

Cardiovascular magnetic resonance from basics to clinical applications

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Cardiac magnetic resonance imaging (CMR) is the sole imaging modality with the ability, in 3 dimensions, to assess cardiac morphology, ventricular function, perfusion, viability and imaging characteristics of the surrounding vasculature without ionizing radiation.¹ CMR uses the same principles as other MR techniques with the addition of ECG gating in order to suspend cardiac motion.

The increasingly sophisticated treatment of patients with cardiac disorders has created the need for accurate and reproducible measurements of cardiac chamber volumes and function.² CMR has the ability to provide this information as well as assess edema, perfusion, viability and vascular anatomy.

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Techniques of CMR

Ventricular morphology

Black blood imaging can be used to create still-frame images with high spatial resolution for morphologic analysis (Figure 1). Bright-blood steady state free precession (SSFP) sequences provide moving cine images with a high signal-to-noise ratio (SNR) and excellent endocardial definition. Standard views are derived from their echocardiography counterparts; 4-chamber, 2-chamber (vertical long axis), left ventricular outflow tract (3-chamber), and short axis stacks (Figure 2). Morphologic variants of atrial, ventricular, valvular, arterial and venous structures can be imaged with unsurpassed definition.³ The ability to image in any plane within the thorax is a unique strength of CMR, of particular advantage in congenital disorders.

Left ventricular function

Cardiovascular MR measurements of left ventricular (LV) function (Figure 2), are usually obtained from a series of ECG-gated SSFP images obtained in the short axis view.⁴ Using either the 2- or 4-chamber view as a reference, a short-axis stack is prescribed from the plane of the mitral valve orthogonal to the long axis of the ventricle in end-diastole. A stack of images extending to the cardiac apex is then acquired during suspended respiration, at a slice thickness

of approximately 6 to 8 mm, with 2 mm interslice gap. Using either automated or manual techniques, the endocardial and epicardial borders are traced for each slice and the ventricular volumes are calculated. Reproducibility is best obtained by including the papillary muscles and trabeculae in the LV volume (Figure 2).⁵ CMR remains the gold standard for measurements of LV mass, volume and ejection fraction (LVEF), as well as for regional wall-motion abnormalities,^{6,7} and is more reproducible than echocardiography.⁸

The major source of error occurs at the basal slice during ventricular systole, because of

- the fixed imaging plane, and
- the descent of the mitral valve towards the LV apex, moving through the imaging plane.

Allow for LV shortening by careful cross-referencing of planes to delineate the position of the mitral annulus at end-systole and end-diastole.

Right ventricular function

Accurate right ventricular (RV) assessment demands 3-dimensional techniques because of the non-geometric shape of the ventricle. The RV position has also traditionally made reliable echocardiographic measurements difficult. RV dysfunction, as assessed by CMR, predicts a poor prognosis late after myocardial infarction (MI),⁹ and

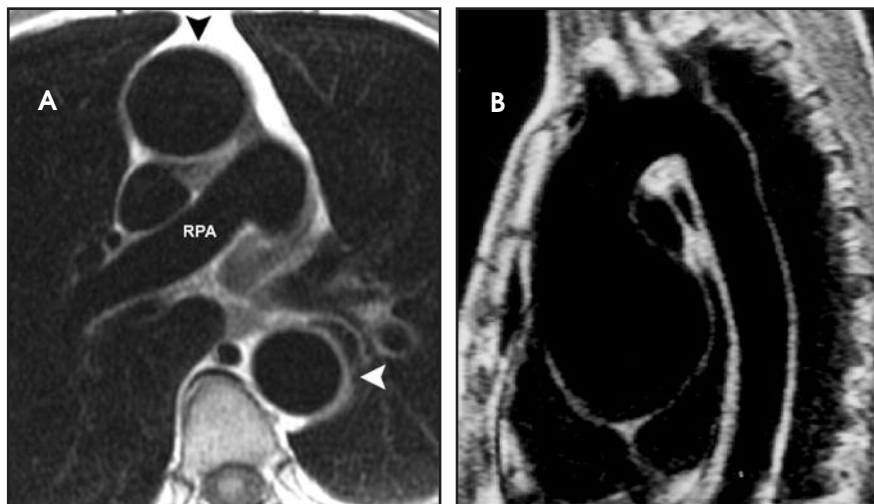


FIGURE 1. Black blood images of the great vessels. Axial image (A) shows the relationship of the ascending and descending aorta to the right pulmonary artery (RPA). Oblique coronal image (B) shows a patient with Marfan's syndrome and severe dilatation of the ascending aorta.

