Clinical Application of Cardiac CMR

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Cardiovascular magnetic resonance (CMR) imaging is an important clinical tool that aids in the diagnosis and management of patients with cardiomyopathy. With its ability to assess morphologic and physiologic myocardial characteristics in the same imaging session, CMR can effectively rule out less common causes of cardiomyopathy, including cardiac hemochromatosis, amyloidosis, and arrhythmogenic right ventricular tachycardia. The combination of cine function, myocardial perfusion at rest and under stress, and late gadolinium enhancement provides a strong assessment that can establish the cause of the cardiomyopathy as well as guide therapy in cases of ischemic cardiomyopathy. CMR can also identify microvascular obstruction in acute myocardial infarction. This technique can be especially helpful in the diagnosis of conditions such as arrhythmogenic right ventricular dysplasia, cardiac sarcoidosis, and myocarditis. It can also be used to evaluate patients with chest pain and pericardial diseases.

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Key words: Cardiovascular magnetic resonance • Late gadolinium enhancement • Cardiomyopathy • Amyloidosis

Cardiovascular magnetic resonance (CMR) imaging is playing an increasing role in the assessment of patients with various cardiovascular disorders. With its enhanced spatial and temporal resolution, improved tissue characterization, and lack of ionizing radiation, it is often the test of choice in the evaluation of patients with new-onset cardiomyopathy of unknown etiology. This article will review the role of CMR in the diagnosis and management of patients with cardiomyopathy, suspected ischemic heart disease, and pericardial diseases.

Evaluation of Cardiomyopathy

CMR is an important clinical tool that aids in the diagnosis and management of patients with cardiomyopathy. It provides accurate and reproducible volumetric assessment of the left and right ventricular (RV) size, volume, wall thickness, and mass, in addition to the ejection fraction, with small interobserver and intraobserver variability.[1] In addition, CMR provides resting and stress perfusion data. With its ability to assess morphologic and physiologic myocardial characteristics in the same imaging session, CMR can effectively rule out less common causes of cardiomyopathy, including cardiac hemochromatosis, amyloidosis, and arrhythmogenic RV tachycardia.

The combination of cine function, myocardial perfusion at rest and under stress, and late gadolinium enhancement (LGE) provides a strong assessment that can establish the cause of the cardiomyopathy as well as guide therapy in cases of ischemic cardiomyopathy. Gadolinium–diethylenetriamine penta-acetic acid (DTPA) is an extracellular agent, and delayed gadolinium retention in the interstitial space is increased in conditions with increased extracellular volume of distribution and/or decreased washout, such as acute necrosis, fibrosis, infiltration, and inflammation. Because gadolinium shortens T1 relaxation time, it results in brighter signal intensity in the areas of necrosis, fibrosis, infiltration, and inflammation. This technique is sensitive and reproducible in the detection of myocardial infarction (MI) across different manufacturers of CMR scanners. In an international, multicenter trial that evaluated the performance of LGE CMR for the detection of myocardial infarction, precontrast and postcontrast CMR images were randomized and then scored for enhanced regions by 3 independent readers. The sensitivity of magnetic resonance imaging (MRI) for detecting MI was 99% (acute) and 94% (chronic) after contrast, compared with 11% before contrast. Likewise, the accuracy of MRI in identifying MI location (compared with infarct-related artery perfusion territory) was 99% (acute) and 91% (chronic) after contrast, compared with 9% before contrast.[2]

LGE CMR has also been used to differentiate ischemic from nonischemic cardiomyopathy. A study that evaluated 90 patients with chronic, stable, dilated cardiomyopathy in an outpatient heart failure clinic found that all 27 patients with known coronary artery disease (CAD) had an ischemic pattern of LGE on CMR. Non-CAD LGE pattern (midwall striae or patchy foci of LGE) was noted in 18 patients, and no evidence of LGE was found in 37 patients (Figure 1).[3]

