Delayed Enhancement Magnetic Resonance Imaging in Nonischemic Myocardial Disease

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Abstract: This review highlights the role of delayed enhancement magnetic resonance imaging for the diagnosis of patients with nonischemic myocardial disease. The authors discuss the use of delayed enhancement for differentiation between ischemic and nonischemic myocardial disease and for narrowing the differential diagnosis when nonischemic etiologies are suspected. In addition, special focus is given to the prognostic applications of delayed enhancement magnetic resonance imaging.

Key Words: delayed enhancement, nonischemic myocardial disease, magnetic resonance imaging

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LEARNING OBJECTIVES

After completing this CME-SAM activity, physicians should be better able to:

1. Differentiate ischemic from nonischemic patterns of myocardial delayed enhancement.
2. List the most common myocardial regions that present with delayed enhancement in patients with hypertrophic cardiomyopathy.
3. Describe how to optimize delayed enhancement imaging in patients with cardiac amyloidosis.
4. Learn the prognostic implication of the presence of myocardial delayed enhancement in patients with myocarditis.

INTRODUCTION

Delayed enhancement is a powerful magnetic resonance imaging (MRI) technique that is largely used in cardiac imaging for the evaluation of both ischemic and nonischemic cardiomyopathies. The role of delayed enhancement in the diagnosis and prognosis of patients with nonischemic myocardial disease has been the topic of several recent publications. Delayed enhancement imaging is a key tool in the differentiation between ischemic and nonischemic cardiomyopathies. Further, the patterns and extent of myocardial delayed enhancement help narrow the differential diagnosis and aid in the prognostic assessment of this group of myocardial diseases. Therefore, myocardial delayed enhancement has become an essential tool for guiding clinical management in patients with nonischemic myocardial diseases.

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MECHANISM

Delayed enhancement MRI studies are performed with an inversion-recovery turbo field echo sequence 10 to 15 minutes after the administration of gadolinium chelate. Gadolinium-based contrast agents shorten the T1 relaxation time of tissues with increased gadolinium content. Gadolinium chelate contrast agents pass through a chelation process, which prevents them from crossing the preserved cell membranes, resulting in contrast distribution restricted to the extracellular space.[1,2] Conditions that increase the extracellular myocardial space will therefore show increased signal in delayed enhancement studies. Examples of such conditions include myocardial inflammation, infiltrative diseases, and fibrosis.[3] In addition, the loss of intact cell membranes in an acute myocardial infarction (AMI) allows the gadolinium chelate to distribute within the intracellular space, which increases the overall volume of distribution of the contrast media and generates increased signal on delayed enhancement MRI.

A T1-weighted inversion-recovery turbo field echo sequence is used to optimize both signal-to-noise and contrast-to-noise ratios between gadolinium-retaining infarcted/scar tissue and healthy or viable myocardium.[4] This technique requires identification of the optimal inversion time (TI) to null the MRI signal from the normal myocardium, which will appear "dark" on delayed enhancement images. Nulling the signal of the normal myocardium allows for a more precise delineation between the gadolinium-retaining areas (abnormal) and the normal/preserved myocardium.

TECHNIQUES

Optimizing Image Acquisition

TI is the most important imaging parameter for obtaining high-quality images and contrast between normal and enhancing myocardium. The typical TI to null the signal for the normal myocardium is between 250 and 300 ms when images are obtained 10 to 15 minutes after gadolinium injection (Fig. 1). The need for an ideal TI is less important when a phase-sensitive delayed enhancement sequence is available.[5] This sequence uses a baseline phase image that is added to a magnitude image to generate a high-contrast final image, which will depict normal myocardium with low signal even when the TI is not ideal (Fig. 2). Although early studies of the delayed enhancement technique used a delay of 10 to 15 minutes, a recent study by Wagner et al[6] demonstrated a high level of agreement between delayed enhancement and histopathology for delineating myocardial infarcts when images were obtained 5 to 40 minutes after gadolinium administration. For

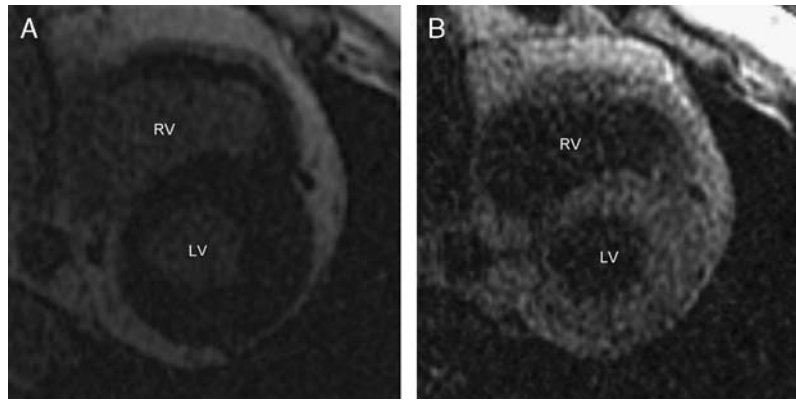

FIGURE 1. TI selection. Delayed enhancement images in the short-axis plane obtained with different TIs are shown. Note the nulled myocardial signal in the acquisition with 300 ms (A). When a TI of 150 ms was used (B), the myocardium does not show appropriate nulling.

optimal imaging contrast, the TI of the normal myocardium must be adjusted for the delay after gadolinium injection, as this parameter increases with longer delay time. It has also been shown that a single dose of gadolinium (0.1 mmol/kg) can generate very similar image quality and accuracy for scar detection when compared with a double dose (0.2 mmol/kg), which was used in the original studies describing this technique.

Other imaging parameters can also be adjusted to optimize image quality in delayed enhancement imaging. As with any other MRI technique, a balance between spatial resolution and scan time is the goal. A short flip angle and a smaller possible field of view are key features in this imaging technique, particularly for large patients. Suggested imaging parameters are shown in Table 1.

Quantification

Quantification of the delayed enhancement area and percentage of myocardial volume with scar has been used in recent clinical trials as predictors for functional recovery, arrhythmias, and cardiac death. Therefore, tools for semiautomatic quantification of delayed enhancement were recently made commercially available.

