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Application of Cardiac Magnetic Resonance Imaging in Cardiomyopathy

Kevin E. Steel, DO and Raymond Y. Kwong, MD, MPH

Abstract

Cardiomyopathies account for a significant portion of morbidity and mortality in patients with heart disease. The diagnosis and identification of the underlying disorder are essential for directing appropriate life-saving therapy. Cardiac magnetic resonance imaging (CMR) is an ideal method for the noninvasive evaluation of cardiomyopathies of unknown etiology. In addition, there is increasing prognostic evidence to support the use of this technology in patient risk stratification. CMR is not limited by anatomic barriers and is able to characterize tissue abnormalities that previously could often be identified only through biopsy. This review discusses the utility of CMR in the assessment of cardiomyopathies, including specific imaging techniques and their application in ischemic and nonischemic settings.

Introduction

Cardiomyopathies are characterized by progressive ventricular dysfunction due to toxins, ischemia, cellular infiltration, infection, and other causes [1]. The diagnosis and identification of the underlying disorder are essential for directing appropriate therapy, which could significantly alter morbidity and mortality. With a capability for tissue characterization, cardiovascular magnetic resonance imaging (CMR) been shown to accurately distinguish ischemic from nonischemic causes of ventricular dysfunction and to differentiate types of cardiomyopathy with noncoronary etiologies, including dilated cardiomyopathy (DCM), hypertrophic cardiomyopathy (HCM), infiltrative cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy (ARVC), and myocarditis [2,3,4•,5•,6]. The American College of Cardiology and American College of Radiology have identified CMR as an appropriate tool for evaluating ventricular dysfunction of unknown cause. With technical advances and increasing clinician awareness and training, the indications for CMR will continue to expand [7•].

Imaging protocols have been published that describe, in a stepwise manner, the sequences that are used in the complete evaluation of cardiomyopathy and can be tailored to answer the specific question at hand [8•]. This review discusses the basic CMR approaches to the various cardiomyopathies from ischemic and nonischemic causes.

CMR Techniques

Cardiomyopathies are typified by alterations in chamber size and function as well as changes at the cellular level. Steady state free precession (SSFP) gradient-echo pulse

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Corresponding author: Raymond Y. Kwong, MD, MPH, Cardiovascular Division, Department of Medicine, Brigham and Women's Hospital, 75 Francis Street, Boston, MA 02115, USA. rykwong@partners.org.

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sequences are currently the most commonly used CMR techniques to obtain segmental motion images of the heart during breath holds. Chamber sizes, mass, and function can be quantified at high accuracy with the aid of postprocessing software [9]. With a breath-holding duration generally of 10 to 15 seconds, high-quality cine images can be achieved at a temporal resolution of 25 to 50 msec and in-plane spatial resolution of 1 to 3 mm. With the application of higher gradients (eg, 3 tesla), temporal or spatial resolution may further improve, given a higher margin of signal–noise ratio.

The ability to differentiate normal from abnormal tissue and to identify the underlying abnormality of diseased tissue makes CMR a powerful noninvasive tool in the diagnostic evaluation of cardiomyopathy. Pulse sequences that demonstrate edema, inflammation, infarct, and tissue iron content are typically used in the setting of ventricular dysfunction of unknown origin. Chelated gadolinium agents are used as contrast for perfusion analysis and late enhancement imaging (late gadolinium enhancement [LGE]). Detailed imaging protocols have been published to standardize the CMR approach to assessing cardiomyopathy and can be used as a guideline for less experienced imaging centers [8•]. Table 1 and Table 2 summarize the common sequences used in the evaluation of cardiomyopathy and their application in the evaluation of the specific types of cardiomyopathy that are discussed in this paper.

Cardiomyopathies

Ischemic cardiomyopathy

The initial role of CMR in the evaluation of cardiomyopathy was to differentiate ischemic from nonischemic causes of ventricular dysfunction. Compared with echocardiography, cine CMR is volumetric in its quantitation of left and right ventricular function and sizes. LGE sequences using chelated gadolinium provide a highly reliable quantitation of the presence and the extent of infarcted myocardial tissue. Previous studies have shown that neither ECG Q waves nor a history of clinical infarction is as accurate as evidence of infarcted myocardium characterized by CMR [10]. Furthermore, our group has recently shown that even a small amount of myocardial scar in patients without a prior history of infarction portends a substantial hazard for death or myocardial infarction when these patients are compared with those without scar, thus supporting the role of infarct characterization in selected high-risk patient groups.