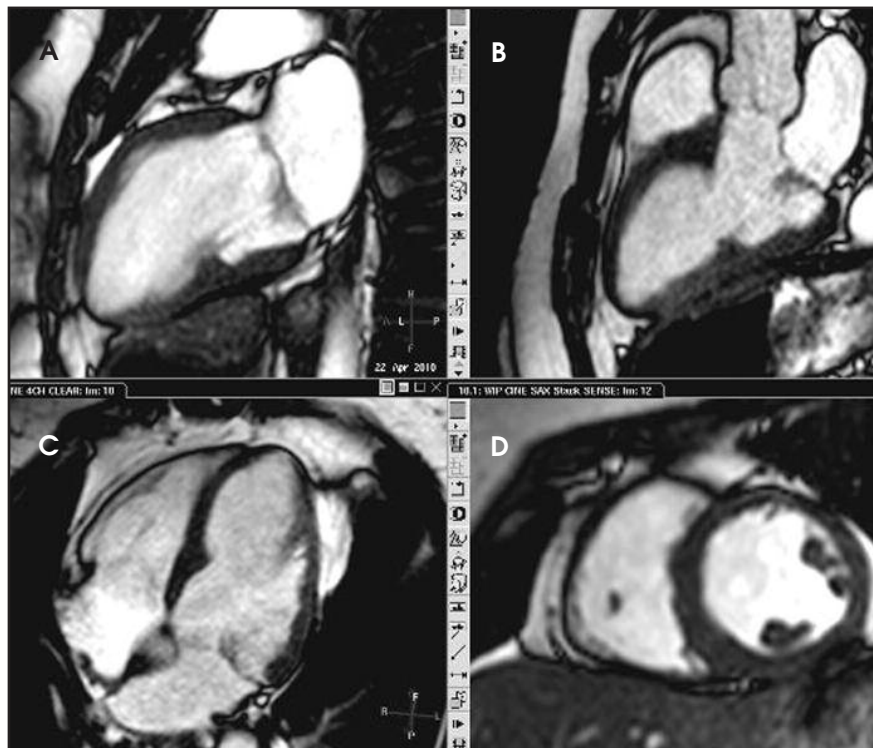


FIGURE 2. Standard CMR views. Vertical long axis (2-chamber, A), LVOT (3-chamber, B), 4-chamber (C), short axis (D).

is of particular importance in pulmonary hypertension and adult congenital heart disease.³

In many centers, it is customary to use the LV short axis view to assess RV volume. However, the basal dimension of the RV is considerably larger than the base of the LV, and when errors

occur in the base of the RV they may significantly affect accuracy, usually by underestimating RV volumes.^{8,10} The tricuspid valve annular plane lies apical of the mitral valve plane. When the same series of LV short axis views is used for RV measurements as for LV measurements, the basal end-diastolic

and end-systolic slice positions may be difficult to determine. This is compounded because, in most cases, both RV and atrial walls are thin, rendering it difficult to identify the exact plane of the tricuspid valve. RV quantification can be improved by using axial views or, preferably, dedicated RV sequences where the tricuspid valve is imaged in the margin of the slice.^{10,11} Axial imaging has disadvantages in congenital heart diseases where the cardiac axis and the skeletal axis may not align, leading to inaccuracies in delineating the atrio-ventricular plane.

Late gadolinium enhancement

Tissue characterization with late gadolinium enhancement (LGE) is one of the unique properties of CMR. This phenomenon results from inherent relative differences in the volume of distribution of gadolinium (Gd) between normal and abnormal myocardium.^{12,13} Gadolinium is normally confined to the extracellular and interstitial space (e.g., it does not penetrate intact myocardial cell membranes). Changes to the interstitium, such as infiltration or fibrosis, increase the volume of distribution, allowing a larger amount of Gd to penetrate into the tissue. T1-weighted (T1W) CMR imaging performed early after Gd administration is used for assessment of myocardial perfusion during “first pass” entry into the myocardial microcirculation, analogous to single-photon emission computed tomography (SPECT) perfusion imaging of the myocardium. Imaging performed late (10 to 20 min) after Gd administration allows washout from the myocardial circulation. Myocardial signal is nulled by the use of an inversion pulse, leaving normal myocardium appearing black and areas of abnormal myocardium appearing relatively bright due to residual Gd in the tissue (hence the term “late gadolinium enhancement”).¹⁴⁻¹⁶ Myocyte necrosis results in loss of cell membrane integrity, allowing intracellular accumulation of Gd as occurs in LGE imaging of patients with MI.^{14,16-18}