Definition of abnormal signal intensity has been a subject of controversy, and there are 2 widely used definitions. Schmidt et al[7] defined abnormal enhancement of the myocardium as an area of signal intensity higher than half peak enhancement. The other definition relies on the intensity of the normal or remote myocardium. The border zone of delayed enhancement is defined as any myocardial tissue with signal enhancement between 2 and 3 SD from the remote, whereas the infarct core is defined as intensity higher than 3 SD from the remote.[8] Semiautomatic software typically uses 1 of these 2 methods for definition of the abnormal myocardium enhancement threshold. Alternatively, the user can manually delineate the infarct area.

CLINICAL APPLICATIONS

Delayed enhancement MRI has an important role in establishing the diagnosis of ischemic versus nonischemic myocardial disease. In addition, several recent studies have demonstrated a prognostic implication for the presence and extent of delayed enhancement in multiple disease processes. The use of delayed enhancement MRI for risk stratification and prognostic characterization established this method as a key component for guiding management of patients with ischemic and nonischemic myocardial diseases.

The differentiation between ischemic and nonischemic cardiac diseases is essential for guiding clinical management. The pattern and distribution of the abnormal areas of

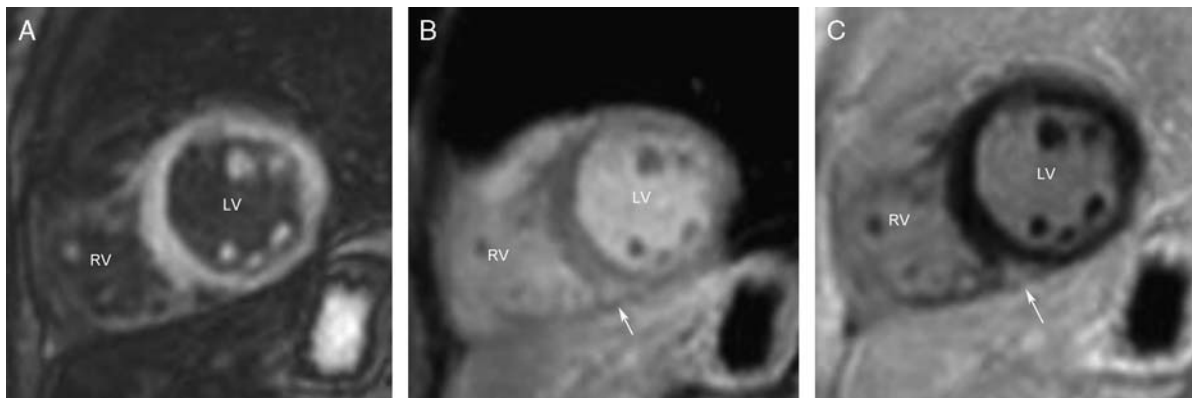

FIGURE 2. Phase-sensitive delayed enhancement imaging in a patient with HCM. Three short-axis images from a phase-sensitive delayed enhancement acquisition are shown, including the baseline phase image (A), the magnitude image (B), and the final image (C). Note how the focal delayed enhancement in the posterior junction (arrows) is better depicted in the final image, as opposed to the original magnitude image.

TABLE 1. Suggested Approximate Parameters for Delayed Enhancement Imaging Acquisition

TI	250-300 ms
Repetition time	4.0 ms
Echo time	2.0 ms
Field of view	32-36 cm^2
Flip angle	15-20 degrees
Slice thickness	6 mm
Number of excitations	1
In plane spatial resolution	190 (frequency) 160 (phase)

delayed enhancement is essential for making this differentiation.[9] The expected pattern of delayed enhancement in patients with ischemic heart disease follows a coronary artery territory distribution. The delayed enhancement initially involves the subendocardial region and can progress to a transmural abnormality.[10] Patchy or diffuse delayed enhancement that does not follow a coronary artery distribution and primarily involves the midwall and/or subepicardial regions of the left ventricular (LV) myocardium (Fig. 3) is a characteristic finding of nonischemic cardiomyopathies[11] (Table 2).

DELAYED ENHANCEMENT PATTERNS IN NONISCHEMIC MYOCARDIAL DISEASES

Myocarditis

According to Angelini et al,[12] the clinical presentation of myocarditis typically mimics AMI in patients with angiographically normal coronary arteries. The use of delayed enhancement MRI for distinguishing between acute myocarditis and AMI has been previously reported.[13] The standard of reference for the differentiation between these 2 entities is endomyocardial biopsy, but delayed enhancement MRI plays an important role as a complementary technique for the evaluation of patients with suspected cardiomyopathies.[14] In contrast with the typical ischemic pattern of delayed enhancement, myocarditis tends to show patchy or diffuse nodular delayed enhancement, predominantly involving the subepicardial or midwall regions of the LV, in a noncoronary territory distribution (Fig. 4). Recently, Lurz et al[15] described the accuracy of delayed enhancement MRI for assessment of myocardial inflammation/myocarditis with a sensitivity of 69% and a specificity of 46%.

A recent publication including 222 patients with biopsy-proven myocarditis demonstrated that the presence of delayed enhancement is an independent predictor for both all-cause mortality and cardiac mortality, with hazard ratios of 12.8 and 8.4, respectively.[16] The presence of delayed enhancement has also been described as a common feature in patients with chronic heart failure due to myocarditis, supporting the notion that abnormal enhancement is associated with worse clinical disease.[17]

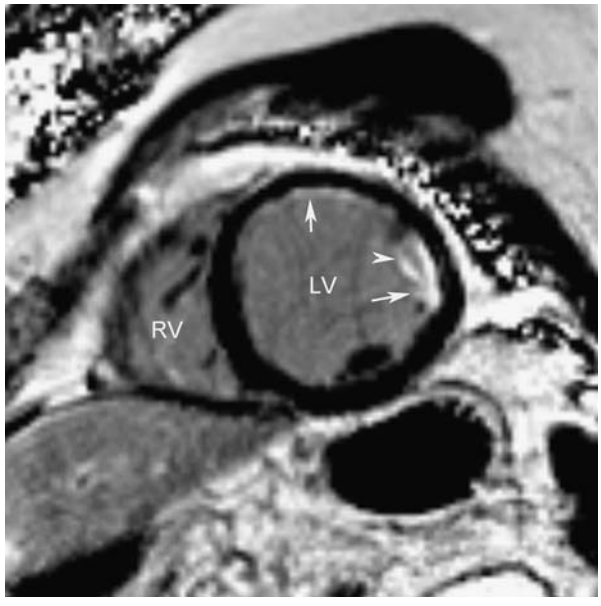

FIGURE 3. Ischemic versus nonischemic delayed enhancement. Short-axis delayed enhancement image in a patient with an old myocardial infarction demonstrates the typical subendocardial delayed enhancement (arrows) in the anterior and lateral LV walls. Horizontal long-axis image from a different patient with cardiac sarcoidosis shows subepicardial (arrows) and midwall (arrowhead) delayed enhancement.