CMR not only can help make the diagnosis but can also direct therapy by providing information regarding the likelihood of functional recovery because revascularization of dysfunctional but viable myocardium may improve left ventricular function and long-term survival. Noncontractile yet viable myocardium can be the result of acute, subacute, or chronic states of myocardial perfusion abnormalities. Given its high temporal resolution, CMR provides an accurate description of regional wall motion abnormalities. Kim and colleagues[4] performed cine and contrast CMR in 50 consecutive patients with chronic left ventricular dysfunction before and 11 weeks after patients underwent surgical or percutaneous revascularization. When all dysfunctional segments before revascularization were considered, the proportion with contractile improvement decreased progressively as the transmural extent of hyperenhancement increased ($P < .001$). When the volume of dysfunctional but viable myocardium before revascularization was calculated on a patient-by-patient basis, an increasing extent of dysfunctional but viable myocardium correlated with greater improvements in the mean wall-motion score ($P < .001$) and the ejection fraction after revascularization ($P < .001$).[4]

In addition, preliminary evidence suggests that CMR may provide important prognostic information in patients with ischemic cardiomyopathy who suffer an MI beyond left ventricular ejection fraction. With its high spatial resolution, CMR can allow the identification and

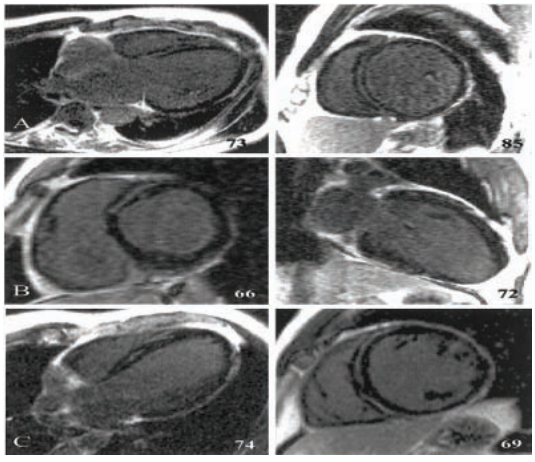


Figure 1. *Three patients with dilated cardiomyopathy with midwall striae of enhancement. Gadolinium enhancement followed the ventricular longitudinal muscle fibers, particularly those involving the septum and basal to mid–left ventricular regions. Reprinted with permission from McCrohon JA et al. Differentiation of heart failure related to dilated cardiomyopathy and coronary artery disease using gadolinium-enhanced cardiovascular magnetic resonance.* Circulation. *2003;108(1):54-59.*[3]