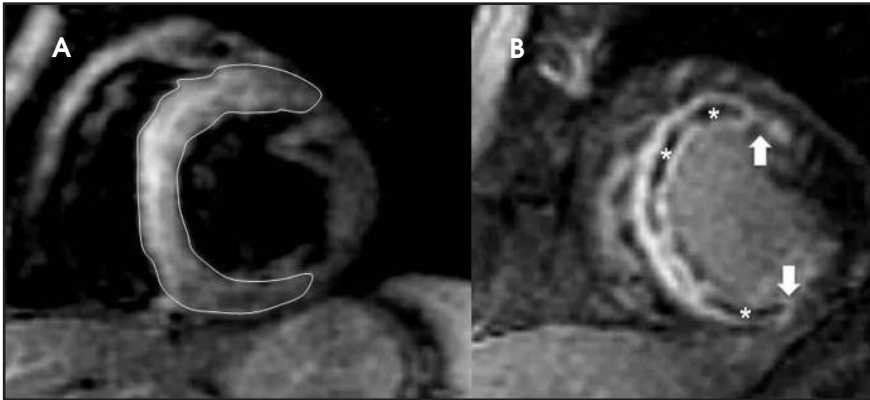


FIGURE 3. T2W CMR after acute myocardial infarction, shows a large area of myocardial edema as increased signal in the antero-septal wall (gray line, A). Late gadolinium enhancement image in the same patient (B) shows a large area of irreversible infarction (between the white arrows) with islands of low-signal within the infarct zone (*), signifying microvascular obstruction.

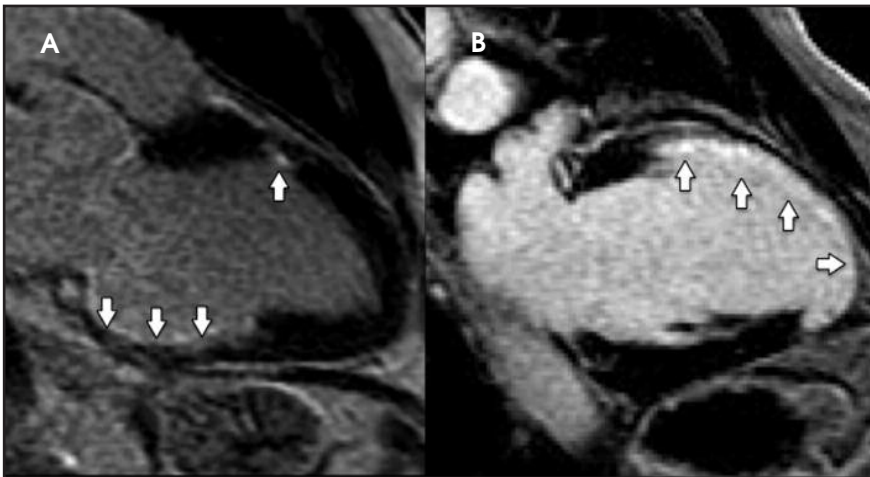


FIGURE 4. Late gadolinium enhancement in ischemic cardiomyopathy. Subendocardial infarction in the basal inferior wall (lower arrows, A) with a small separate infarct in the anterior wall (upper arrow). Transmural infarction in the anterior wall (arrows, B).

