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Cardiac magnetic resonance imaging in heart failure: where the alphabet begins!

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Abstract Cardiac Magnetic Resonance Imaging has become a cornerstone in the evaluation of heart failure. It provides a comprehensive evaluation by answering all the pertinent clinical questions across the full pathological spectrum of heart failure. Nowadays, CMR is considered the gold standard in evaluation of ventricular volumes, wall motion and systolic function. Through its unique ability of tissue characterization, it provides incremental diagnostic and prognostic information and thus has emerged as a comprehensive imaging modality in heart failure. This review outlines the role of main conventional CMR sequences in the evaluation of heart failure and their impact in the management and prognosis.

Keywords Left ventricular dysfunction · Cardiomyopathy · Heart failure · Cardiovascular magnetic resonance · Late gadolinium enhancement · Myocardial delayed enhancement · Diagnosis · Prognosis

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Introduction

Cardiac Magnetic Resonance Imaging (CMR) has emerged as a comprehensive tool in the management of wide spectrum of cardiovascular diseases [1–3]. The high special resolution, the lack of ionizing radiation and ability to characterize biological tissue make CMR very attractive for initial diagnosis and follow up of many cardiovascular conditions across various age groups [4, 5]. The ability of CMR to provide valuable information about the diagnosis, etiology, current status and prognosis made it a versatile and pivotal part of the comprehensive evaluation of heart failure.

Heart failure (HF) is a clinical syndrome caused by inability of the heart to meet the physiological demands of the body organs resulting in either symptoms of volume overload or low cardiac output or both [6]. Although left ventricular ejection fraction (LVEF) is thought to be a sine qua non of heart failure, HF can present irrespective of LVEF. Hence, there are two types of HF; HF with reduced ejection fraction (HFrEF), LVEF ≤40%, and HF with preserved ejection fraction (HFpEF), LVEF ≥50% [7]. In the US, the annual cost of HF care exceeds 30 billion dollars and the mortality rate is around 50% within 5 years [7, 8]. The aging population and the advances in coronary care further contributed to the epidemic of HF adding to the financial burden of HF [9, 10]. Therefore, early detection and accurate diagnosis are essential in the care of patients with HF. Traditionally; 2-Dimisional echocardiogram (2D–Echo) is the first step in the diagnosis of HF. However, it is limited by geometric assumption, inter-observer variability and poor acoustic window in some cases [11, 12]. CMR overcomes most of the limitations of 2D–Echo and provides incremental information in relation to the etiology and prognosis of HF, thus answering the majority of pertinent clinical questions of HF. In this

review we will explore the role of CMR in the evaluation and management of HF from different etiologies.

CMR Protocols & Safety

Most of the contemporary magnetic resonance imaging (MRI) scanners are capable of performing the main cardiac protocols. A CMR study for HF takes around 30–45 min and typically starts with ECG-gated cine (steady-state free precession (SSFP)) imaging to assess ventricular morphology, size and systolic function followed by images to assess myocardial edema (T2-weighted images) as well as the pericardium [13, 14]. Gadolinium is an extracellular contrast agent that does not cross intact cell membrane. Acute and chronic myocardial infection (MI) increases the volume of distribution due to cellular damage and extracellular fibrosis respectively [15, 16] resulting in delayed washout of the contrast in areas with myocardial damage, thus, differentiating normal and abnormal myocardium. The abnormal myocardium appears white, hyper-enhanced, while normal myocardium appears black because gadolinium has completely washed out from normal myocardium at the time of image acquisition (8–10 min after gadolinium administration) [17]. Hence the term, myocardial delayed enhancement (MDE) or late gadolinium enhancement (LGE) images. The pattern and distribution of MDE provide diagnostic and prognostic information in HF (Fig. 1). Several sequences are obtained after administration of intravenous gadolinium-based contrast agent (GBCA). GBCA could be used for stress and rest myocardial perfusion imaging (MPI) [13, 18]. It is also used for myocardial tissue characterization through delayed myocardial enhancement images [19]. Additionally, flow quantification sequences are used to evaluate and presence and the severity of valvular regurgitation as well as other flow abnormalities [20, 21]. (Table 1).

CMR can be safely performed in the majority of cardiac patients. It does not employ ionizing radiation limiting the concerns about radiation injury. Nowadays most of the modern cardiac devices are MRI conditional or compatible allowing the performance of CMR with some precautions [22]. All the coronary stents and prosthetic valves are generally safe.

However, there are some limitations to CMR in heart failure patients. Many heart failure patients have concomitant renal failure and GBCA should not be used in patients with severe renal dysfunction (glomerular filtration rate < 30 ml/min/1.73 m^2) or heamodialysis due to risk of Nephrogenic Systemic Fibrosis (NSF). NSF is a serious and non-curable condition characterized by fibrosis of the skin and internal organs following exposure to gadolinium [23]. Additionally, claustrophobia is a common cause of cancelation of CMR studies [24]. This can be sometimes overcome with administration of small doses of anxiolytic medications. In addition, many advanced heart failure patients have devices that are not compatible to CMR imaging.

Early detection of heart failure

The current definition of HF identifies patient who already have clinical symptoms and therefore, may lack the ability to identify subclinical and asymptomatic patients or those with early stages of HF. Identifying early stages of HF is of paramount importance. It helps in early initiation of medical therapy of asymptomatic patient and controlling risk factors of patients at risk of HF. Further to its superiority in assessment of LVEF and LV mass, CMR is the only cardiac imaging modality capable of precisely recognizing the presence and the extent of prior myocardial infarction irrespective of its size [25, 26]. Identifying a prior infarct necessitate initiation of medication according to the ACC/AHA guidelines [7].

Quantification of ventricular size and systolic function

CMR is the gold standard modality for assessment of ventricular volumes and systolic function [27, 28]. Accurate quantification of LVEF is essential in the classification and management decisions of HF. [7]. CMR has the edge over other imaging modalities in assessment of LVEF and wall motion abnormalities owing its high spatial resolution, good temporal resolution, unrestricted view of the heart and high reproducibility [3, 29, 30]. CMR calculate the ejection fraction (EF) by true volumetric evaluation without geometric assumption, resulting in accurate EF calculation irrespective of the degree of LV remodeling or systolic dysfunction. The inter and intra observer reproducibility of LVEF by CMR is high [28, 31]. Equivalently, CMR can assess the right ventricular EF despite its complex 3D–dimensional and highly variable shape [29, 32].