TABLE 2. Patterns of Delayed Enhancement in Nonischemic Heart Disease

Disease	Transmural Distribution	Preferred Region Involved
Myocarditis	Subepicardial or midwall	Variable
Dilated cardiomyopathy	Midwall	None
Hypertrophic cardiomyopathy	Midwall or subepicardial	Areas of most severe hypertrophy Ventricular-septal connection points
Sarcoidosis	Subepicardial or midwall	RV side of the septum Basal anterolateral and anteroseptal
Amyloidosis	Subendocardial, midwall, or subepicardial	Global (when subendocardial)
Arrythmogenic right ventricular dysplasia	Transmural, subepicardial, or subendocardial	RV free wall RV side of the septum
Anderson-Fabry	Midwall	Basal inferolateral
Chagas	Transmural or subepicardial	Apical Basal inferolateral
Churg-Strauss	Subendocardial, possibly midwall, or subepicardial	Ventricular septum
Lyme	Midwall	Basal anteroseptal
Endomyocardial fibrosis	Subendocardial	None

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Dilated Cardiomyopathy

Dilated cardiomyopathy can be ischemic or nonischemic in etiology. This myocardial disorder typically presents with ventricular dilatation and reduced systolic function, which results in congestive heart failure and arrhythmias. Nonischemic dilated cardiomyopathy is most

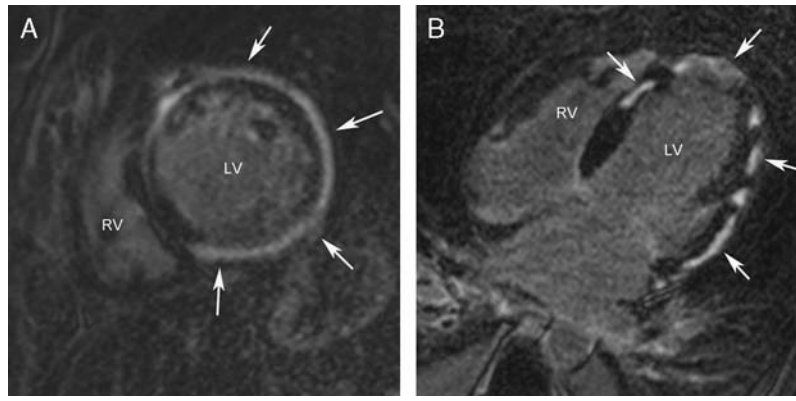

FIGURE 4. Myocarditis. Delayed enhancement images in the short axis (A) and horizontal long axis (B) in a patient with viral myocarditis. Note the extensive enhancement (arrows) in the midwall of the septum, in the subepicardial layer of the lateral LV wall, and apex of the RV.

commonly idiopathic in etiology. However, identifiable causes include alcohol abuse, drug toxicity, obesity, and chronic hypertension.

Typical MRI findings of dilated cardiomyopathy include ventricular enlargement and a reduced ejection fraction. Delayed enhancement is the key imaging method to distinguish between dilated cardiomyopathy secondary to ischemic disease and other etiologies.[18] Lack of delayed enhancement or the presence of midwall LV delayed enhancement in a noncoronary distribution strongly suggests a nonischemic etiology[11] (Fig. 5). McCrohon et al[19] reported that 59% of patients with dilated cardiomyopathy who had normal coronary arteries at conventional angiography showed no myocardial delayed enhancement, whereas 28% of patients showed a midwall distribution of delayed enhancement consistent with a nonischemic etiology.

The presence of delayed enhancement also has a prognostic implication in patients with dilated cardiomyopathy. Assomull et al[20] demonstrated the presence of midwall fibrosis in 35% of patients with dilated cardiomyopathy, which was associated with a higher rate for the predefined primary combined endpoint of all-cause death and hospitalization for a cardiovascular event (hazard ratio 3.4, $P = 0.01$), independent of ventricular remodeling. The presence of midwall fibrosis by delayed enhancement MRI was also considered a predictor of sudden cardiac death/ventricular tachycardia.

Hypertrophic Cardiomyopathy (HCM)

HCM is a disorder characterized by extensive disarray of myocardial fibers and regional fibrosis. Delayed enhancement in patients with HCM seems to be related to ischemic injury due to myocardial collagen replacement.[21] It was also hypothesized by Kuribayashi et al[22] that the areas of myocardial disarray, which are characteristically seen in HCM at the junction of the ventricular septum and the right ventricular (RV) free wall, could also lead to increased extracellular distribution of the contrast medium and consequently to delayed enhancement. HCM is typically asymmetric and primarily involves the basal ventricular septum, but atypical patterns with prominent hypertrophy in the mid or apical segments can be seen.