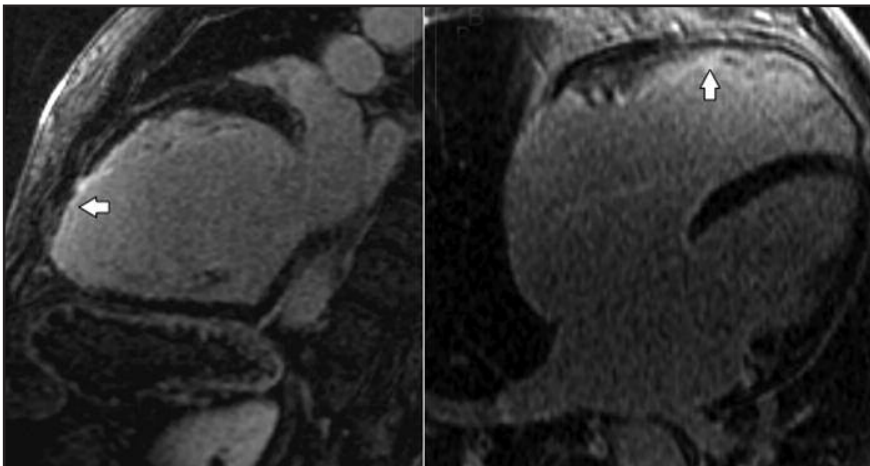


FIGURE 5. Extensive partial thickness LGE in the left ventricle is demonstrated (arrow, A) and in the right ventricle (arrow, B) in 2 patients with ischemic cardiomyopathy, severely dilated ventricles and poor systolic function.



Table 1. Common Indications and Contraindications for CMR in Patients with Heart Failure

Common Indications	Contraindications
<ul style="list-style-type: none"> • Accurate assessment of left ventricular ejection fraction for device implantation (ICD/CRT) • Myocardial viability (ischemic cardiomyopathies) • Detection of interventricular thrombus • Interstitial fibrosis (dilated and infiltrative cardiomyopathies) • Congenital heart disease • Right ventricular quantification • Evaluation for ARVD/C • Post cardiac transplantation surveillance • Constrictive pericarditis • Quantification of valvular dysfunction • Aortic and vascular measurement • Iron overload quantification (T2*) 	<p>Absolute</p> <ul style="list-style-type: none"> • Non-MR compatible implantable devices • Severe claustrophobia <p>Relative</p> <ul style="list-style-type: none"> • Dysrhythmia affecting ECG-gating • Severe renal impairment (risk of nephrogenic systemic fibrosis)

ICD = implantable cardiac defibrillator. CRT = cardiac resynchronization therapy, ARVD/C = arrhythmogenic right ventricular dysplasia/cardiomyopathy.

Table 2. Delayed Enhancement Patterns in CMR

Ischemic cardiomyopathies	<ul style="list-style-type: none"> • Subendocardial or full thickness enhancement in a typical coronary distribution (Figure 4)
Non-ischemic cardiomyopathies	<ul style="list-style-type: none"> • Mid-wall, often mild enhancement
Hypertrophic cardiomyopathy	<ul style="list-style-type: none"> • Patchy mid-wall enhancement in the areas of myocardial thickening • Faint “plexiform fibrosis” enhancement • Auto-infarction due to burnt-out disease (Figure 7E)
Sarcoidosis	<ul style="list-style-type: none"> • Patchy, often bright LGE in a non-coronary distribution
Myocarditis	<ul style="list-style-type: none"> • Mid-wall LGE in the lateral LV (Figure 7A)
Amyloidosis	<ul style="list-style-type: none"> • Thickened myocardium with diffuse LGE of relatively low signal, involving both ventricles and the atria.
Systemic auto-immune syndromes	<ul style="list-style-type: none"> • Subendocardial and/or subepicardial enhancement • Areas of full thickness LGE (Figure 7B)
Endomyocardial fibrosis	<ul style="list-style-type: none"> • Bright subendocardial enhancement in a shortened ventricle • Often with triangular-shaped LV apical thrombus (Figure 7F).
Iron overload	<ul style="list-style-type: none"> • Low signal on SSFP imaging • Quantification with T2*