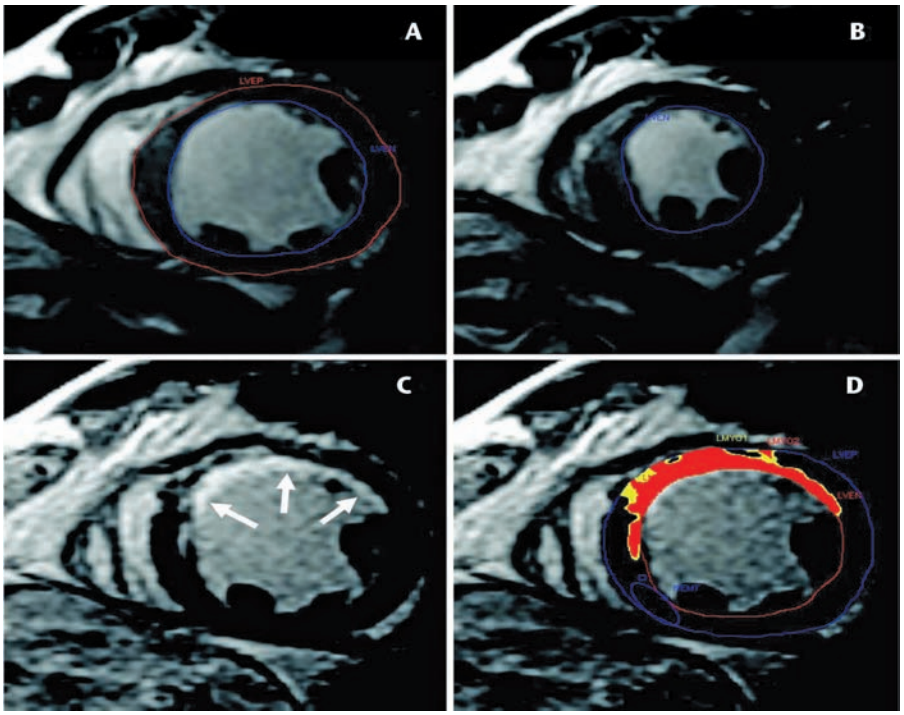


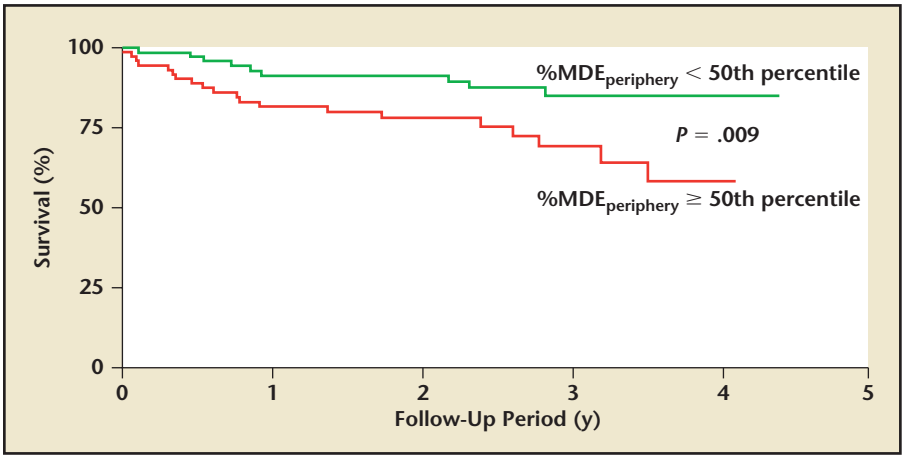
Figure 2. *Diastolic* **(A)** *and systolic* **(B)** *cine images showing akinesis in the anteroseptum and anterior wall. The endocardial (blue) and epicardial (red) borders were manually traced. In images* **C** *and* **D**, *a computer-assisted, semiautomatic technique for quantifying* $\%MDE_{periphery}$ *on delayed-enhancement images shows an anterior myocardial infarction (white arrows) in the same patient. A computer-assisted algorithm applied the signal-intensity thresholds of more than 3 standard deviations and 2 to 3 standard deviations above the normal myocardial segment to delineate the infarct core (red region) and peri-infarct zone (yellow region), respectively. Reprinted with permission from Yan AT et al. Characterization of the peri-infarct zone by contrast-enhanced cardiac magnetic resonance imaging is a powerful predictor of postmyocardial infarction mortality.* Circulation. *2006;114(1):32-39.*[5]

quantification of the peri-infarct zone. In a study of 144 patients with documented CAD and abnormal LGE consistent with MI, a computer-assisted, semiautomatic algorithm quantified the total infarct size and divided it into the core and peri-infarct regions based on signal-intensity thresholds (Figure 2). After a median follow-up of 2.4 years, patients with an above-median peri-infarct zone (expressed as a percentage of the total LGE size) experienced higher hazards for death compared with those with a below-median peri-infarct zone (28% vs 13%; log-rank $P < .01$) (Figure 3). Multivariable analysis showed that left ventricular systolic volume index and the extent of the peri-infarct zone were the strongest predictors of mortality. Thus, the extent of the peri-infarct zone characterized by CMR provides incremental prognostic value beyond the left ventricular systolic volume index or ejection fraction. Infarct characteristics may prove to be a unique and valuable noninvasive predictor of post-MI mortality.[5]