Delayed enhancement is seen in approximately 81% of patients with HCM and typically involves the anterior and posterior junction of the RV free wall with the ventricular septum[23] (Fig. 6). Usually, the pattern of delayed enhancement shows a midwall or subepicardial distribution sparing the subendocardial region in a noncoronary

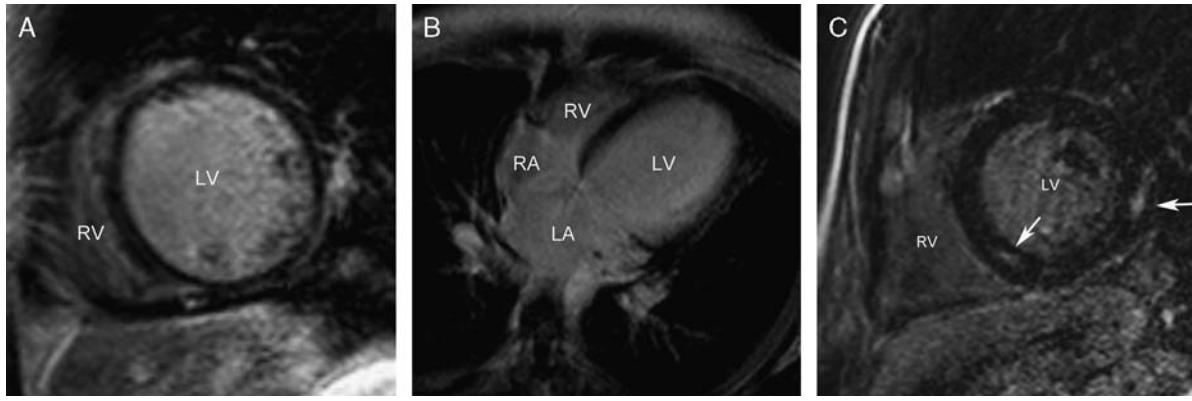

FIGURE 5. Nonischemic dilated cardiomyopathy. Short-axis (A) and horizontal long-axis (B) delayed enhancement images from a patent with idiopathic dilated cardiomyopathy demonstrate complete nulling of the normal myocardium. Note dilatation of the LV. Delayed enhancement image in the short-axis plane is also shown from a different patient with nonischemic dilated cardiomyopathy. The arrows point to regions of midwall enhancement in the inferoseptal and inferolateral LV walls (C). LA indicates left atrium; RA, right atrium.

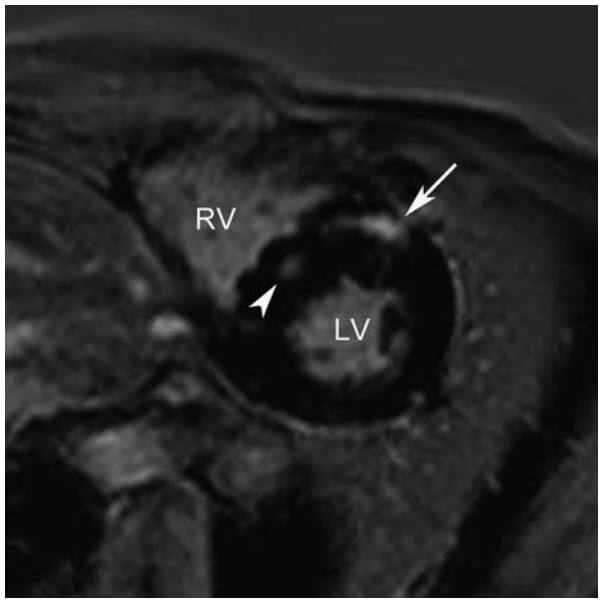

FIGURE 6. Asymmetric HCM. Short-axis delayed enhancement image from a patient with HCM shows enhancement in the anterior junction of the RV free wall with the ventricular septum (arrow) and in the midwall of the LV septum (arrowhead).

territory distribution. The amount of delayed enhancement tends to be greater in the regions of most severe hypertrophy (eg, the asymmetrically hypertrophied basal ventricular septum) (Fig. 7).

Recent studies have demonstrated a significant role for delayed enhancement MRI as a prognostic indicator in patients with HCM. It has been shown that patients with HCM and any degree of delayed enhancement have a 7-fold higher risk for nonsustained ventricular tachycardia on Holter monitoring compared with patients without evidence of delayed enhancement.[24] In 2009, Satoh et al[25] demonstrated that the presence of delayed enhancement is not only related to ventricular arrhythmias but is also associated with a higher New York Heart Association functional class, impaired global LV function, conductance disturbance, abnormal Q waves, and giant T waves. Bruder et al[26] demonstrated that the presence of a scar detected by delayed enhancement in MRI is an independent predictor of all-cause mortality as well as of cardiac mortality in HCM patients (odds ratio of 5.47 for all-cause and 8.01 for cardiac mortality) and that the presence of delayed enhancement could be useful for noninvasive risk stratification in asymptomatic and mildly symptomatic HCM patients. In 2012, Green et al[27] in a meta-analysis of 4 MRI studies also reported that the presence of delayed enhancement on MRI of patients with HCM is a significant predictor of all-cause mortality and cardiac mortality. In addition to the presence of delayed enhancement, the extent of myocardial involvement has been associated with progressive disease and is considered a marker of poor outcome. Another study[21] has shown that patients with progressive HCM have a higher extent of delayed enhancement (approximately 28.5%) compared with patients with stable disease (8.7%).[28]

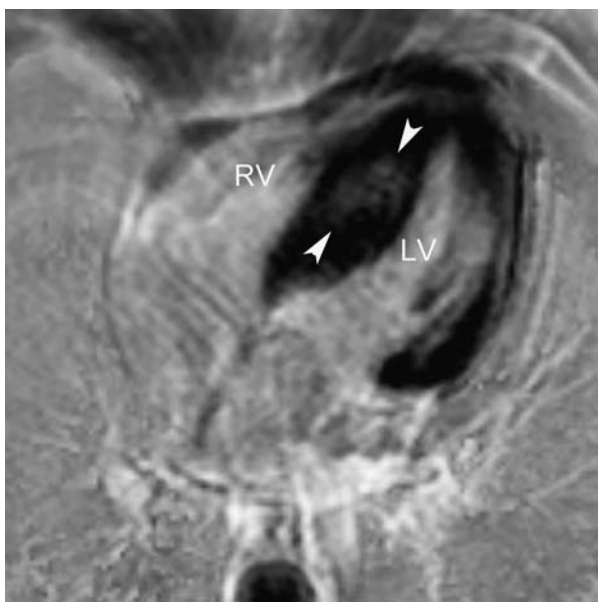

FIGURE 7. Asymmetric HCM. Horizontal long-axis delayed enhancement image from a patient with asymmetric HCM. Note the predominant delayed enhancement (arrowheads) in the most severely hypertrophied area of the ventricular septum.