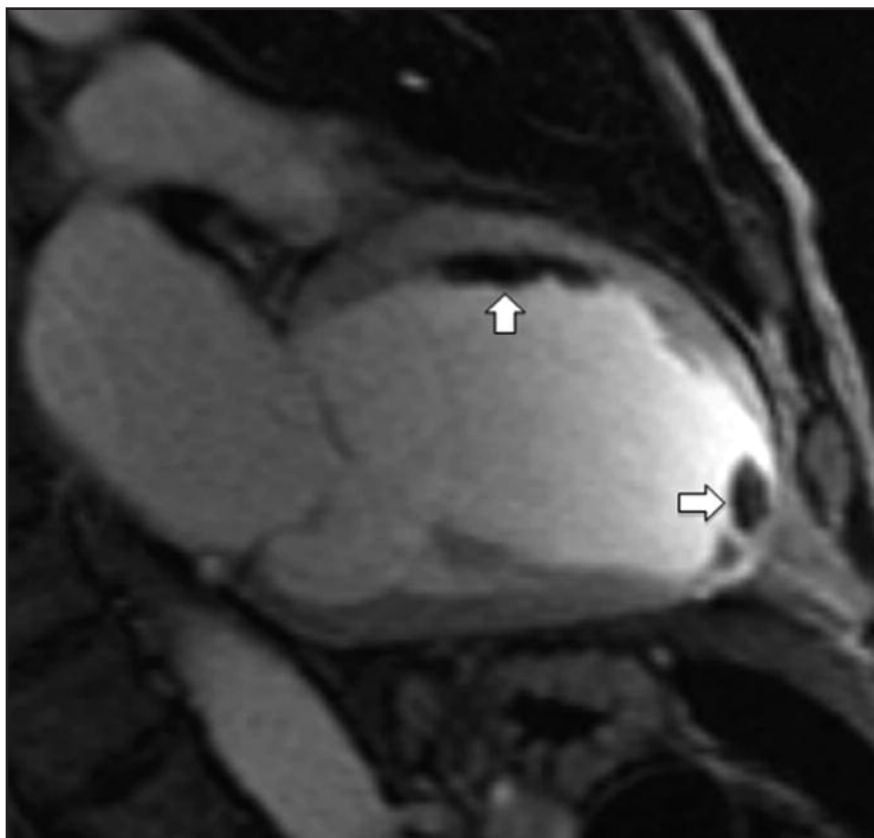
LGE = late gadolinium enhancement, SSFP = steady state free precession, LV = left ventricle

T2-weighted ‘edema’ imaging

T2-weighted (T2W) short-tau inversion recovery (STIR) imaging is a CMR sequence sensitive to increased myocardial water content, allowing the delineation of high-signal areas of

myocardial edema. Increased mobile water content associated with edema appears hyperintense on T2W-STIR images (Figure 3).¹⁹ Quantification of myocardial edema, using tissue signal thresholds, has been shown to strongly

correlate with ischemic time.²⁰⁻²² Myocardial edema may be present after any form of myocardial injury such as myocardial ischemia, acute infarction, myocarditis, sarcoidosis or trauma.¹⁹ CMR is able to assist in the



...a cleaner image with
the patient in mind.



FIGURE 6. Early-enhancement image demonstrating 2 separate mural thrombi (arrows).

diagnosis and surveillance of a range of cardiac disorders (Table 1).

Clinical role and indications for CMR *Ischemic cardiomyopathy*

Early studies show that increased signal in the myocardium following intravenous Gd accurately depicted irreversible ischemic myocardial injury independent of age.^{23,24} This was subsequently confirmed by others.²⁵ The presence and transmural extent of LGE following myocardial ischemia (Figures 3B and 4) gives important predictive information about the likelihood of functional recovery after revascularization.²⁶⁻²⁹ CMR is now considered the gold standard investigation for the assessment of myocardial viability.

The extent of LGE is also predictive of LV remodelling in patients with HF from both ischemic and non-ischemic causes.³⁰ Previous reports have shown that the presence of LGE can distinguish ischemic from non-ischemic

dilated cardiomyopathy with good sensitivity and specificity (Figure 5).^{31,32} However, patients with either severe left main or diffuse coronary disease may not have undergone infarction, and therefore may not exhibit ischemic-type LGE, despite large areas of ventricular hibernation. CMR with LGE combined with CT coronary angiography may improve the non-invasive detection of ischemia as a cause for heart failure with high sensitivity and specificity.³³ Stress CMR perfusion imaging may also have a role in this scenario.³⁴

Interstitial fibrosis

The presence of mid-wall fibrosis as demonstrated by LGE (Figure 5) has also been shown to correlate with a higher rate of all-cause mortality and hospitalization in patients with non-ischemic dilated cardiomyopathy (DCM).^{35,36} In a group of patients with DCM, the presence of mid-wall LGE occurred with poorer ejection fractions