Another study evaluated the relationship between tissue heterogeneity in the infarct periphery and arrhythmic substrate. A group of 47 patients underwent cine and contrast-enhanced CMR to measure left ventricular function, volume, mass, and infarct size prior to cardioverter defibrillator implantation for primary prevention. Quantification of tissue heterogeneity at the infarct periphery was strongly associated with inducibility for monomorphic ventricular tachycardia (noninducible vs inducible: 13 ± 9 vs 19 ± 8 g; $P = .015$). This finding suggests that anatomic tissue heterogeneity increases susceptibility to ventricular arrhythmias in patients with prior MI and left ventricular dysfunction and may be the mechanism for the increased mortality in patients with an increased peri-infarct zone.[6]

CMR also identifies microvascular obstruction in acute MI. This is demonstrated early (1-2 minutes) after intravenous injection of gadolinium. Microvascular obstruction detected by CMR has been linked to ventricular remodeling and adverse

Figure 3. *Kaplan-Meier survival curves for all-cause mortality, stratified by median* $\%MDE_{periphery}$. *Reprinted with permission from Yan AT et al. Characterization of the peri-infarct zone by contrast-enhanced cardiac magnetic resonance imaging is a powerful predictor of postmyocardial infarction mortality.* Circulation. *2006;114(1):32-39.*[5]



cardiovascular events.[7] Finally, the transmural extent of late gadolinium-enhancement CMR predicts recovery of function following acute MI.[8]

It is important to note, however, that LGE is not specific to ischemic cardiomyopathy. It can also be seen in infiltrative and inflammatory pathology of the myocardium, but in different patterns. Several different causes of dilated cardiomyopathy with associated LGE are discussed below.

Arrhythmogenic Right Ventricular Dysplasia

Arrhythmogenic right ventricular dysplasia (ARVD) is an uncommon but life-threatening cardiomyopathy most often described in young adults. Although a noninvasive diagnosis is crucial in prevention of a potentially fatal consequence of the disease, the diagnosis of ARVD is challenging because it lacks a unique genetic inheritance pattern, has heterogeneous clinical presentation, and has nonspecific electrocardiogram (ECG) findings. Its diagnosis is currently based on the presence of major and minor criteria that include structural, functional, histologic, electrocardiographic, arrhythmic, and genetic factors. The diagnosis of ARVD is based on the presence of 2 major criteria, 1 major plus 2 minor criteria, or 4 minor criteria. Minor criteria include a family history of premature sudden cardiac death (< 35 years) or suspected ARVD, ECG abnormalities in the right precordial leads (V1-V3), and mild global or segmental RV wall motion abnormalities. Major criteria include family disease confirmed at necropsy or surgery, ε waves on ECG, severe segmental or global RV dilatation, RV aneurysms, and fibrofatty replacement of RV myocardium.

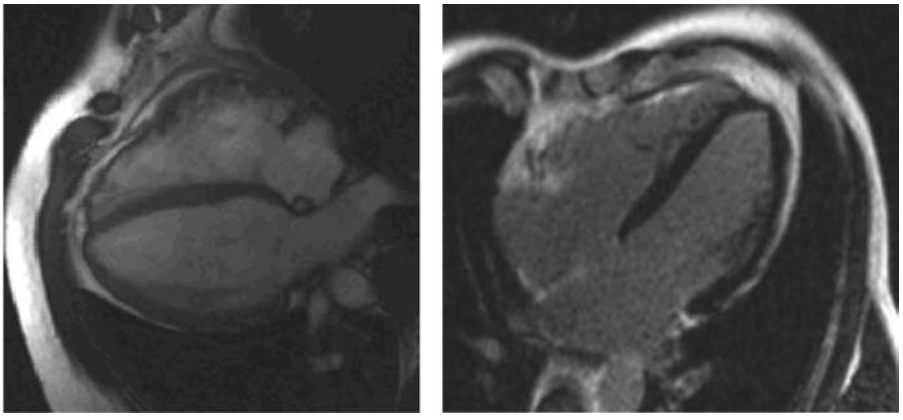
Endomyocardial biopsy is unreliable for the diagnosis of ARVD because the patchy distribution of the fibrofatty change may lead to sampling error, and there is therefore a need to develop reliable imaging criteria to make the diagnosis. The imaging techniques used to evaluate RV abnormalities include echocardiography, computed tomography, radionuclide angiography, and CMR. Among these, CMR is the most accepted method because it can characterize RV wall motion abnormalities, thinning of the myocardium, RV dilatation, and any fibrofatty infiltration of the RV myocardium. The diagnosis of ARVD is suggested on CMR when there are areas of dyskinesia, sacculation, and aneurysm formation along with fat infiltration of the RV wall (Figure 4). In addition, the hallmarks of ARVD include fatty infiltration of the RV free wall that can be suppressed in fat saturation sequences. In a study of 40 patients with suspected ARVD, the sensitivity of fat infiltration, RV enlargement, and regional RV dysfunction for diagnosing ARVD was 84%, 68%, and 78%; the specificity for these factors was 79%, 96%, and 94%, respectively.[9,10]