In addition to their role in the diagnosis of HF, both LV dilatation and LVEF have prognostic value. LVEF is a strong and independent predictor of arrhythmic death in heart failure and it dictates the need for device therapy for primary prevention [7, 33]. In addition, LV dilatation is also a marker of poor prognosis [34]. CMR can also provide excellent assessment of RVEF which is an independent prognostic value in patients with dilated cardiomyopathy [35, 36] and in patients with myocardial infarction irrespective of LVEF and infarct size [37].

Etiology of heart failure

Etiology of HF is fundamental for the management and prognosis. While some of HF cases can be completely treated by

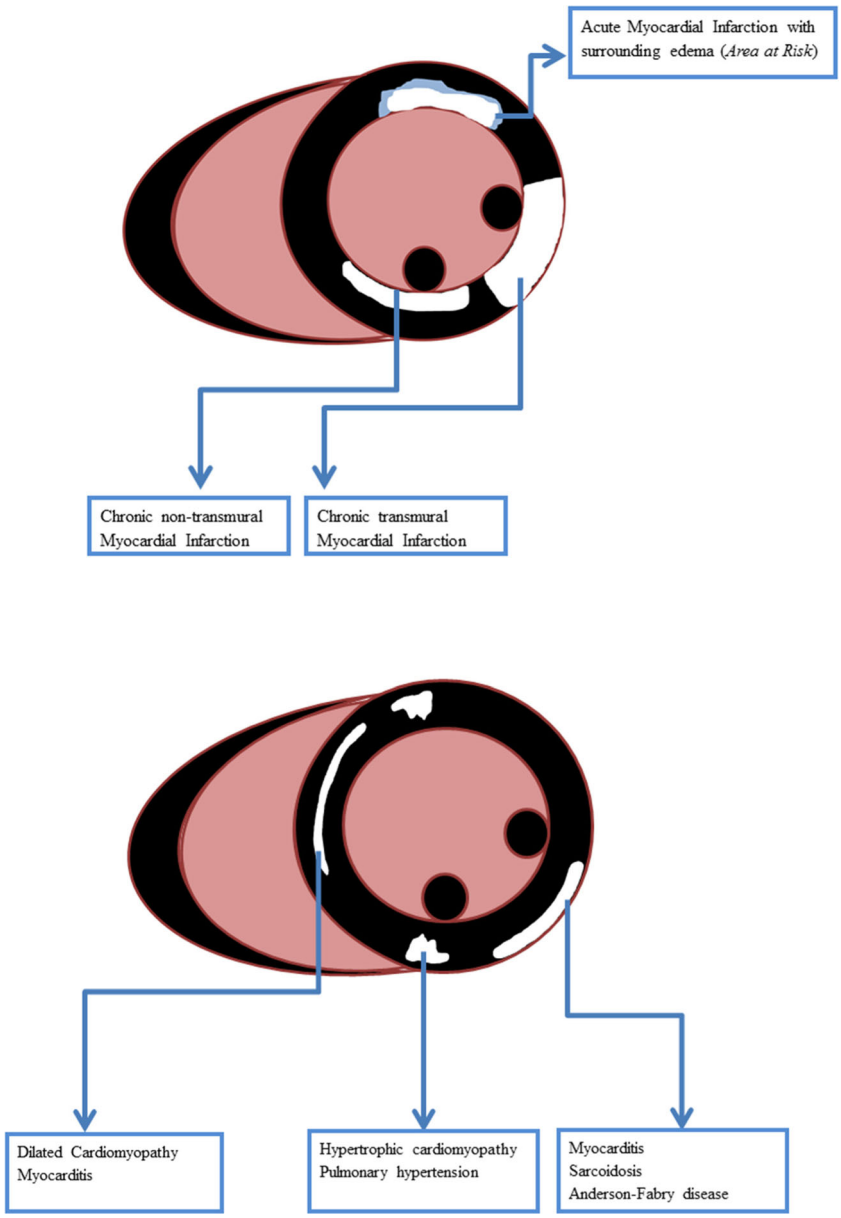

Fig. 1 Distribution and pattern of delayed myocardial enhancement in different types of cardiomyopathy

controlling the underlying condition such as inflammation in cardiac sarcoidosis and enzyme replacement Anderson-Fabry disease [38, 39], knowing other causes such as coronary artery disease (CAD) is essential for the choice of therapy and initiation of secondary prevention measures [6, 7].

CAD remains the most common cause of HF and it carries the worse prognosis [40–42]. Therefore, CAD has to be ruled out in any case with new diagnosis of HF. Traditionally, CAD is evaluated with invasive coronary angiography (ICA). However, due to its invasive nature, ICA should be avoided if possible particularly in cases of low likelihood of CAD. Since its inception, CMR provided a reliable method for assessing CAD in patients with HF through myocardial tissue characterization.

Heart failure due to ischemic heart disease

In cases of ischemic heart disease, the DME is typically located in the subendocardail area corresponding to a coronary artery territory and may extend to the epicardial area depending on whether the infarct is transmural or subendocardail [25, 43] (Fig. 2). The presence of MDE can differentiate between infracted and stunned myocardium in the presence of wall motion abnormalities. Furthermore, it predicts the likelihood of recovery of myocardial segments after revascularization. There is an inverse relation between the extent of the MDE and recovery of the contractile function after revascularization [44]. The larger the extent of the MDE within a myocardial segment, the less likely it is viable [45, 46].

Table 1 Basic CMR protocols performed for evaluation of heart failure

CMR sequence Technical terminology	CMR sequence Simplified terminology	Provided Information
Steady-state free precession (SSFP) Axial, Cardiac Short and long axis views	Cine CMR images	Evaluation of ventricular size, morphology and systolic function. Evaluation of wall motion abnormalities
T2-weighted images Double-inversion recovery/triple-inversion	Edema CMR images	Evaluation of myocarditis and acute ischemic injury
In plane/through-plane motion-encoded phase-sensitive spoiled gradient echo	Flow CMR	Quantification of valvular regurgitation Quantification of shunt (Qp;Qs)
T2*-weighted spoiled gradient echo sequence	Iron CMR	Evaluation of cardiac iron overload Assessment of intra-myocardial hemorrhage in acute myocardial infarction
First Pass Perfusion Images (with & without stressor)	Perfusion CMR	Ischemia evaluation
Inversion-recovery gradient echo sequence Delayed (hyper) enhancement sequence	Late gadolinium enhancement (LGE) CMR	
Early Delayed Enhancement Images (3 min after contrast administration)		Evaluation of the possible thrombus
Delayed Enhancement Images (8–10 min after contrast administration)		Evaluation of viability, presence and type of scar (Ischemic Vs non-ischemic myocardial injury)