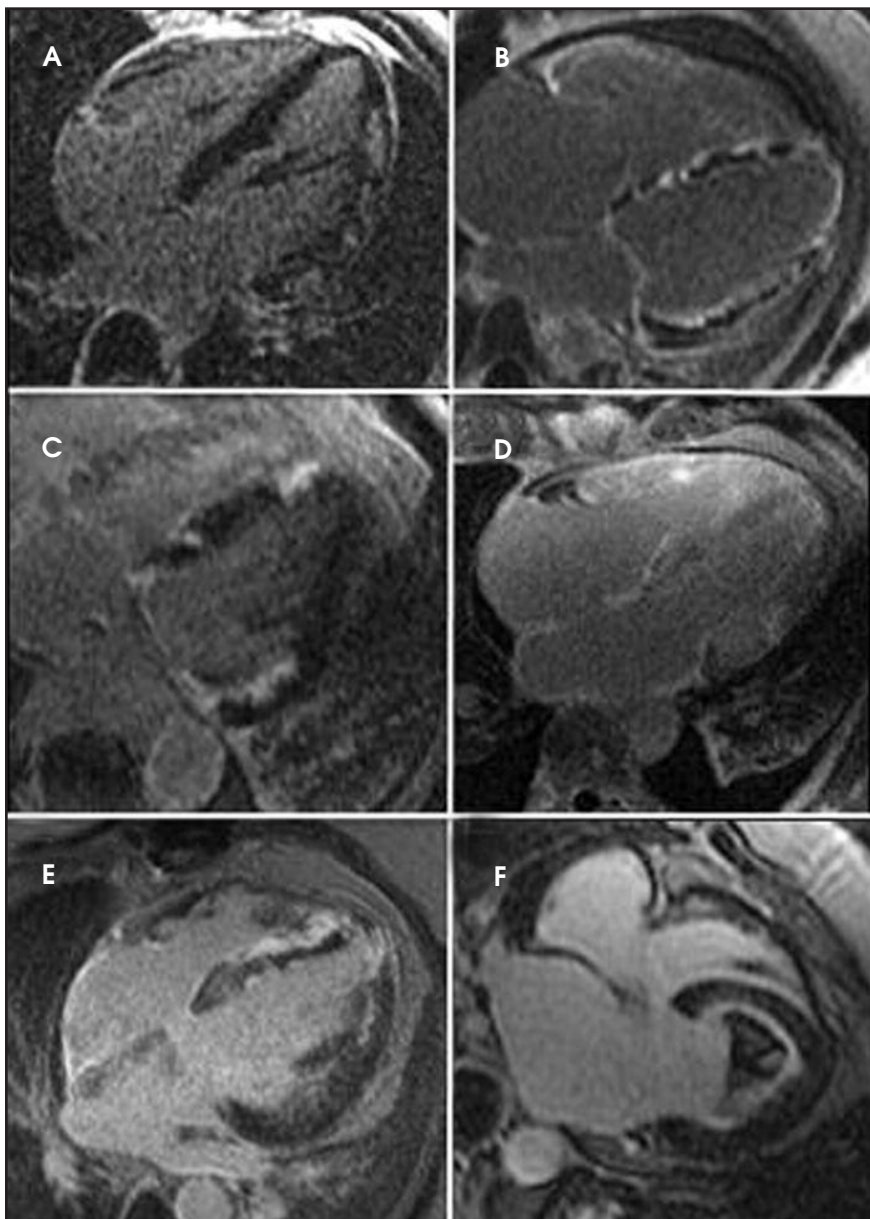


FIGURE 7. Infiltrative myopathies. Myocarditis (A): Mid-wall LGE in the lateral LV. Systemic lupus erythematosus/overlap syndrome (B): sub-endocardial, sub-epicardial and full thickness areas of LGE. Sarcoidosis (C): Patchy, bright LGE in a non-coronary distribution. Amyloid (D): Thickened myocardium with diffuse LGE of relatively low signal involving both ventricles and the atria, due to amyloid infiltration. Burnt-out hypertrophic cardiomyopathy (E): Patient with known HCM presenting with HF, showing focal areas of LGE. Endomyocardial fibrosis (F): Thick, sub-endocardial LGE is seen in a shortened ventricle, with a large triangular-shaped LV apical thrombus.

and larger volumes when compared with a group of patients with DCM and absence of mid-wall enhancement (Figure 6).³⁷ The extent of this interstitial fibrosis has also been assessed by the use of T1 mapping following intravenous Gd.³⁸ Interstitial fibrosis is a final common pathway for many patients suffering

myocardial damage from various etiologies.³⁹ Measurements of T2 signal intensity and relaxation times also correlate with biopsy-proven heart transplant rejection.⁴⁰ The degree of interstitial fibrosis on CMR also correlates with increased arrhythmic events in patients with hypertrophic cardiomyopathy.^{41,42}