In addition, it has been suggested that LGE imaging provides additional evidence of fibrosis, which often coexists in the infiltrated RV myocardium in affected individuals. It is of note that high spatial resolution is required to identify intramyocardial fat by conventional T1-weighted fast spin echo images. This approach can often be challenging because the right ventricle is a thin structure, and areas of affected myocardium can be small. In addition, proximity to the surface coil, truncation band artifacts, and various motion-related ghosting artifacts may cause high-signal intensities to be projected onto the myocardium and mistaken for fat. Although most CMR centers routinely perform fast spin-echo imaging that provides potentially supportive evidence in suspected cases, only data from regional and global RV dilatation and dysfunction are incorporated into the criteria for diagnosing ARVD.

Amyloidosis

Diagnosis of cardiac involvement in amyloidosis is a diagnostic challenge because of the nonspecific morphologic features that often exist in other common medical conditions, such as hypertension or other infiltrative heart disease. At a histologic level, amyloidosis is made evident by extracellular deposition of insoluble fibrillar proteinaceous material in various organs and tissues. Cardiac involvement has been described in

Figure 4. *Cine magnetic resonance imaging and delayed enhancement in a patient with arrhythmogenic right ventricular dysplasia. Note the right ventricular free wall aneurysms with associated late gadolinium enhancement.*



most forms of amyloidosis, although it is most common and most often clinically significant with type AL amyloidosis (primary amyloidosis), which is often associated with multiple myeloma or other monoclonal gammopathies. Differentiation of amyloidosis from other forms of restrictive cardiomyopathy is of utmost importance because it has significant therapeutic implications.

Studies in the literature regarding cardiac involvement in amyloidosis remain limited but are increasing. Maceira and colleagues[11] studied 29 patients with systemic amyloidosis and 16 hypertensive controls using gadolinium-enhanced cardiac CMR. Myocardial enhancement was associated with increased ventricular mass and reduced left ventricular systolic function. Shortly after intravenous administration of gadolinium contrast (in less than 5 minutes), amyloidosis patients had a diminished T1 difference between the myocardium and blood as compared with the difference observed in control patients. Most importantly, this study confirmed that LGE can detect myocardial infiltration by β-pleated amyloid protein based on pathologic specimens. Patients with amyloidosis were noted to have qualitative global and subendocardial gadolinium enhancement of the myocardium.[12] This pattern of LGE could be seen in both atria and was associated with reduced left atrial emptying function (Figure 5). Recently, this pattern has been associated with a sensitivity of 80%, yielding a specificity of 94%. The positive predictive value was 92%, and the negative predictive value was 85%, for diagnosis of cardiac amyloidosis.[13]