Cardiac Amyloidosis

Cardiac involvement in amyloidosis is common and typically presents as restrictive cardiomyopathy. Diastolic dysfunction is caused by the deposition of proteinaceous amyloid material in the myocardial wall and may cause the impairment of diastolic filling and consequently diastolic heart failure in late stages of the disease.[18]

The most characteristic imaging pattern in cardiac amyloidosis is a diffuse subendocardial delayed enhancement, which helps in the differential diagnosis with other cardiomyopathies.[29] This "amyloid pattern" has been described in approximately 70% of patients with biopsy-proven cardiac amyloidosis.[30] Another characteristic feature of cardiac amyloidosis is the involvement of the right atrial wall, which can appear with thick enhancing walls on delayed enhancement imaging (Fig. 8).

When performing a T1 inversion-recovery sequence with delayed enhancement MRI images, the establishment of the optimal TI to null the signal of the normal myocardium can be problematic and misleading, resulting in the equivocal inversion of the signal from the abnormal enhancing myocardium (Fig. 9). Therefore, a sequence with multiple TIs called the "TI scout" aids in differentiating between normal and abnormal myocardium. Generally, in normal hearts or in other cardiac diseases, the blood pool shows a higher concentration of gadolinium-based contrast media and a shorter T1 relaxation time compared with the myocardium. Therefore, the blood pool nulls at a shorter TI compared with the myocardium. One distinguishable feature of cardiac amyloidosis reported by Maceira et al[30] is that areas with amyloid deposition in the myocardium typically demonstrate a TI shorter than or equal to that of the blood pool, which can be appreciated on the "TI scout" (Fig. 10). It has also been shown that the delayed enhancement images acquired 5 to 8 minutes after gadolinium administration provide superior contrast between normal and abnormal myocardium in patients with amyloidosis when compared with the traditional scan delay of 10 to 15 minutes.[30]

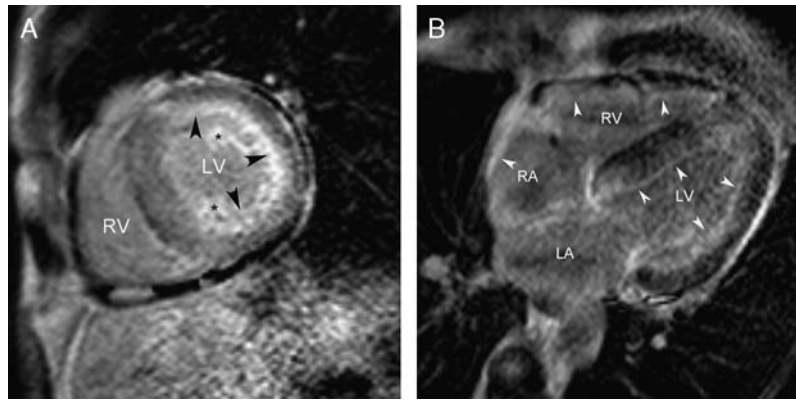

FIGURE 8. Cardiac amyloidosis. Short-axis delayed enhancement image (A) from a patient with cardiac amyloidosis demonstrates global subendocardial delayed enhancement (arrowheads) in the LV—the typical amyloid pattern of distribution. Horizontal long-axis image (B) from the same patient demonstrates global subendocardial delayed enhancement in the LV, RV, and right atrium (RA) (arrowheads). Note the enhancement of the papillary muscles (*), commonly seen with cardiac amyloidosis. LA indicates left atrium.

Selvanayagam and colleagues reported that the presence of global late gadolinium enhancement was associated with more severe interstitial cardiac amyloid deposition.[31] They also concluded that there would be a biologically plausible association between the degree of late gadolinium enhancement and markers of disease severity (New York Heart Association functional class, cardiac mass and ejection fraction on cardiovascular magnetic resonance, electrocardiographic parameters, and cardiac biomarkers).[31]

Cardiac Sarcoidosis

Only a minority of patients with sarcoidosis demonstrate clinically evident cardiac involvement, which most commonly presents as dilated cardiomyopathy and/or arrhythmias.[32] Cardiac involvement has a reported prevalence of 7% in patients with pulmonary sarcoidosis.[33] However, 20% to 30% of postmortem examinations revealed cardiac involvement in sarcoidosis.[34,35] Patel et al[36] demonstrated the presence of abnormal delayed enhancement on MRI in 26% of 81 patients with biopsy-proven extracardiac sarcoidosis compared with only 12% of patients with cardiac involvement diagnosed by standard clinical criteria.

The reported prevalence of delayed enhancement on MRI of patients with documented cardiac sarcoidosis varies from 42% to 91%.[37,38] Abnormal enhancement can be seen in areas of myocardial inflammation and focal areas of scarring in patients with cardiac sarcoidosis.[32] Patients with myocardial sarcoidosis most commonly show patchy areas of delayed enhancement in the mid myocardial wall or in the subepicardial region, generally sparing the subendocardium. The findings do not follow a coronary territory distribution, which helps in the differential diagnosis with ischemic heart disease. It has been reported by different authors that delayed enhancement in sarcoidosis involves predominantly basal segments of the LV, with the anteroseptal and anterolateral segments most frequently affected. Delayed enhancement of the RV side of the septum is considered to be a characteristic feature[39] (Fig. 11). Delayed enhancement of the RV free wall occurs in sarcoidosis and may mimic the features of arrhythmogenic RV dysplasia (ARVD). The 2 entities may be distinguished using ^{18}F-fluorodeoxyglucose-positron emission tomography, which typically demonstrates intense ^{18}F-fluorodeoxyglucose uptake in the free wall of the RV in patients with sarcoidosis (Fig. 12).