CMR is highly sensitive in detecting MI, 99% and 94% in acute and chronic infractions respectively [47]. Additionally, CMR can evaluate the presence of CAD through ischemia evaluation by vasodilator MPI with sensitivity and specificity of 91% and 83% respectively [48]. Alternatively, dobutamine stress CMR can evaluate the presence of ischemia and improves the detection of contractile reserve in segments with intermediate MDE [49]. On the other hand, 15% of patient with HF due to CAD has no or non-ischemic DME on CMR, this is thought to be due to coronary collaterals or myocardial hibernation without infarct [50, 51]. Accordingly, MDE-CMR can be reasonably used alone or with CMR ischemia evaluation to rule out CAD in HF particularly in cases with low-intermediate likelihood of CAD. In fact, about 13% of patients with HF and normal ICA, have evidence of ischemic MDE on CMR that may be related to recanalization of the culprit artery or embolic phenomenon [52]. Therefore, CMR provides incremental information about CAD that may help guide further therapy even in patients with normal ICA.

Edema CMR images provide additional information to MDE. It helps differentiate acute from chronic MI, detect ischemic injury without infarction and identify peri-infarct area, area at risk, which represents salvageable myocardium [53–56]. Thus, it accurately differentiates areas of reversible and irreversible damage. Both MDE and edema images provide incremental prognostic information. While, the peri-infarct zone and infract heterogeneity are associated with higher risk of future arrhythmia [57, 58], MDE has 6-fold increase in adverse cardiac outcomes and 11-fold increase of all-cause mortality [59, 60]. Microvascular obstruction (MVO) in the setting of acute MI has shown to be a strong and independent predictor of LV remodeling irrespective of infarct size [61]. It is also associated with increased cardiac death, nonfatal MI and ischemic stroke [62, 63]. MVO can be easily identified in the DME images, where it appears as dark areas within the hyper-enhanced infracted myocardium (Fig. 3). Moreover, CMR is superior to other imaging modalities in detecting complications of CAD that may contribute to HF morbidities, including aneurysm formation and intramural thrombi [64–67]. There upon, CMR can evaluate all the pathological processes involved in the development and progression of HF due to CAD.

Heart failure due to non-ischemic etiologies

Dilated cardiomyopathy

Dilated Cardiomyopathy (DCM) is a common phenotypic end-result of many pathophysiological processes. It can be reversible or irreversible and albeit mostly idiopathic, about half of the cases of DCM are familial type [68, 69]. The diagnosis is usually made after excluding CAD [68]. However, ICA is invasive and it may miss ischemic component of HF in approximately 13% of cases [52], CMR may provide an alternate option for the evaluation of CAD in DCM particularly in patients with low likelihood of CAD. With the current advances in CT imaging, CT angiography is a more commonly used test in this population.

As discussed above, Ischemic MDE has high sensitivity in detecting obstructive CAD in HF [70]. On the other hand, the majority of cases with DCM (59%) have no MDE on CMR, and around 28% have the classical mid wall longitudinal striae