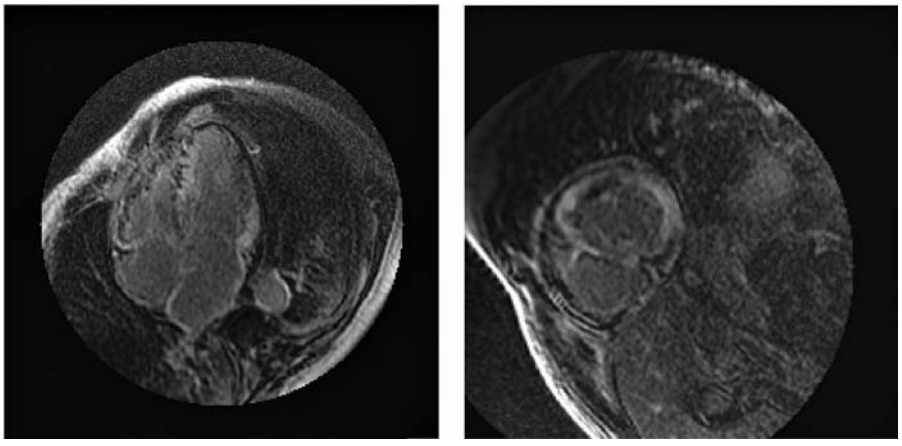
Sarcoidosis

The diagnosis of cardiac sarcoidosis, like that of amyloidosis, is a challenge. The clinical features of cardiac sarcoidosis include congestive heart failure, cor pulmonale, supraventricular and ventricular arrhythmias, conduction disturbances, ventricular aneurysms, pericardial effusion, and sudden death. Criteria proposed by the Japanese Ministry of Health (JMH) for the diagnosis of cardiac sarcoidosis are currently the gold standard. These criteria include histologic, clinical, echocardiographic, nuclear, and ECG factors. LGE CMR could potentially detect more cases than the proposed JMH criteria. Smedema and colleagues[14] analyzed the accuracy of CMR for the diagnosis of cardiac sarcoidosis in 58 patients with confirmed systemic sarcoidosis. The diagnosis was made according to the JMH criteria in 12 of the 58 patients (21%); CMR revealed LGE, mostly involving basal and lateral segments (typical in sarcoidosis), in 19 patients. The sensitivity, specificity, positive predictive value, negative predictive value, and overall accuracy of CMR in detecting cardiac sarcoidosis using the JMH criteria as the gold standard were 100%, 78%, 55%, 100%, and 83%, respectively.[14] Interestingly, 7 patients had evidence of LGE on CMR, but they did not meet the JMH criteria. Although delayed gadolinium enhancement is not specific for the cardiac sarcoidosis, its pattern and location adds to its specificity.[15] The transmural extent of LGE on CMR correlates significantly with regional ^{201}Tl uptake and with regional wall motion in prior series of patients. However, ^{201}Tl perfusion defects or wall motion abnormalities were undetectable in some segments with mild-to-moderate LGE, suggesting that LGE may be a more sensitive technique for detecting cardiac involvement.[16] In addition, sarcoid infiltrates are visible on MRI as intramyocardial focal zones with increased signal intensity on T2-weighted images because of edema associated with inflammation. In addition, cine MRI could reveal regional wall motion abnormalities corresponding to the area of cardiac involvement.[17] In addition, cardiac MRI could reveal other findings in patients with sarcoidosis. RV hypertrophy, dilatation, and hypokinesis are more often related to pulmonary hypertension secondary to sarcoid lung disease rather than to specific RV involvement by sarcoidosis.

Myocarditis

Myocarditis is another cause of dilated cardiomyopathy that is occasionally complicated by fulminant heart

Figure 5. *Late gadolinium enhancement in a patient with cardiac amyloidosis.*



failure and sudden death. The initial clinical presentation is insidious and could be difficult to recognize clinically. Thus, there is a need for early diagnostic tools and strategies to diagnose this potentially fatal disease.

In a study of 32 patients diagnosed with myocarditis by clinical criteria, LGE on CMR was present in 28 patients (88%) and was usually seen with 1 or more foci in the myocardium. Foci were most frequently located in the lateral free wall. Among the 21 patients in whom biopsy was obtained from the region of contrast enhancement, 19 had active myocarditis, as revealed by histopathologic analysis. At follow-up, the area of contrast enhancement decreased from 9 ± 11% to 3 ± 4% of the left ventricular mass, as the left ventricular ejection fraction improved from 47 ± 19% to 60 ± 10%.[18]

Evaluation of Patients With Chest Pain

Chest pain is one of the most common complaints in the emergency department. It results in 5 million emergency department visits and 2 million hospitalizations annually, with a cost of more than $8 billion. However, cardiac etiology is found in less than one-third of these patients. Management of chest pain in the emergency department remains a challenge with current diagnostic strategies. There are several approaches to detecting CAD using CMR. They include the visualization of the effects of induced ischemia (wall motion, perfusion) and direct visualization of coronary arteries (coronary angiography and flow).