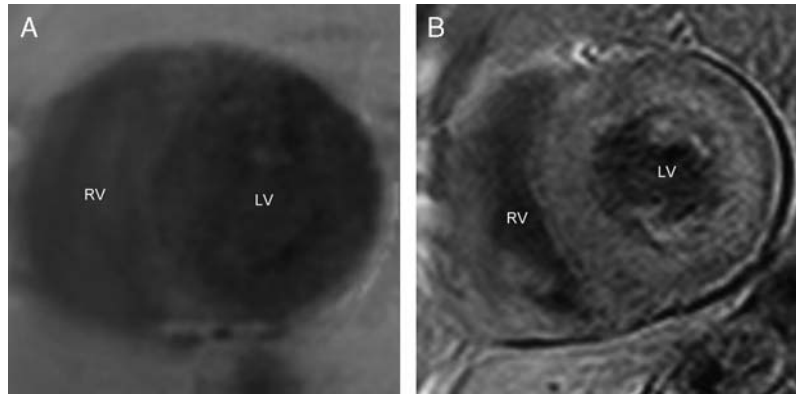

FIGURE 9. Cardiac amyloidosis. Short-axis delayed enhancement images from a patient with cardiac amyloidosis. Initial acquisition (A) demonstrates nulling of the signal of the myocardium in the LV and RV at the same TI as the blood pool. An additional delayed enhancement image was obtained with a longer TI (B) showing global delayed enhancement in the ventricles, predominantly in the subendocardial region, corresponding to areas of amyloid deposition.

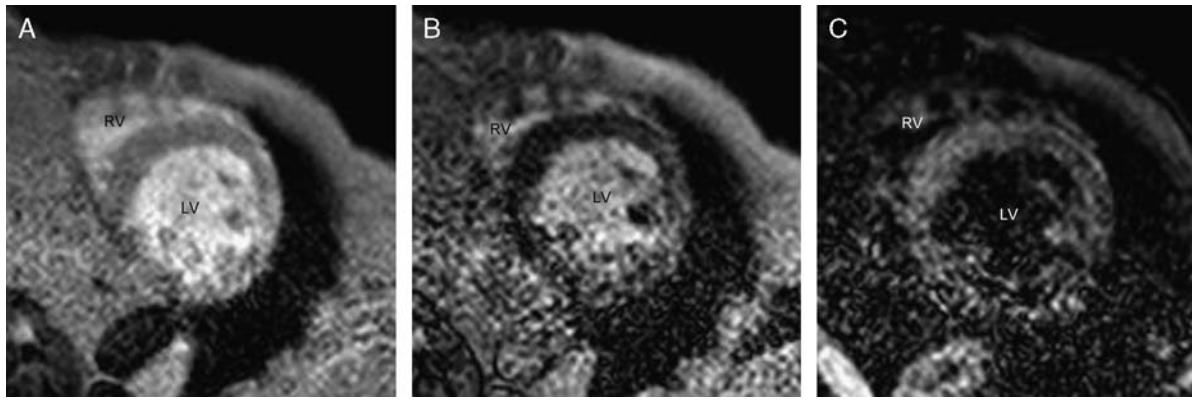

FIGURE 10. Cardiac amyloidosis. Three short-axis images from a TI scout series obtained at 200 (A), 300 (B), and 350 ms (C). The initial image (A) shows increased signal on both the blood pool and the myocardium. At 300 ms (B), the signal from the myocardium is nulled, whereas the blood pool signal remains. At 350 ms, the nulling time of the blood pool is achieved. Myocardial TI shorter than that of the blood pool is characteristic of patients with diffuse cardiac amyloidosis.

ARVD

ARVD is characterized histologically by fat and/or fibrous tissue replacement of normal myocytes, particularly in the RV, but it can also involve the LV in later stages.[40,41] Tandri et al[42] have reported that delayed enhancement has an excellent correlation with the histopathologic diagnosis of fibrofatty infiltration in patients with ARVD and was present in approximately 60% of patients with confirmed ARVD[42] (Fig. 13). In a small series of patients with ARVD, delayed enhancement was reported in 7 (88%) of 8 patients with histology-proven ARVD.[43] The delayed enhancement was not restricted to the RV free wall but involved the right side of the interventricular septum as well and was associated with regional contraction abnormality in most of the patients. Depiction of abnormal delayed enhancement in a thin RV wall can be challenging, and therefore special attention should be given to areas of abnormal contractility.

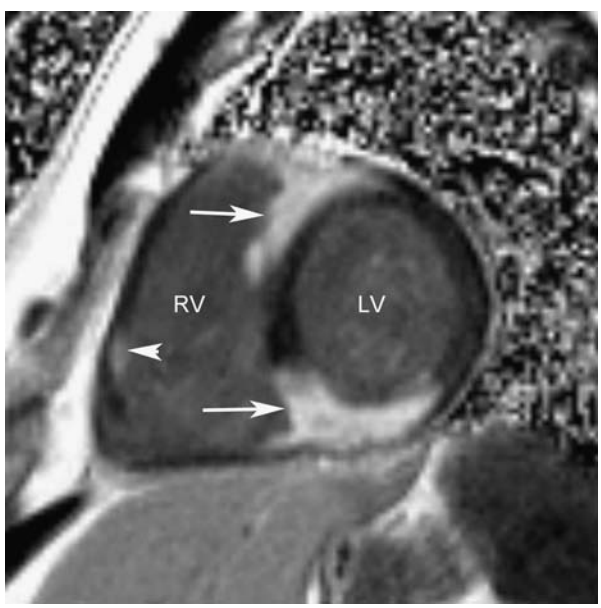

FIGURE 11. Cardiac sarcoidosis. Short-axis delayed enhancement image of a patient with cardiac sarcoidosis demonstrates a transmural high signal predominantly in the basal anteroseptal and inferoseptal segments of the LV (arrows). Note the involvement along the RV side of the septum and in the RV free wall (arrowhead).