Infiltrative cardiomyopathies

Infiltrative cardiomyopathies, which may present either with systolic or diastolic heart failure, arrhythmias or sudden cardiac death, can be difficult to diagnose with traditional imaging techniques. CMR provides accurate assessment of ventricular morphology and LGE, allowing imaging of abnormal areas of myocardium, with particular patterns of LGE correlating with the underlying infiltrative diagnosis.⁴³ Such conditions include sarcoidosis, hypertrophic cardiomyopathy, connective tissue diseases, endomyocardial fibrosis and amyloid infiltration (Figure 7).⁴⁴⁻⁴⁷ These conditions have patterns of LGE that may be characteristic, and assist in diagnosis, when combined with the clinical features and ventricular morphology (Table 2). Knowledge of such etiologies in patients presenting with HF may influence treatment decisions, such as a need for implantable defibrillator insertion, and provide an opportunity for disease-specific therapy. CMR can readily determine the extent of iron infiltration in thalassemia, hemochromatosis, and other states of iron overload, and quantify them with the use of the T2* sequence.⁴⁸

Intraventricular thrombus

CMR has advantages over echocardiography in the detection of intraventricular thrombi.⁴⁹ Following IV Gd, the blood pool signal is enhanced on T1W images and the signal from thrombus remains low (black). T1W images acquired immediately after administration of Gd can distinguish thrombus from surrounding myocardium or tumor. When imaging immediately after Gd contrast administration, myocardium or tumor demonstrate increased signal due to vascularity and perfusion, whereas thrombus, being avascular, remains dark. Left atrial appendage thrombus can also be seen on CMR, but the diagnostic accuracy of CMR in this area is yet to be determined.⁵⁰

Valvular dysfunction

The anatomic mechanisms and quantification severity can be assessed

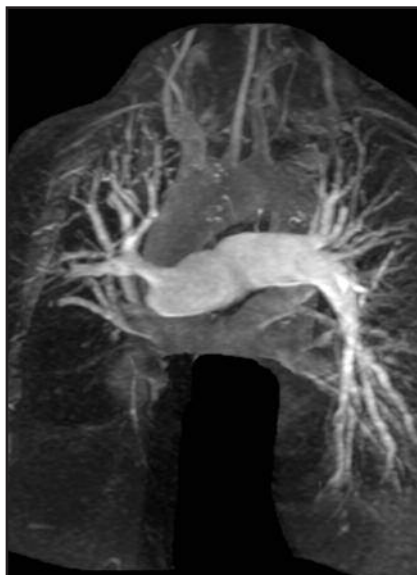


FIGURE 8. MR pulmonary angiography in chronic thromboembolic pulmonary hypertension. Image demonstrates severely abnormal vasculature with stenosis, vessel cut-off, webs and poor perfusion.

by CMR. Echocardiographic measurements of regurgitation may be inaccurate, particularly in dilated ventricles.⁵¹ The 3-dimensional volumetric nature of CMR helps overcome the problems with inhomogeneity and eccentricity of regurgitant jets.⁵² Comparison of LV stroke volume from volumetric LV measurements can be compared with aortic forward flow measurements using phase contrast flow imaging in the ascending aorta. The regurgitant volume can readily be calculated.⁵³⁻⁵⁵ Regurgitant orifice area, most often calculated by the proximal isovelocity surface area (PISA) method in echocardiography, can also be performed by CMR.⁵⁶ The CMR measurement of anatomic orifice area, however, has not been prospectively evaluated to provide equivalent prognostic information to echocardiography.

Similar CMR techniques can be used for quantification of tricuspid, aortic and pulmonary regurgitation. In particular, phase-contrast flow quantification across the semilunar valves offer an accurate measurement of forward and backward flow,⁵⁷ and may offer superior reproducibility to echocardiography.