CMR first-pass perfusion imaging is a promising myocardial perfusion technique that captures the first-pass dynamics of a contrast bolus as it transits through the coronary bed and myocardium at very high spatial resolution. CMR first-pass perfusion imaging has the following advantages: the lack of soft tissue attenuation, accurate delineation of the myocardium from the blood pool due to high spatial and temporal resolution, a stable contrast agent with an excellent safety profile, and the lack of need for ionizing radiation. In addition, the technique can be performed in less than an hour. It has been shown to accurately identify patients with possible or probable acute coronary syndrome. Kwong and colleagues[19,20] prospectively evaluated 161 consecutive patients in the emergency room who had more than 30 minutes of chest pain but whose ECGs were not diagnostic of acute myocardial infarction. Cardiac CMR was performed at rest within 12 hours of presentation and included perfusion, left ventricular function, and gadolinium-enhanced MI detection. The sensitivity and specificity for detecting acute coronary syndrome were 84% and 85%, respectively, by CMR. CMR was more sensitive than strict ECG criteria for identification of ischemia and peak troponin-I, and it was the strongest independent predictor of acute coronary syndrome. It added diagnostic value to the clinical parameters.[19,20] Recently, other series have confirmed the high diagnostic accuracy and negative predictive value of CMR perfusion imaging.[21,22]

Evaluation of Pericardial Diseases

Although echocardiography is the most common initial imaging tool to assess pericardial pathology, CMR offers the following distinct advantages: unrestricted imaging of all areas of the pericardial sac, superior tissue contrast to assess for contents of pericardial fluid and the myocardium, and sufficient temporal and spatial resolution to assess for any constrictive physiology of affected cases. Normal pericardium typically appears on CMR as a hypointense linear structure on T1-weighted imaging. The pericardium is most prominent adjacent to the RV free wall and the inferior and apical aspect of the left ventricle (Figure 6). Abnormal pericardium may appear thickened and may be associated with increased pericardial effusions. CMR is accurate in quantifying the thickness of the pericardium because it visualizes the entire pericardium or loculated pericardial effusion. In addition, CMR allows examination of the entire chest and detection of the associated abnormalities in the mediastinum and lungs.

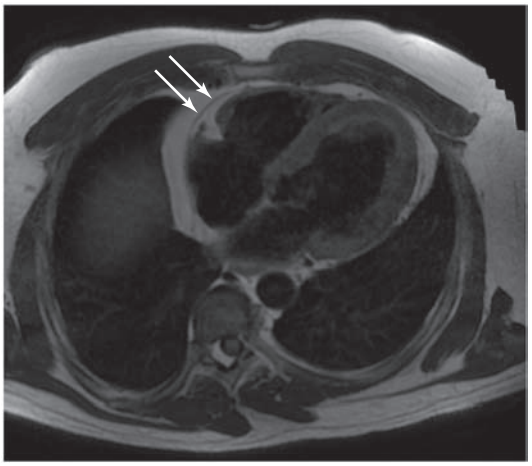


Figure 6. *The pericardium is most prominent adjacent to the right ventricular free wall and the inferior and apical aspect of the left ventricle. The arrows show normal pericardium.*

Soft-tissue contrast on CMR is superior to echocardiograms. Typically, a combination of T1W and T2W sequences and cine gradient echo imaging in matching slice locations can provide complementary information regarding pericardial structure, fluid flow, and content of the pericardial effusion. Real-time CMR imaging produces low signal-to-noise ratio motion images but free-breathing images, thus allowing the evaluation of the septal motion during respiratory movements and interventricular dependence.