The presence of delayed enhancement on MRI is not part of the new Task Force Criteria for the diagnosis of ARVD, which relies primarily on the presence of an enlarged RV with abnormal contractility.[44] Nevertheless, it has been described that the presence of RV delayed enhancement is predictive of inducible ventricular tachycardia at programmed electric stimulation, suggesting a possible role of viability imaging in the prognostic assessment of patients with ARVD.[42]

Systemic Sclerosis

Systemic sclerosis is a connective tissue disorder with widespread microvascular damage and multiorgan involvement, including the heart. Although the prevalence of clinical cardiac abnormalities in patients with systemic sclerosis has been a subject of debate, contraction band necrosis or focal myocardial fibrosis was identified in 23 of 52 cardiac autopsy specimens of patients with systemic sclerosis.[45] Tzelepis et al[46] described the presence of myocardial delayed enhancement in 66% of patients with systemic sclerosis without cardiac symptoms. The most commonly identified delayed enhancement pattern was linear and involved the midwall, sparing the subendocardium and the epicardium, in a noncoronary artery distribution. Kobayashi et al[47] demonstrated that abnormalities on stress perfusion MRI of asymptomatic patients with systemic sclerosis are frequently seen in isolation or associated with areas of delayed enhancement. These findings suggest an association between myocardial fibrosis and microvascular impairment in patients with cardiac systemic sclerosis. A comprehensive cardiac MR study with stress perfusion and delayed enhancement seems to improve the sensitivity of the method for detection of subclinical cardiac involvement in systemic sclerosis.

Churg-Strauss Syndrome

The Churg-Strauss syndrome is a rare form of systemic necrotizing vasculitis of small vessels, which can mimic acute coronary syndrome. Patterns of delayed enhancement on MRI may vary and include patchy LV distribution involving the subendocardial, midwall, and subepicardial regions. Baccouche et al[48] described a high

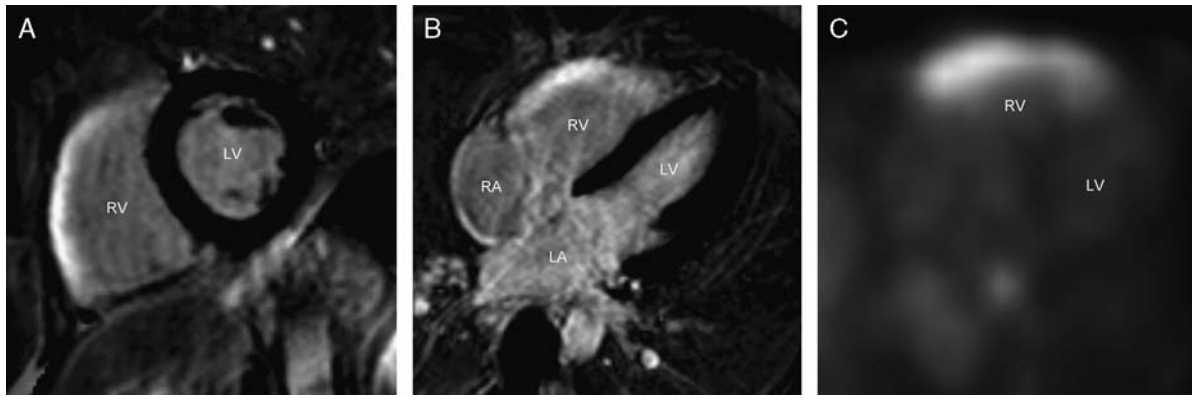

FIGURE 12. Cardiac sarcoidosis. Short-axis (A) and horizontal long-axis (B) delayed enhancement images from a patient with cardiac sarcoidosis demonstrate diffuse delayed enhancement in the RV free wall. Axial ^{18}F-fluorodeoxyglucose-positron emission tomography (FDG-PET) image (C) of the same patient shows intense FDG uptake corresponding to the abnormal enhancement. LA indicates left atrium; RA, right atrium.

positive histopathologic correlation between regional myocardial delayed enhancement and the presence of eosinophilic infiltrates.

Dennert et al,[49] in a cohort of 32 patients with clinically remissive Churg-Strauss syndrome, found a 62% prevalence of cardiac abnormalities compared with 3% in age-matched and sex-matched control subjects. In this study, 38% of asymptomatic patients with a normal electrocardiogram demonstrated echochardiographic or MRI findings consistent with subclinical cardiac disease.

Lyme Disease

Lyme cardiomyopathy is a rare disorder, affecting between 1.5% and 10% of patients with Lyme disease, typically presenting as an atrioventricular block.[50] The most common finding on delayed enhancement MRI images is hyperenhancement on the midwall of the basal anteroseptal LV region, which is the expected location of the atrioventricular node.[51] A decrease in the extent of delayed enhancement after 6 weeks of treatment was shown to be associated with improvement in the atrioventricular block.

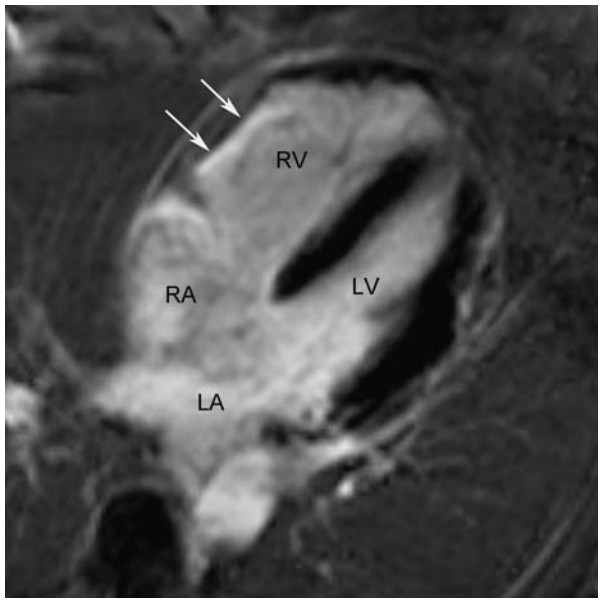

FIGURE 13. ARVD. A horizontal long-axis delayed enhancement image of a patient with ARVD demonstrates abnormal enhancement in the RV free wall (arrows). LA indicates left atrium; RA, right atrium.

CONCLUSIONS

Delayed enhancement in MRI has become very useful in clinical practice, not only for the detection of myocardial diseases but also for differentiation between ischemic and nonischemic myocardial diseases. In the setting of clinical suspicion of nonischemic cardiac diseases, this technique is very helpful for narrowing the differential diagnosis among these entities. Numerous recent studies have emphasized the role of delayed enhancement MRI for prognostication.