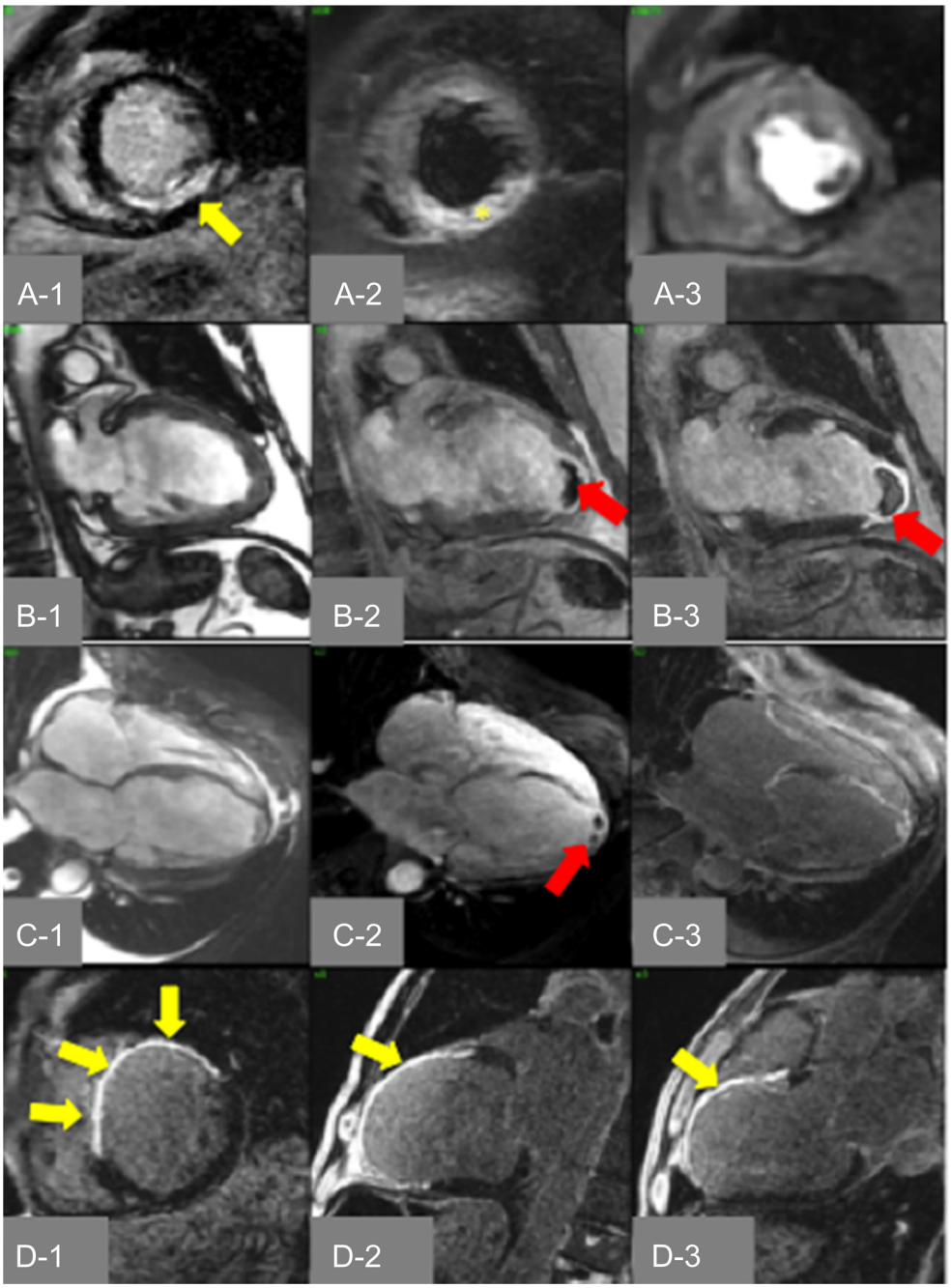

Fig. 2 Role of CMR in evaluation of ischemic heart disease. Panel A demonstrates acute myocardial infection showing transmural enhancement (yellow arrows) with myocardial edema (asterix) (A-1: Myocardial delayed enhancement image, A-2: CM edema image and A-3: rest perfusion image). Panels B and C: demonstrate the role of CMR in evaluation of thrombi (red arrows) complicating myocardial infarction (B-1 and C-1: SSFP images of the LV, B-2 and C-2: early delayed enhancement images and B-3 and C-3: Myocardial delayed enhancement images. Panel D shows a transmural myocardial delayed encashment (yellow arrows) in the left anterior descending coronary artery

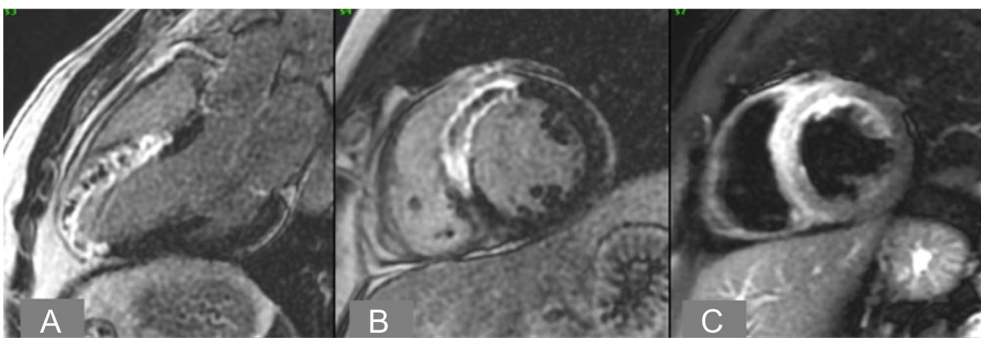

Fig. 3 Myocardial delayed enhancement (A and B) and CME edema image (C) showing acute anteroseptal myocardial infarction with microvascular obstruction/ hemorrhage (black areas within the delayed enhancement)

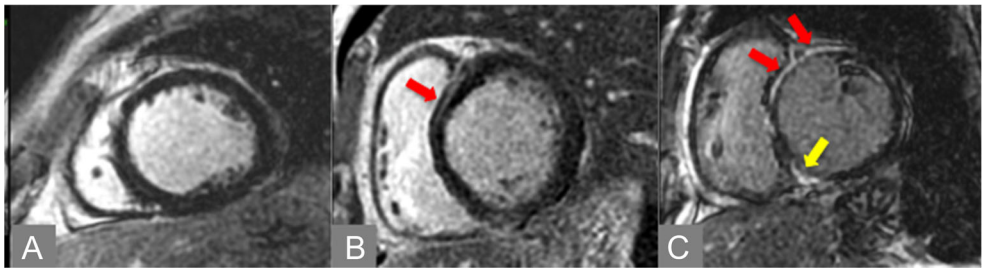

Fig. 4 Patterns of myocardial delayed enhancement in dilated cardiomyopathy. Absence of MDE (A), classical midwall MDE (B and C): red arrows point to the non-ischemic injury while yellow arrow points to small ischemic myocardial injury indicating concomitant coronary artery disease with dilated cardiomyopathy

(Fig. 4) or patchy DME that does not correspond to a particular coronary artery territory [52]. On the other hand subepicardial MDE denotes previous myocarditis [71, 72]. An additional value of CMR is its ability to accurately evaluate the size and systolic function of the RV, which is commonly involved in this cardiomyopathy, further aiding the diagnosis of non-ischemic DCM.

MDE also aids in the prognostic stratification of DCM. It presence is associated with increased risk of all-cause mortality (3-folds increase), sudden cardiac death (5-fold increase) and adverse cardiac events independent of LV volumes and LVEF [73–79].

Myocarditis

Acute myocarditis can present as new heart failure. Around 9% of DCM are thought to be a chronic sequelae of myocarditis [68]. The diagnosis of myocarditis is usually made after excluding CAD and confirmed by endomyocardial biopsy. However, this approach is not attractive because it is invasive and associated with sampling error due to patchy nature of acute myocarditis. CMR has emerged as a robust tool for the diagnosis of acute myocarditis. The non-ischemic MDE in myocarditis is typically subepicardial and more commonly affecting the lateral and inferolateral walls (Fig. 5). It can also be seen as mid-wall distribution similar to that classically seen in DCM [71, 80, 81]. It should be kept in mind that absence of MDE does not exclude the diagnosis of myocarditis. Some cases do not have cellular damage and hence will not have MDE, in which case the diagnosis is made based on the presence of myocardial edema [72]. In addition, MDE usually disappears in cases of healed myocarditis [81]. CMR diagnosis of myocarditis is based on Lake Louise Criteria, which takes in account all the pathophysiological processes in acute myocarditis. Diagnosis of acute myocarditis is made when there are two out of three criteria, edema by T2-weighted images, hyperemia by early T1-weighted images and the presence of non-ischemic MDE [82]. Other newer tools used in the diagnosis of myocarditis including T1 and T2 mapping will be discussed in a separate article in this special issue.