Reimer and Jennings [11] described the wavefront phenomenon, in which ischemic injury follows a subendocardial-to-epicardial segmental pattern and transmural ischemic extent affects the development of resting wall motion abnormality. The degree of transmural involvement currently can be best quantified by LGE and is proportional to the viability of these segments upon revascularization. Kim et al. [12] reported that myocardial scar, identified by LGE, with more than 75% transmural thickness in an area of dyskinesia or akinesia has less than a 5% chance of contractile recovery from revascularization, whereas a scar of less than 25% transmural extent has a greater than 80% likelihood of contractile recovery [12]. In addition, with extensive validation against infarct morphology and size in animal models, superior tissue contrast, and in-plane resolution of 2 mm, there is strong argument for LGE as the current gold standard in noninvasive infarct and viability imaging.

The presence of LGE also has been evaluated in the setting of risk stratification of patients who presented with an acute myocardial infarction. The presence and quality of the scar, specifically the peri-infarct area, have been associated with an increased incidence of arrhythmogenic events [13]. In one study of patients with recent myocardial infarction, infarct tissue heterogeneity as quantified by CMR was predictive of an increased incidence of ventricular inducibility during electrophysiologic testing [14].

LGE has further been used to differentiate responders from nonresponders in the setting of biventricular pacing. The presence of significant scar in the inferolateral wall of the left ventricle was associated more often with nonresponders [15]. Although the initial data appear promising, more studies, including multicenter trials, are needed to further evaluate the concept of risk stratification for sudden death and evaluation of dyssynchrony by CMR before its utility in these settings can be determined.

Nonischemic DCM

Nonischemic DCM is identified by progressive dilatation of the left ventricle associated with the absence of obstructive coronary artery disease. Histopathologic studies have associated DCM with myocardial inflammation and fibrosis, which may be secondary to prior viral infection, toxins, iron overload, or other causes. In single-center experiences, the absence of infarct-appearing LGE of the myocardium excludes coronary artery disease as the cause of LV dysfunction [2]. Occasionally, a more specific diagnosis of DCM can be reached by observing patchy or linear midwall enhancement that does not follow a coronary artery territory.

Ventricular arrhythmia is a frequent cause of sudden death in patients with DCM with a depressed left ventricular ejection fraction [16]. The risk of sudden death in this population has been shown to be reduced with the implantation of a cardiac defibrillator. The severity of transmural involvement of LGE in patients with nonischemic cardiomyopathy has been linked with ventricular arrhythmias and may become a risk-stratifying tool in this population [17].

Myocarditis

Acute myocardial inflammation due to viral infection is a leading identifiable cause of sudden death in young military recruits and up to 20% of men under the age of 40 [18,19]. Vaccinations, such as for smallpox, have been implicated as a cause of myocarditis [20]. Nonspecific flu-like symptoms as the initial manifestations of myocarditis are often discounted, thus leading to an underestimation of the actual incidence of this condition.

LGE plays a limited role in the evaluation of acute myocarditis. As the condition evolves, focal to diffuse enhancement of the ventricular myocardium occurs [6]. Friedrich et al. [21] described specific focal LGE patterns that were related to biopsy-proven parvovirus B19 (PVB19) and human herpes virus 6 (HHV6), the most common viruses in patients with clinical signs of myocarditis. LGE occurred more often in the epicardial lateral free wall in those with PVB19 and in the midwall of the interventricular septum in those with HHV6 [21]. Contrast may be absent depending on the temporal phase of myocarditis (acute, subacute, chronic) present during imaging. During the acute phase of myocarditis, T2-weighted black blood sequences are able to demonstrate focal or diffuse myocardial edema. T2 relaxation is greatly enhanced by the physical mobility of protons associated with water molecules, thereby increasing signal intensities within edematous regions [6]. Because LGE imaging alone is currently unable to differentiate acute from chronic processes, tissue edema imaging can assist in identifying the chronicity of myocarditis by evidence of myocardial edema in cases of recurrent symptoms.

Left ventricular apical ballooning syndrome

Takotsubo cardiomyopathy is characterized by the sudden onset of chest pain, electrocardiographic changes, elevated cardiac enzymes, and left ventricular apical hypokinesis in the setting of nonobstructive epicardial coronary arteries. The underlying cause is unknown, but theories such as sudden transient increases in catecholamine levels versus aborted myocardial infarction have been proposed. The typical feature seen on CMR

in individuals without a history of prior infarct is the absence of LGE within the left ventricle despite significant ventricular dysfunction [22]. Follow-up evaluation characteristically shows resolution of wall motion abnormalities and LV ejection fraction [23].