Diastolic function

Echocardiography is the best-established non-invasive technique for the evaluation of diastolic dysfunction.⁵⁸ Cardiovascular MR analysis of ventricular filling velocity, 3-dimensional myocardial strain analysis and real-time CMR tissue tagging are promising methods to assess regional diastolic function.⁵⁹ Cardiovascular MR using phase contrast flow imaging is capable of measuring flow across the mitral valve and in pulmonary veins. Measurements of mitral A-wave and E-wave velocity and deceleration times, and systolic and diastolic wave velocities in the pulmonary flow traces, have been shown to be reliable and easy to obtain.⁶⁰ Cardiovascular MR measurements show good correlation with echocardiographic measurements in limited numbers of patients with normal and abnormal ventricles.^{60,61}

Arrhythmogenic RV dysplasia

CMR provides the best imaging technique available for assessment of RV free wall contraction abnormalities and is particularly valuable in assessing patients with suspected arrhythmogenic RV dysplasia (ARVD).^{62,63} Regional wall motion abnormalities are more difficult to interpret in the RV than in the LV because of the structure of the RV free wall. Exaggerated diastolic distortion of the RV free wall, or the “accordion sign,” is also associated with genotype-positive ARVD.⁶⁴ However, some systolic distortion of the RV, due to contraction of the moderator band and insertion of the trabeculae into the thin RV free wall, may be present in normal patients. There is also growing recognition of LV involvement in ARVD, which can be imaged by CMR.⁶² Identification of fatty infiltration of the RV wall by CMR can be supportive of a diagnosis of ARVD, but is not a reliable sign due to the thinness of the RV wall. It should be noted that fatty infiltration, as defined in the current diagnostic criteria, is a histological diagnosis not an imaging diagnosis, and the prevalence of RV fat by CMR in the normal population has not been fully elucidated.

Dyssynchrony

Improved temporal resolution ECG-gated SSFP images allow approximately 40 frames per heart beat to be obtained. In the 4-chamber view, the LV lateral wall and septal contractility can be carefully evaluated frame-by-frame in relation to tricuspid and mitral valve opening as well as atrial contraction. Cardiac MR techniques using SSFP imaging show promise in the evaluation of mechanical dyssynchrony.⁶⁵ The improved spatial resolution of CMR, combined with high temporal resolution or real-time imaging, may offer improved reproducibility for the assessment of dyssynchrony, which has proven difficult in randomized echocardiographic trials of dyssynchrony quantification.⁶⁶⁻⁶⁸ The ability for CMR to predict responders to cardiac resynchronization therapy is an area of ongoing research. It has been well demonstrated, however, that patients with a large volume of myocardial scar, as determined by CMR, respond poorly to biventricular pacing.⁶

Constriction and constrictive pericarditis

Constrictive pericarditis is notoriously difficult to diagnose, often with subtle echocardiographic and invasive hemodynamic findings. CMR provides an accurate assessment of pericardial thickness, using double and triple inversion recovery sequences, as well as being able to non-invasively demonstrate ventricular interdependence. Real-time imaging allows assessment of the ventricular interdependence and abnormal septal motion seen in constrictive physiology. This is done in the LV short axis view with images, including the domes of the diaphragm, in order to observe respiratory motion.

Magnetic resonance angiography

The anatomy of the great vessels and branches are exquisitely shown on CMR, particularly with contrast-enhanced 3-dimensional magnetic resonance

angiography (3D-MRA). Lack of ionizing radiation and high reproducibility make 3D-MRA useful for longitudinal follow-up of patients with vascular abnormalities, particularly surgical shunts or dissection. MR pulmonary angiography is also useful in evaluation and surgical planning for chronic thromboembolic disease (Figure 8).⁷⁰

In spite of its advantages, CMR does have some limitations in patients with dysrhythmias that affect ECG-gating, claustrophobia, implantable devices, and severe renal impairment. Parallel imaging has improved acquisition time, and automated software has reduced analysis time, however CMR remains a specialized technique requiring considerable expertise for both acquisition and interpretation.

Conclusion

CMR uniquely provides accurate and reproducible measures of volumes and function of all 4 cardiac chambers and surrounding vasculature. It provides excellent morphological information with unparalleled definition between blood pool and myocardium. Combined with the known patterns of LGE, CMR provides a powerful tool for the diagnosis and quantification of myocardial infarction. It provides prognostic information prior to either revascularization or ventriculoplasty. Non-ischemic patterns of LGE may reveal infiltrative conditions that are often difficult to diagnose with other techniques, and which may significantly alter clinical management. CMR is an ideal technique to evaluate complications such as intracardiac thrombus or valve dysfunction. It has significant advantages in evaluation of the RV, which is increasingly recognized as an important and prognostic factor in HF and congenital heart disease.

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