LV non-compaction cardiomyopathy

Isolated LV non-compaction (LVNC) is a rare cause of heart failure. The hallmark of the diagnosis of LVNC is the presence of prominent LV trabeculations with deep inter-trabecular recesses, hence, the classical appearance of two layers, the compacted and noncompacted myocardium. [83]. The diagnosis is usually made on echocardiography when the maximum ratio of noncompacted to compacted myocardium >2:1 at end-systole and the presence of color Doppler flow in the deep inter-trabecular recesses [84]. However, echocardiography can miss or over-diagnose cases particularly in African-Americans and athletes [85, 86]. CMR improves the diagnostic accuracy of LVNC through quantitative diagnostic criteria. There are three proposed criteria for the diagnosis of LVNC on CMR, either, maximum end-diastolic noncompacted to compacted myocardial thickness ratio of >2.3 (sensitivity of 86% and specificity of 99%), trabeculated LV mass > 20% of total LV mass (sensitivity of 94% and a specificity of 94%) or end-systolic noncompacted to compacted ratio ≥ 2.0 (Fig. 6) [87–89]. The latter is more strongly associated with heart failure, death, arrhythmia and embolic phenomena [89]. Additionally, CMR is superior to echocardiography in detecting small thrombi within the trabeculations. It is worth noting that since LVNC can share some phenotypic features of other cardiomyopathies; the diagnosis of LVNC should not be solely based on the compacted\noncompacted ratio [90].

Cardiac sarcoidosis

Cardiac involvement with sarcoidosis is heterogeneous. It can be asymptomatic or present with heart failure, heart block or malignant arrhythmia [91, 92]. It is seen in 25% of patients with sarcoidosis and 25–75% of sarcoidosis mortality is due to HF [93–95]. CMR can accurately assess the pathological spectrum of sarcoidosis from active inflammation to myocardial fibrosis and subsequent HF. Ventricular size, wall motion and systolic function as well as the pericardium can be accurately assessed by CMR. In addition DME can aid the diagnosis and prognosis of

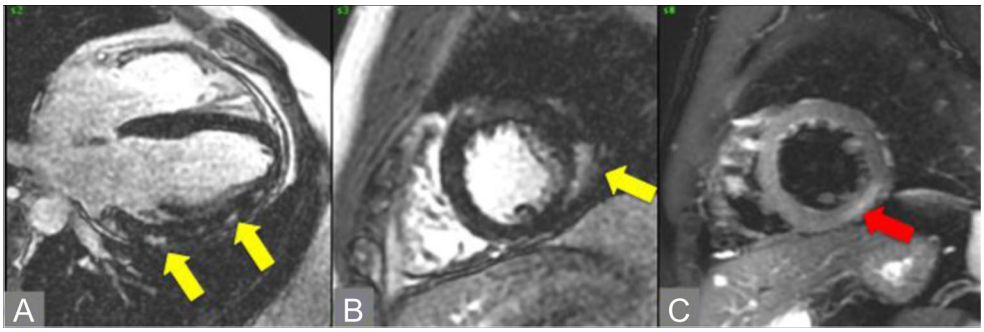

Fig. 5 Myocardial delayed enhancement images (A and B) and CMR edema image (C) showing the classical subepicardial myocardial delayed enhancement (yellow arrows) and myocardial edema (red arrow)

patients with cardiac sarcoidosis. In fact, CMR is currently part of the Japanese Ministry of Health criteria for the diagnosis of cardiac sarcoidosis [96]. Active inflammation can be detected with edema images while areas of myocardial fibrosis can be easily identified with DME images [97, 98]. The MDE distribution in cardiac sarcoidosis is variable and can affect any part of the myocardium of left and right ventricles or even the papillary muscles [92, 99]. It is typically patchy and non-ischemic affecting the midwall of the basal anteroseptal and inferolateral walls [92, 99, 100]. The presence of MDE in sarcoidosis is associated with poor outcomes. It is associated with higher risk of sudden cardiac death (10-fold increase) and adverse events including appropriate ICD discharge and bradycardia requiring pacemaker insertion. CMR can monitor disease activity and response to therapy through edema and MDE images [101, 102]. Furthermore, MDE in the right ventricle has also been shown to predictor of arrhythmia independent of the LVEF [103].

Stress induced (tako-tsubo) cardiomyopathy

Stress induced cardiomyopathy is a reversible condition characterized by a transient wall motion abnormalities and LV systolic dysfunction [104, 105]. It is diagnosed after excluding CAD since it usually mimics acute myocardial infarction. Multiple types have been reported based on the distribution of wall motion abnormalities including apical (typical presentation), mid-ventricular, basal and focal types [106, 107]. CMR readily and accurately assess the extension of the regional wall motion abnormalities in each type and can identify possible thrombi within the dysfunctional areas that can be easily missed by echocardiography [108]. Moreover, MDE can differentiate stress cardiomyopathy, which classically has no delayed enhancement, from myocardial infarction and myocarditis that have typical MDE distributions [109, 110]. CMR diagnosis of stress cardiomyopathy is based on typical pattern of LV dysfunction (apical akinesia and ballooning), myocardial edema and the absence of MDE [110]

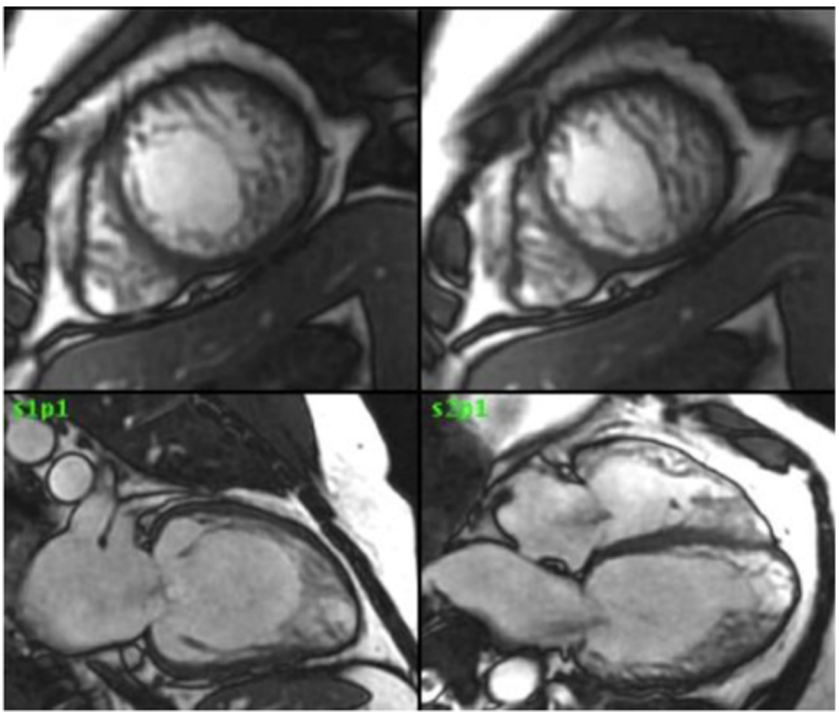

Fig. 6 SSFP images showing increased LV trabeculation in LV non-compaction cardiomyopathy

(Fig. 7). In addition, the resolution of the cardiomyopathy within few days/weeks confirms the diagnosis.