Cardiac amyloidosis

Cardiac amyloidosis is characterized by the accumulation of immunoglobulin light chains or transthyretin proteins within the myocardial interstitial space and the progressive development of myocardial fibrosis. As a result, the myocardium thickens, cardiac mass increases, diastolic function of the heart is reduced, and restrictive cardiomyopathy may ensue. Although endomyocardial biopsy is considered the clinical gold standard diagnostic test, it has the disadvantage of being invasive; thus, histologic evidence of disease is often only available postmortem [24]. Echocardiographic images may demonstrate ventricular hypertrophy with reduced function. The “sparkling” myocardium sometimes demonstrated with amyloid has a reported sensitivity of 26% to 30% and can be seen in various forms of ventricular hypertrophy [25]. Harmonic imaging can enhance this appearance but can also produce a granular appearance similar to normal myocardium [26].

CMR plays a significant role in identifying the phenotypic expression of cardiac amyloidosis. The kinetics of chelated gadolinium is significantly altered in systemic amyloidosis and corresponds to both cardiac amyloid burden and ventricular dimensions with a diagnostic accuracy of 97% [24]. Chelated gadolinium accumulates in the expanded nonfibrotic interstitial space and demonstrates diffuse, global, subendocardial LGE. Perhaps because of a high systemic burden of amyloid, the contrast is quickly removed from the blood pool, causing rapid loss of signal intensity from gadolinium after injection and rapid recovery of blood T1 values. The combined result of these altered contrast kinetics is that, at best, it becomes difficult to differentiate the blood pool from myocardium. T1 relaxivity of the myocardium is significantly shorter than in controls at 4 minutes and is typically absent at 8 minutes; therefore, LGE sequences need to be performed earlier than the typical 10-minute delay after contrast injection when using doses of 0.1 mmol/kg [4•]. The rapid uptake of contrast and shortened T1 relaxivity make the visualization of enhanced myocardium difficult [27]. LGE imaging of the amyloid heart can be challenging, but it is this feature that sets it apart from other forms of cardiomyopathies.

Cardiac sarcoidosis

Cardiac sarcoidosis, a disease of unknown etiology in which noncaseating granulomas infiltrate the heart, has remained a diagnostic challenge for noninvasive imaging. Sudden cardiac death is the leading cause of death in people with sarcoidosis, but individuals will rarely present with cardiac symptoms. In patients with cardiac sarcoid who reported symptoms of palpitations, presyncope, or syncope, transthoracic echo-cardiography was abnormal in only 25% and findings were independent of the degree of pulmonary impairment [28]. In this potentially treatable disease, it is important to make an accurate diagnosis in order to modify outcomes. Using LGE imaging, CMR is able to demonstrate cardiac involvement independent of wall motion abnormalities. Although the pattern of enhancement in cardiac sarcoidosis can be nonspecific, typically demonstrating diffuse gadolinium accumulation throughout the ventricular myocardium, there is a predilection to the anteroseptal and inferolateral left ventricular epicardial areas [29,30]. LGE also has been shown to be an independent predictor of adverse outcomes, including cardiac death [31].

Applying T2-weighted spin-echo sequences to evaluate for the presence of myocardial edema can be advantageous in differentiating sarcoidosis from acute myocarditis. Used in conjunction with LGE, the absence of T2-weighted signal enhancement in this sequence (ie,

a lack of myocardial edema) suggests a chronic infiltrative disease such as sarcoidosis rather than an acute myocarditis [32] (Fig. 1).

Iron overload

The systemic accumulation of iron in hereditary hemochromatosis or through frequent blood transfusions in thalassemia can result in the deposition of iron and fibrosis within the myocardium and other organs. Cardiac involvement, even in cases of mild myocardial fibrosis, frequently presents with conduction disturbances and systolic and diastolic dysfunction. Hemochromatosis is the leading cause of death in β -thalassemia major, with 50% of patients dying before the age of 35 [33].

MRI takes advantage of the magnetic interaction on a molecular level of iron and hydrogen protons to generate a signal that can quantify iron content, follow the effectiveness of chelation therapy, and determine prognosis. By using gradient-echo images collected from stepwise prolonged echo times, the signal intensity of the myocardium weakens accordingly due to T2* effects. By plotting a region of interest over the myocardium (often the interventricular septum), such a rate of signal decay can be quantified by an exponential decay curve [34]. The reported normal value for T2* is 36 to 68 msec. Values less than 20 msec are considered pathologic and are consistent with iron overload. Left ventricular dysfunction is frequently seen with values below 10 msec [35]. T2* also has been used to monitor the effectiveness of chelation therapy and typically rises as left ventricular function improves [33].

