outcome of incidental finding of myocardial LGE[114,121-123]. The definitive role of CMRI for prognostication in AHS remains to be defined outside of its roles in excluding other important diagnoses such as HCM and ARC.



Figure 5 Cardiac magnetic resonance imaging of athlete's heart syndrome. A: Cardiac magnetic resonance imaging of an endurance athlete. Increased right and left ventricular volumes. Overall muscle mass may be increased although wall thickness remains within standard reference range[102]; B: A 51-year-old athlete training 7 h/wk in the last 30 years. The short-axis view shows subepicardial late gadolinium enhancement (LGE) in the inferior apical wall; C: A 55-year-old athlete training 8 h/wk in the last 30 years. Mild intramyocardial LGE in the lateral wall is shown in the four-chamber view; D: A 55-year-old athlete training 10 h/wk in the last 28 years. Mesocardial LGE in the apical-septal wall shown in three-chamber view image. Reproduced from Pujadas *et al*[122] and licensed under CC BY 4.0 (<https://creativecommons.org/licenses/by/4.0/>).

COMMENTS AND LIMITATIONS

Traditionally, gross morphological cine scanning and determination of LGE location in the myocardial wall has formed key diagnostic features in the use of CMRI for many conditions. The advent of more advanced techniques such as T1 and T2 mapping has allowed a deeper understanding of the pathophysiological process involved.

The limitations for the use of CMRI include: (1) Access to CMR facilities with trained staff to perform the scan and process the images; (2) Standardised protocols; (3) Duration of procedure; (4) High cost; and (5) Lack of superiority to cheaper, faster and more accessible imaging modalities.

Moving forward, improving access to CMRI, increasing the number of skilled personnel and developing clear scanning guidelines are needed. Further research including large randomised trials are necessary to further define the role of CMRI in the assessment of MI, TTS, myocarditis, AHS and COVID-19-related cardiac conditions.

CONCLUSION

Troponinemia or an elevation in serum troponin levels can result from several different conditions making the diagnosis difficult. CMRI provides a powerful insight into the pathological mechanisms of disease, diagnostic features, as well as potential prognosis. With advancement in technology and research, this will only continue to improve.

FOOTNOTES

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