Heart failure due to valvular heart disease

Untreated valvular heart diseases can lead to heart failure. LV dilatation and systolic dysfunction are among the indications of valve intervention in asymptomatic patients [111]. ACC/AHA guidelines recommend the use of CMR for evaluation of ventricular size and systolic function particularly if the echocardiography images are suboptimal. Moreover, CMR provides accurate quantitative assessment of valvular regurgitation by direct measurement regurgitant volume and calculation of the regurgitant fraction [112–115] (Fig. 8). In cases of mitral regurgitation, CMR adds to the management by providing insight about the etiology through evaluation the morphology of the LV and papillary muscle, wall motion abnormalities and detection of the CAD by MDE [116]. Valvular stenosis parameters that can be evaluated by CMR include measurement of valve area by planimetry, peak antegrade velocity and pressure gradients. [117–119]. In addition, MDE is seen in patients with severe aortic stenosis and has demonstrated to be an independent predictor of mortality post valve replacement [120, 121].

Heart failure with preserved ejection fraction

Hypertrophic cardiomyopathy

Hypertrophic Cardiomyopathy (HCM) is an inherited disease with variable phenotypes characterized by asymmetrical hypertrophy of the LV [122]. Owing its superior spatial resolution, CMR precisely assess the location and extent of the hypertrophy particularly the apical form that can be easily missed with echocardiography [123–125]. It can also identify the presence of apical aneurysm that can complicate apical form and associated with embolic complications [126]. The MDE in HCM has diverse pattern and distribution (Figs. 9 and 10). It is most frequently seen as patchy midwall at the RV insertion points and in areas of hypertrophy but it has also been observed in normal segments [125, 127]. Prognostic information can be provided by assessment of anterior systolic motion of the mitral valve, a marker of left ventricular outflow obstruction [128, 129], and the presence of MDE. MDE is an independent predictor of sudden cardiac death and ventricular arrhythmia, while absence of MDE incurs a very low risk [130, 131]. Anderson-Fabry disease (AFD) is a condition commonly mistaken with HCM [132, 133]. Differentiation between the two cases is crucial since AFD is a treatable and responds well to enzyme replacement therapy [134]. CMR can help ascertain the diagnosis since the MDE in AFD is distinctly seen in the epicardium of the basal and mid segments of the anterolateral and inferolateral walls [135, 136].

Amyliodosis

Cardiac amyloidosis (CA) can be the first manifestation of systemic amyloidosis and it carries grave prognosis [137–139]. When HF ensues, untreated patients die within 6 months; therefore early recognition is of paramount importance [3]. CMR readily detect the morphological abnormalities including ventricular hypertrophy, thickening of the atrial septum and valves, atrial dilatation and pericardial effusion. MDE in cardiac amyloidosis is not constant; it can be

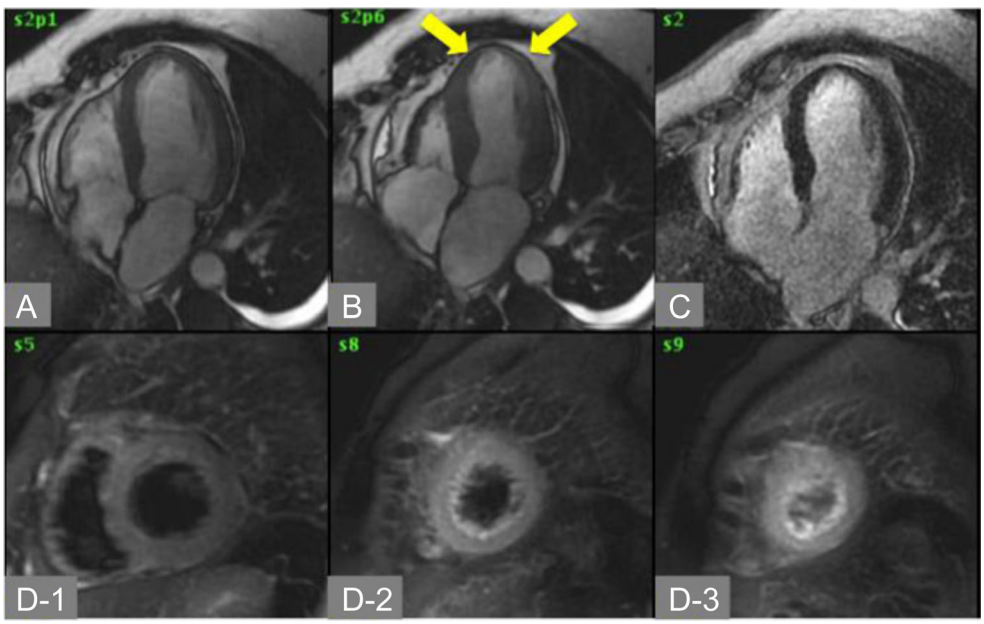

Fig. 7 CMR images demonstrating the findings in Tako-Tsubo cardiomyopathy. A and B: SSFP images in diastole (A) and systole (B) demonstrating the apical wall motion abnormalities. C: myocardial delayed enhancement images showing absence of MDE. D1–3: CMR edema images showing the edema of the apical segments of the LV