Despite an abnormal T2*, left ventricular mass and end diastolic volumes may be elevated and systolic volumes may be decreased in thalassemia, giving the appearance of an elevated ejection fraction. One should be careful with the interpretation of left ventricular function in this situation, and ejection fraction should not be used alone as a screening tool for myocardial iron overload.

Hypertrophic cardiomyopathy

HCM is a common genetically transmitted disorder that affects 1 in 500 individuals and is characterized by myocardial hypertrophy, fibrosis, diastolic dysfunction, dynamic left ventricular outflow tract obstruction and, rarely, sudden cardiac death. Echocardiography is typically used as a screening tool, but it can underestimate septal thickness and has been shown to miss up to 6% of cases compared with CMR [36]. This is especially true for the apical hypertrophy variants; thus, CMR is suggested for screening this specific phenotype [37].

The ability to image the heart in any plane makes CMR an ideal tool for the assessment of HCM. SSFP can be used to accurately measure septal thickness and shows excellent interobserver agreement. Flow turbulence and systolic anterior motion of the anterior mitral valve leaflet can be seen, which suggests the presence of resting outflow obstruction. However, Doppler echocardiography remains the mainstay for assessment of resting and dynamic obstruction [38,39].

The myocardium does demonstrate LGE in many cases, but the exact etiology is unclear. Myocardial fibrosis, inflammation, and ischemia have been proposed as causes of the retention of gadolinium contrast agents. Whatever the cause, the degree of LGE appears to correlate with other risk factors for sudden death [39].

Arrhythmogenic right ventricular cardiomyopathy

ARVC is a disease characterized by progressive right ventricular dysfunction. The diagnosis is typically made by the application of major and minor criteria proposed in 1994 by the arrhythmogenic right ventricular dysplasia/ cardiomyopathy (ARVD/C) task force.

According to the task force, CMR can add one major or one minor criterion from category I: Global and/or Regional Dysfunction and Structural Alterations, based on the presence of right ventricular aneurysms or the degree of right ventricular dilatation and dysfunction [40]. Imaging sequences for ARVD need to include short- and long-axis SSFP imaging planes to evaluate for the presence of right ventricular abnormalities, including free wall aneurysms, ventricular dilatation, and dysfunction [8,40].

The presence of fibrofatty replacement of the right ventricular myocardium is one of the hallmarks of this disease process. Using T1-weighted black blood turbo spin-echo sequences, fat is enhanced and can be detected infiltrating the right ventricular wall with a reported sensitivity of 84% and specificity of 79%. The presence of fatty infiltration has an unclear significance in the context of normal ventricular size and function. This technique is further limited by the spatial resolution required to differentiate pericardial and epicardial fat from the thin right ventricular free wall, which typically is 2 to 4 mm thick [5,41]. With the application of higher magnetic fields (eg, 3 tesla), the opportunity to improve spatial resolution, given the higher signal–noise ratio, may allow improved visualization of right ventricular fibrofatty infiltration. The clinical significance of this finding needs to be further studied.

LGE also has a limited role in ARVC. Right ventricular enhancement may be present but is again limited by the spatial resolution required to differentiate areas of enhancement from fat and the blood pool. LGE sequences that suppress the fat signal may be helpful, but the signal generated from the stagnant blood pool in a dysfunctional right ventricle may still limit diagnosis.

Currently, quantitative analysis of right ventricular structure and function is most helpful in the diagnosis of ARVC [5]. In a multicenter clinical trial, a right ventricular ejection fraction below 50% and an end-systolic diameter above 3.3 cm (measured 1 cm below the tricuspid valve) were the most accurate measures used to identify individuals with clinical evidence of ARVC [41].

Left ventricular noncompaction

Left ventricular noncompaction is a congenital cardiomyopathy in which the trabecular layer fails to recede. The left ventricle is filled with complex trabeculations that project into the chamber, interposed by deep inter-trabecular recesses. The clinical impact can be variable, ranging from an asymptomatic presentation to the development of progressive heart failure, thromboembolic disease, and sudden cardiac death [42].

Case reports have described both perfusion abnormalities and LGE of involved myocardium by CMR, but the currently accepted criteria are based on the ratio of non-compacted to compacted myocardium. A ratio above 2.3, measured in end diastole, is currently considered the cutoff value for the presence of left ventricular noncompaction and offers the highest degree of diagnostic accuracy [43,44].