Soft-tissue contrast on cardiovascular magnetic resonance is superior to echocardiograms.

The off-label use of intravenously injected gadolinium-DTPA has been reported to enhance visualization of the pericardium in patients with pericarditis. The diagnosis of acute pericarditis can be made in the presence of LGE in the pericardium that suggests diffuse pericardial inflammation.[23]

In addition, CMR is more accurate in locating and sizing loculated pericardial effusion, identifying pericardial hemorrhage, and characterizing the content of the pericardial mass or fluid. Table 1 summarizes the characteristics of different types of pericardial effusion using CMR.

CMR also plays an important role in the differentiation between constriction and restriction. The hallmark of constriction on CMR includes increased pericardial thickness (> 4 mm), epicardial and pericardial adhesions noted on tagging, atrial enlargement, tubular right ventricle, dilated inferior vena cava, and septal bounce or "shiver." Recently, the use of real-time MRI made it possible to demonstrate septal inversion with inspiration.[24] In a study of 29 patients with unsuspected constrictive pericarditis, CMR had a sensitivity, specificity, and accuracy of 88%, 100%, and 93%, respectively, in making the diagnosis of constriction using surgery and/or catheterization as the gold standard. Thickened pericardium was observed in 88% of patients with proven constrictive pericarditis.[25]

Current Limitations of Cardiac MRI

Cardiac MRI is limited by multiple factors, including the availability of the technology and of trained physicians and technicians who can perform and interpret these scans. In addition, as the population grows older, many patients are requiring pacemakers and defibrillators. These devices are mostly not CMR safe, and these patients cannot undergo MRI when needed. (Some recent studies, however, have suggested that MRI can be safely performed in patients who have selected types of pacemakers and implantable defibrillators.[26,27]) In addition, cardiac MRI still lags behind in the anatomic imaging of the coronary arteries compared with cardiac computed tomography.

The recently described nephrogenic systemic fibrosis syndrome is associated with gadolinium use in patients with impaired renal function. It has limited the use of CMR in this patient population, which has a high prevalence of coronary disease.[28] This serious disease is characterized by subacute swelling of distal parts of the extremities, followed by severe skin induration and, sometimes, anatomic extension involving the thighs and lower abdomen. The skin induration may be aggressive and associated with constant pain, muscle restlessness, and loss of skin flexibility. In some cases, nephrogenic systemic fibrosis leads to serious physical disability and death.[29]

Table 1
Distinguishing Characteristics of Types of Pericardial Effusion

Pericardial Effusion	T1W Fast Spin-Echo	T2W Fast Spin-Echo
Transudative	Low	High
Exudative	Medium	High
Proteinaceous	High	Very high
Acute hemorrhagic	Homogeneous, high	Homogeneous, low
Subacute/chronic hemorrhagic	Heterogeneous	Subacute: heterogeneous Chronic: homogeneous

Summary

CMR imaging plays a key role in the evaluation of patients with various cardiovascular disorders, such as cardiomyopathy, suspected ischemic heart disease, and pericardial diseases. This modality provides essential clinical information that is necessary to the workup and management of these patients. Thus, it is not surprising to see a significant increase in the number of CMR scans performed. ■

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Main Points

- With its high temporal resolution, cardiovascular magnetic resonance (CMR) imaging provides accurate description of regional wall motion abnormalities.
- Preliminary evidence suggests that CMR may provide important prognostic information in patients with ischemic cardiomyopathy who suffer a myocardial infarction beyond left ventricular ejection fraction.
- The extent of the peri-infarct zone characterized by CMR provides incremental prognostic value beyond the left ventricular systolic volume index or ejection fraction. Infarct characteristics may prove to be a unique and valuable noninvasive predictor of post-MI mortality.
- There are several approaches to detecting coronary artery disease using CMR, including the visualization of the effects of induced ischemia (wall motion, perfusion) and direct visualization of coronary arteries (coronary angiography and flow).
- Use of cardiac CMR may be limited in some patients, including those with pacemakers or defibrillators and those with impaired renal function.