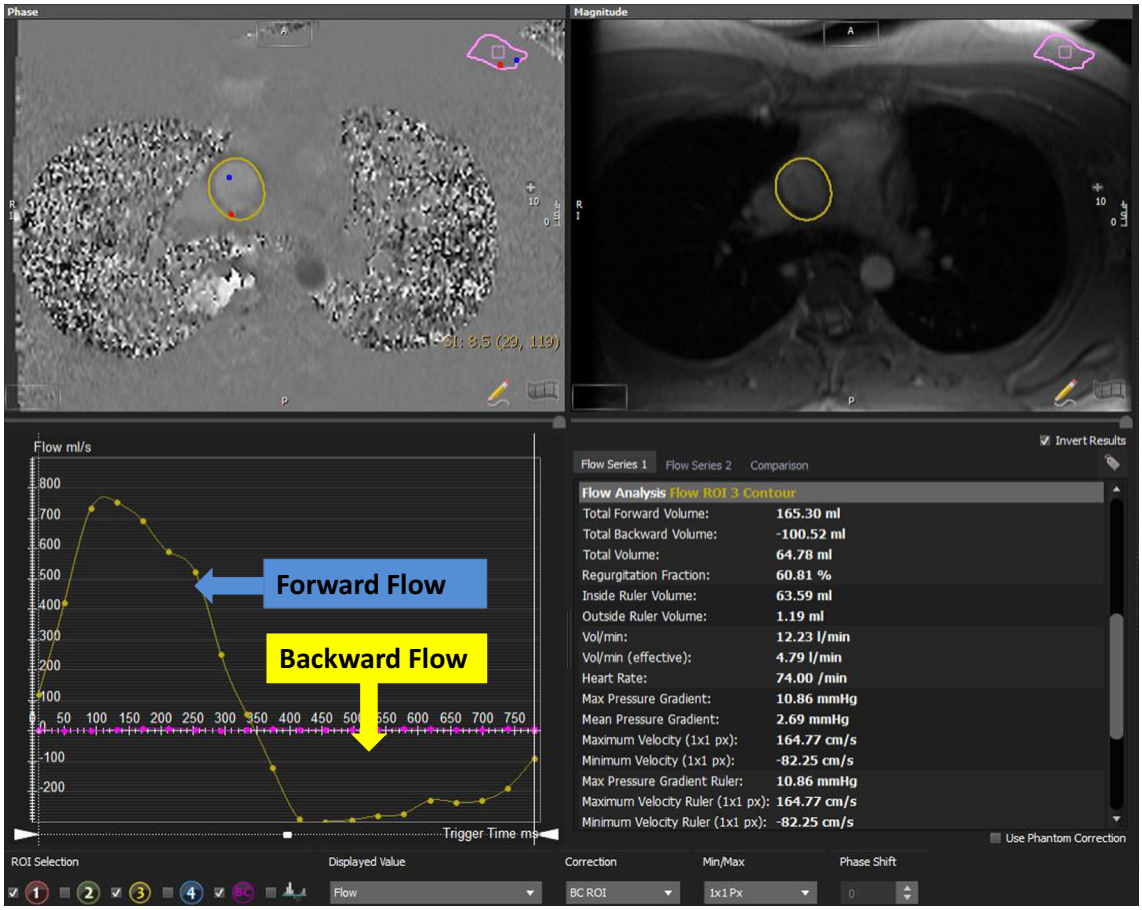

Fig. 8 Flow CMR of the aortic valve demonstrating the forward and backward flow across the aortic valve in a case of severe aortic regurgitation

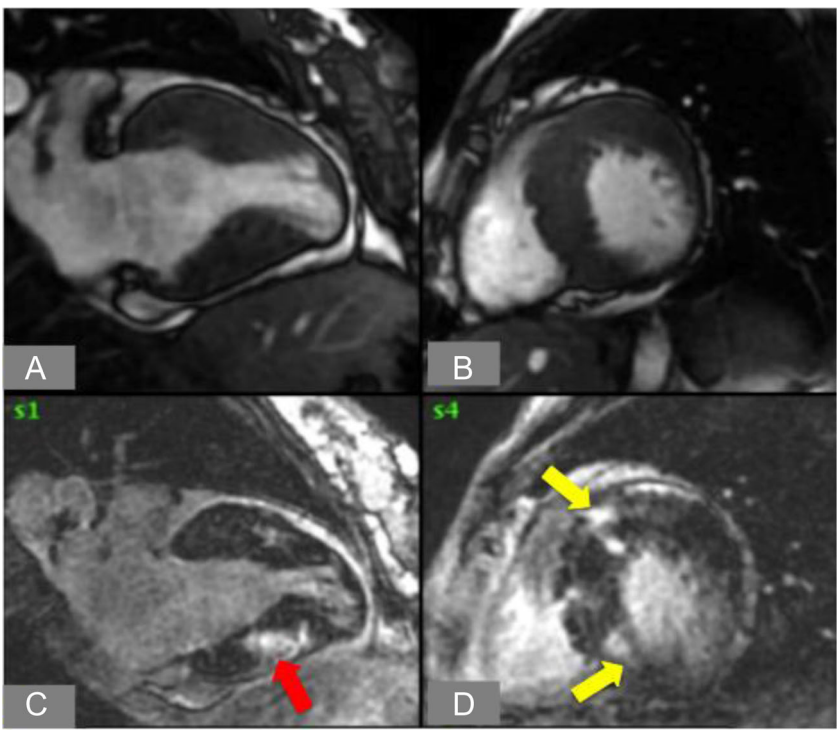

Fig. 9 SSFP (A,B) and myocardial delayed enhancement (C, D) images showing the asymmetrical hypertrophy of the septum (hypertrophic cardiomyopathy) and the classical delayed enhancement in the RV insertion points (yellow arrows) as well as delayed enhancement in the hypertrophic areas (red arrow)

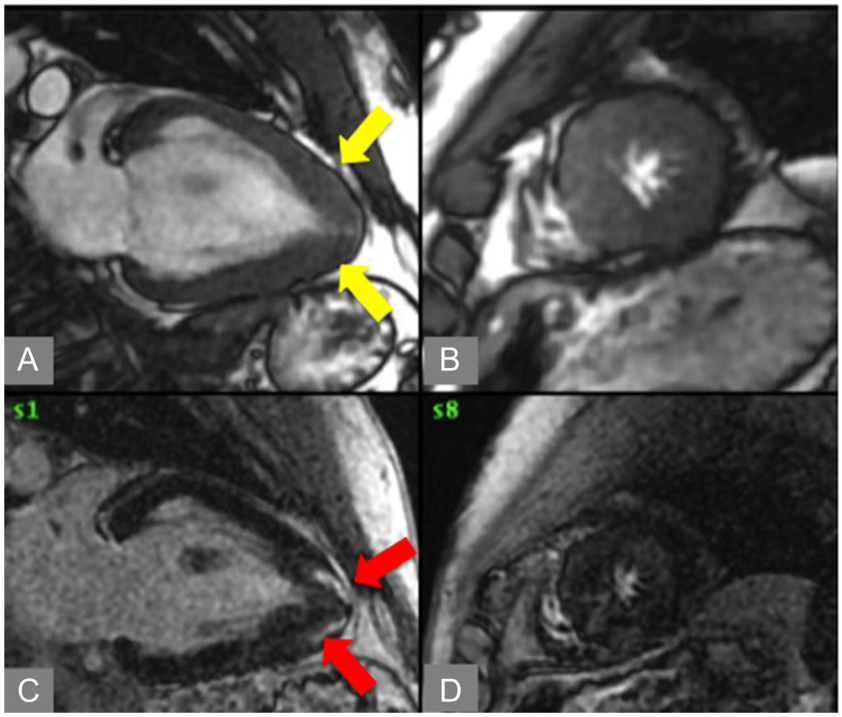

Fig. 10 SSFP (A,B) and myocardial delayed enhancement (C, D) images showing the apical hypertrophy (apical HCM) showing hypertrophy of the LV apex (yellow arrows) and delayed enhancement in the apex (red arrows)