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CME-SAM EXAM
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CME-SAM EXAMINATION

Please mark your answers on the ANSWER SHEET.

After completing this CME-SAM activity, physicians should be better able to:

- Differentiate ischemic from nonischemic patterns of myocardial delayed enhancement
- List the most common myocardial regions that present delayed enhancement in patients with hypertrophic cardiomyopathy
- Describe how to optimize delayed enhancement imaging in patients with cardiac amyloidosis
- Learn the prognostic implication of the presence of myocardial delayed enhancement in patients with myocarditis

*1. The main advantage of Phase-sensitive delayed enhancement sequence compared with standard inversion recovery gradient technique is:

A. No need for gadolinium administration
B. Less need for selection of the ideal myocardial inversion time
C. Shorter delay from contrast administration to image acquisition
D. Faster image acquisition
E. It can be performed in any cardiac plane

See Reference 5 of the article for further study.

*2. In a patient with ischemic heart disease the most common delayed enhancement pattern is:

A. Subendocardial and following a coronary artery distribution
B. Subepicardial and following a coronary artery distribution
C. Midwall, not following a coronary artery distribution
D. Along the right ventricular side of the septum
E. Subendocardial, not following a coronary artery distribution

See Reference 10 of the manuscript for further study.

*3. The presence of delayed enhancement in a patient with myocarditis is associated with:

A. Treatment response
B. Concomitant myocardial infarction
C. Increased mortality

D. Better long-term survival
E. A non-viral etiology

See Reference 16 of the manuscript for further study.

*4. In patients with hypertrophic cardiomyopathy, the presence of delayed enhancement is:
A. Commonly seen in the right side of the ventricular septum
B. Subendocardial, in a coronary artery distribution
C. Global and independent of the degree of myocardial hypertrophy
D. Not associated with prognosis
E. Seen at the junction of the right ventricular free wall with the ventricular septum

See Reference 22 of the manuscript for further study.

*5. Diffuse myocardial infiltration in amyloidosis can be problematic for delayed enhancement imaging because:
A. Patients cannot receive gadolinium
B. Images must be obtained with a higher scan delay
C. It is difficult to detect abnormal delayed enhancement on thin ventricular walls
D. Nulling of the abnormal myocardium may mimmic a normal study
E. Global subepicardial enhancement is difficult to separate from epicardial fat

See Reference 30 of the manuscript for further study.

*6. On figure 13 of the manuscript, the mechanism of abnormal contrast delayed enhancement along the right ventricular free wall is increased volume of distribution of gadolinium due to:
A. Fibrosis
B. Inflammatory reaction
C. Ischemic injury
D. Viral infection
E. Drug reaction

See Reference 40 of the manuscript for further study.

*7. In a patient with dilated cardiomyopathy, which delayed enhancement pattern is most consistent with a nonischemic etiology?
A. No delayed enhancement
B. Subendocardial
C. Subepicardial
D. Transmural

See Reference 11 of the manuscript for further study.

*8. Delayed enhancement and 18-FDG avidity on PET involving the right ventricular free wall are most characteristic of which disease?
A. Amyloidosis
B. Lyme disease
C. Sarcoidosis
D. Churg-Strauss syndrome
E. Arrhythmogenic right ventricular dysplasia

Please see the following references for further study:
1. Vignaux O. Cardiac sarcoidosis: Spectrum of MRI features. *AJR*. 2005;184:249–254.
2. Ishimaru S, Tsujino I, Takei T, et al. Focal uptake on 18F-fluoro-2-deoxyglucose positron emission tomography images indicates cardiac involvement of sarcoidosis. *Eur Heart J*. 2005;26:1538–1543.

ANSWER SHEET FOR JOURNAL OF THORACIC IMAGING
CME-SAM PROGRAM EXAM
MARCH 2013

Please answer the questions on pages 93–94 by filling in the appropriate circles on the answer sheet below. Please mark the circle until the letter is no longer visible. To process your exam, you must also provide the following information:

Name (please print): _____
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1.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ
2.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ
3.	Ⓐ	**Ⓑ**	Ⓒ	Ⓓ	Ⓔ
4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ
5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ
6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ
7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	Ⓔ

Your evaluation of this CME activity will help guide future planning. Please respond to the following questions below.

Please rate these activities 1 (minimally) to 5 (completely)	1	2	3	4	5
These activities were effective in meeting educational objectives	○	○	○	○	○
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Please rate your ability to achieve the following objectives, both before and after this activity: 1 (minimally) to 5 (completely)	Pre-test 1	2	3	4	5	Post-test 1	2	3	4	5
1 Differentiate ischemic from nonischemic patterns of myocardial delayed enhancement.	○	○	○	○	○	○	○	○	○	○
2. List the most common myocardial regions that present delayed enhancement in patients with hypertrophic cardiomyopathy	○	○	○	○	○	○	○	○	○	○
3. Describe how to optimize delayed enhancement imaging in patients with cardiac amyloidosis	○	○	○	○	○	○	○	○	○	○
4. Learn the prognostic implication of the presence of myocardial delayed enhancement in patients with myocarditis	○	○	○	○	○	○	○	○	○	○

How many of your patients are likely to be impacted by what you learned from this activity?

<20% 20-40% 40-60% 60-80% >80%

Do you expect that these activities will help you improve your skill or judgment within the next 6 months (1 – definitely will not change, 5 – definitely will change)	1	2	3	4	5
	○	○	○	○	○

How will you apply what you learned from these activities (mark all that apply):

In diagnosing patients	○	In making treatment decisions	○
In monitoring patients	○	As a foundation to learn more	○
In educating students and colleagues	○	In educating patients and their caregivers	○
As part of a quality or performance improvement project	○	To confirm current practice	○
For maintenance of board certification	○	For maintenance of licensure	○

How committed are you to applying these activities to your practice in the ways you indicated above (1 – minimally, 5 completely)	1	2	3	4	5
	○	○	○	○	○

Did you perceive any bias for or against any commercial products or devices? If yes, please explain:	Yes	No
	○	○

How long did it take you to complete these activities ____hours ____minutes

What are your biggest clinical challenges related to radiology?

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