Cardiac transplantation

Rejection of the transplanted heart is limited by the use of immunosuppressive medications but still can occur in up to 75% of allograft recipients within the first year [45]. Typical findings of rejection include myocardial lymphocytic infiltration and inflammation as well

as coronary arteriopathy. Cardiac biopsy is the gold standard for rejection surveillance, but it places the patient at risk for adverse events and can be limited by false negative results and variations in interpretation of the sample [45,46]. With its quantitative assessment of myocardial perfusion and high tissue contrast, CMR has been evaluated for its role as a noninvasive means of detecting allograft rejection and coronary arteriopathy. Although CMR's role in this regard needs to be further explored and CMR has not been incorporated into the current standard of practice, this technology shows strong promise in the monitoring and screening of disease given the lack of symptoms experienced by heart transplant recipients due to cardiac denervation.

A variety of CMR sequences have been used to assess for the presence of acute rejection, including SSFP, T2-weighted black blood fast spin-echo, myocardial perfusion reserve, and LGE [46–48]. While each of these has its advantages and disadvantages, tissue edema imaging and alterations in myocardial perfusion reserve appear to be the most promising techniques for detecting early rejection. In one study, T2 imaging was able to detect moderate to severe acute rejection, as defined by biopsy, with a sensitivity of 89% and specificity of 70%. The authors also demonstrated that, compared with transmitral Doppler and baseline biopsy, T2 imaging was the most significant predictor of acute rejection within the first 3 months of transplantation, with a sensitivity of 80% in the first month of follow-up [49].

The quantitative evaluation of segmental myocardial perfusion reserve has been studied in the setting of coronary artery disease, syndrome X, and transplant arteriopathy. Comparing alterations in contrasted signal intensity over time, myocardial perfusion of the epicardial and endocardial layers at rest and during vasodilator stress can be separately evaluated with an in-plane resolution of 2 mm. An endomyocardial perfusion reserve greater than 2.3 excluded the presence of transplant arteriopathy, with positive and negative predictive values of 63% and 100%, respectively. In addition, using only resting endocardial–epicardial perfusion ratios greater than 1.3, in the absence of left ventricular hypertrophy and/or prior rejection, transplant arteriopathy could be excluded, with positive and negative predictive values of 78% and 100%, respectively [50]. The long-term prognostic implication of CMR perfusion findings in this disease need to be further studied.

Conclusions

The evaluation of cardiomyopathy can be clinically challenging. Determining the underlying pathology of disease in these conditions can direct the clinician to appropriate and possibly life-saving therapies. The application of CMR in the diagnostic evaluation of cardiomyopathy of unknown origin has been identified as an appropriate use of this technology [7•]. The unique features of CMR, including exceptional temporal and spatial resolution, tissue characterization, perfusion analysis, and ability to image in any tissue plane, make it an ideal tool for use in this clinical setting. Cardiomyopathy sequencing protocols have been published to standardize and streamline the approach to the clinical scan [8•]. An experienced staff can perform a thorough study within 30 minutes and provide a multimodality cardiac survey to help the clinician determine therapies that may ultimately improve patient outcomes.

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Figure 1.

Imaging in a 42-year-old man with a history of pulmonary sarcoidosis with syncope revealed (A) normal left ventricular dimensions and function (short-axis steady state free precession [SSFP]); (B) an absence of tissue edema (T2-weighted black blood); (C) epicardial delayed enhancement of the anteroseptum and inferolateral wall (myocardial delayed enhancement [MDE]); and (D) normal myocardial perfusion (perfusion). Two months later, a repeat study demonstrated (E) normal left ventricular dimensions and function (four-chamber SSFP) and (F) increased delayed enhancement of the anteroseptum and lateral wall (MDE).

Table 1

Typical CMR sequences for evaluation of cardiomyopathies

Sequence name	Sequence function
Cine SSFP or fast gradient echo	Myocardial structure and function
T1-weighted black blood	Myocardial fat imaging
T2-weighted black blood	Tissue edema
Inversion recovery late enhancement	Late gadolinium enhancement
T2*	Myocardial iron content

CMR—cardiac magnetic resonance imaging; SSFP—steady state free precession.

Table 2

CMR approach to cardiomyopathies

Cardiomyopathy	Sequences			
	SSFP	LGE	T1 black blood	T2 black blood T2*
Ischemic	•	•		•
DCM	•	•		•
Myocarditis	•	•		•
Takatsubo	•	•		•
Amyloidosis	•	•		•
Sarcoidosis	•	•		•
Iron overload	•			•
HCM	•	•		
ARVC	•		•	
Noncompaction	•	•		
Transplant	•	•		•

ARVC—arrhythmogenic right ventricular cardiomyopathy; CMR—cardiac magnetic resonance imaging; DCM—dilated cardiomyopathy; HCM—hypertrophic cardiomyopathy; LGE—late gadolinium enhancement; SSFP—steady state free precession.