circumferential subendocardial enhancement of the LV, a zebra-stripe appearance with subendocardial enhancement of the LV and RV or patchy transmural appearance [137, 140, 141]. Rapid exchange of gadolinium between blood pool and amyloid fibrils within the myocardium precludes assessment of DME in some cases. Recently, MDE has been

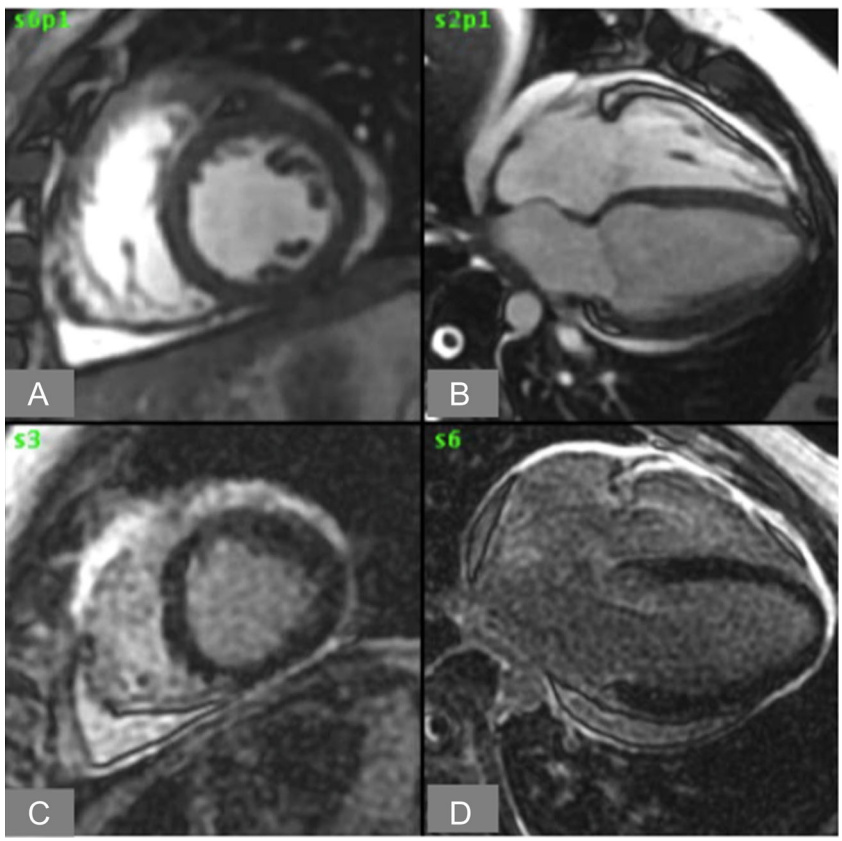

Fig. 11 SSFP (A,B) and myocardial delayed enhancement (C, D) images showing pericardial thickening as well as delayed enhancement in a case of acute pericarditis

observed in the left atrium and is used to improve diagnosis of CA on CMR [142]. MDE of the LV correlates well with HF severity and survival in CA [143, 144].

Constrictive pericarditis

CMR can help in the diagnosis of constrictive pericarditis (CP) by demonstrating the thickening of the pericardium and the other morphological changes such as dilated atria and small tubular ventricles and inter-ventricular dependence (septal bounce on cine images) [145, 146]. Pericardial thickness > 4 mm highly suggests constriction although 18% of surgically proven constriction have normal pericardial thickness. [147]. Pericardial inflammation can be assessed by edema and delayed enhancement images, which can also be used to monitor therapy [145, 148] (Fig. 11). Thus, CMR is very useful in differentiating CP from restrictive cardiomyopathy (93% diagnostic accuracy) [147].

Arrhythmogenic right ventricular cardiomyopathy

CMR is fundamental in the diagnosis of Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC). The diagnosis requires accurate evaluation of RV volume, systolic function and wall motion, which can readily be evaluated by CMR. According to the modified task force criteria for the diagnosis of the ARVC, regional RV akinesia or dyskinesia and either ratio of RV end-diastolic volume to BSA 110 mL/m2 (male) or 100 mL/m^2 (female), or RV ejection fraction 40% constitutes a major criterion, while regional RV akinesia or dyskinesia and one of the following: whether ratio of RV end-diastolic volume to BSA 100 to 110 mL/m^2 (male) or 90 to 100 mL/m^2 (female) or RV ejection fraction >40% to ≤45% is considered a minor criterion [149]. MDE has low diagnostic accuracy for diagnosing ARVC [150], however it correlates well to fibrofatty infiltration of the RV and inducible arrhythmia [151].

Advanced heart failure

LVEF is pivotal for decision of device therapy in advanced HF [7]. CMR provides accurate assessment of LVEF and can predict response to cardiac resynchronization therapy (CRT). Large scar burden and MDE in septum or posterolateral wall are associated with limited response to CRT [152–155]. Moreover, CMR is promising in the detection of CAD and rejection post cardiac transplant as well as monitoring response to stem-cell therapy in advance HF [156, 157].

In conclusion, CMR has emerged as a robust and pivotal tool in the comprehensive evaluation of HF. It can answer all the clinical questions across the entire HF pathological spectrum. Although CMR requires sophisticated software, most of the modern MRI scanners have the ability to perform basic cardiac sequences that can accurately evaluate cardiac anatomy, function and wall motion. In addition, CMR distinct ability to characterize myocardial tissue provides indispensable diagnostic and prognostic information incremental to other imaging modalities. CMR has a real potential of being a one-stop shop in evaluation of HF and should be considered in the comprehensive evaluation of all new and established cases of heart failure.

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