PICTORIAL REVIEW

Differentiation of myocardial scar from potential pitfalls and artefacts in delayed enhancement MRI

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ABSTRACT. Delayed enhancement cardiac magnetic resonance (DE-CMR) imaging is used increasingly to identify and quantify focal myocardial scar. Our objective is to describe factors used in the interpretation of DE-CMR images and to highlight potential pitfalls and artefacts that mimic myocardial scar. Inversion recovery gradient recalled echo sequence is commonly accepted as the standard of reference for DE-CMR. There are also alternative sequences that can be performed in a single breath-hold or with free breathing. Radiologists need to be aware of factors affecting image quality, and potential pitfalls and artefacts that may generate focal hyperintense areas that mimic myocardial scar.

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Delayed enhancement cardiac magnetic resonance (DE-CMR) imaging can identify the presence, location and extent of myocardial scar (dense myocardial fibrosis) due to ischaemic and non-ischaemic heart diseases. This technique is also called late gadolinium-enhanced MRI (LGE-MRI), myocardial delayed enhanced (MDE) MRI and delayed hyperenhanced MRI (DHE-MRI) in the literature. DE-CMR has been shown to be more sensitive than other imaging methods in detecting small subendocardial infarctions [1] and has recently been used in population-based studies to assess the presence of myocardial scar [2, 3]. The technique is relatively straightforward to implement, and images can be interpreted visually for the presence of myocardial scar (hyperenhanced region) without the need for post-processing.

This article is intended to describe quality control procedures, factors affecting image quality, assessment of myocardial scar, and potential pitfalls and artefacts causing focal hyperintensities that mimic myocardial scar in delayed enhancement imaging.

Myocardial delayed enhancement MRI technique

DE-CMR is typically obtained using a segmented inversion recovery gradient recalled echo sequence (IR-GRE) with breath-holds, which is accepted as the standard of reference. Each slice is acquired in one

breath-hold 10–20 minutes after intravenous administration of 0.1–0.2 mmol kg⁻¹ gadolinium-based contrast agent. This sequence applies an 180° inversion pulse to null the signal of normal myocardium and enhance the signal difference between normal and scarred myocardium. The time between inversion recovery pulse and image acquisition is the “inversion time” or TI (Figure 1). The inversion time needs to be optimized for each case individually before image acquisition. With optimal settings, the normal myocardium appears nulled or “black” and myocardial scar areas appear enhanced or “bright”. The mechanism underlying delayed enhancement in the chronic disease setting is increased gadolinium concentration in expanded interstitial spaces because of replacement of non-viable myocytes with highly collagenous scar tissue (dense myocardial fibrosis) [4].

Delayed enhancement images are usually acquired in a stack of 10–12 short-axis slices in combination with a horizontal and a vertical long-axis image perpendicular to the short axis. Slice thickness is 6–8 mm. The gap between slices is 2–4 mm. The in-plane spatial resolution is typically 1.2–1.8 mm. The field of view is 300–380 mm and should be minimized to improve the spatial resolution while avoiding wrap-around artefact [5]. Images are typically obtained with an electrocardiogram (ECG) delay such that mid-diastolic imaging is performed. Obtaining delayed enhancement images at the same spatial location as short-axis cine images is desirable because it enables comparison between suspected scar and local wall motion. Advances in pulse sequences allow us to acquire the entire heart in a single breath-hold with inversion recovery prepared steady-state free precession (IR-SSFP) sequence or with rapid three-dimensional (3D) inversion recovery gradient

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Figure 1. Diagram of inversion recovery gradient echo delayed enhancement sequence and relaxation curves of infarcted and normal myocardium. Trigger delay is the time between R-wave trigger and non-selective 180° inversion pulse. Inversion time is the time between 180° inversion pulse and image acquisition. By adjusting the inversion time at the null point of myocardium for each patient, the signal difference between infarcted areas of myocardium (black line) and normal myocardium (grey line) can be maximised. Inversion time is 300 ms in this example. (Adapted from Vogel-Claussen J et al. *Radiographics* 2006;26:795–810.)

recalled echo sequences. Navigator gated 3D acquisitions cover the entire heart during free breathing. All these sequences can be acquired with phase-sensitive inversion recovery reconstruction. Major characteristics of alternative pulse sequences are compared with the standard reference sequence in Table 1.

Quality control and image interpretation

The following factors should be checked before interpreting images to evaluate the diagnostic quality of images.

1. *Presence of contrast in the images:* If a sufficient amount of contrast material has been administered, liver and kidney in the imaging plane should be enhanced (Figure 2). All structures will appear too dark if contrast has extravasated or washed out.
2. *Inversion time adjustment:* If inversion time is appropriate, normal myocardium should appear hypointense, the blood pool signal should be of intermediate intensity and myocardial scar should be the most intense structure in the image (Figure 3).

The fibrous skeleton of the heart (e.g. mitral valve annulus) is often hyperintense because of gadolinium accumulation in these highly collagenous areas. Heart valves also faintly enhance (Figure 4).

If the inversion time is slightly shorter than optimal inversion time, normal myocardium appears grey in between hypointense endo- and epicardial lines (Figure 5a).

If very long inversion time is used, the image contrast is reduced. Images acquired with suboptimal inversion time may lead to inaccurate assessment of scar size [6]. An IR-SSFP look-locker sequence is used to select the

optimal inversion time visually for maximising the signal intensity between normal and scarred myocardium [7] (Figure 6).

Since each slice can be obtained in one breath-hold with the IR-GRE sequence, the TI time should be slightly lengthened as the delay after contrast increases. Another way to achieve a consistent contrast over a wide range of inversion times is to apply phase-sensitive reconstruction. This method uses phase information to correct magnitude images when TI times are incorrectly determined [8] (Figure 5a,b). The phase-sensitive reconstruction method also reduces the variation in apparent infarct size. In patients with breath-holding difficulty, DE-CMR can be performed using a nominal inversion time value in combination with IR-SSFP sequence which will obviate the need for extra breath-hold for the look-locker sequence to optimise inversion time. A disadvantage of phase-sensitive inversion recovery images is that noise may be more prominent in the image.

We interpret a hyperintense region as definite when it is confirmed either in two adjacent short-axis images or in one short-axis image and a long-axis image at a corresponding location. Confirmation of scar in two different images avoids misinterpretation of artefacts or image features that may be due to motion artefacts, morphological variations or septal clefts. However, very small focal myocardial scar areas might be missed since slice thickness is 6–8 mm.

Assessment of myocardial scar size

Myocardial scar size in DE-CMR can be quantified by automated or manual segmentation methods. These methods are reader dependent for delineation of endocardium and epicardium to calculate left ventricular

Table 1. Major characteristics of delayed enhancement MRI sequences

Characteristic	Standard reference: Multishot sequence	Alternatives		
		Single-shot sequence [14–16]	3D sequence with navigator gating [17, 18]	3D sequence with breath-hold [19, 20]
Sequence name	IR-GRE; with or without PSIR	Single-shot SSFP; with or without PSIR	3D inversion recovery gradient recalled echo; with or without PSIR	3D inversion recovery gradient recalled echo
Left ventricular coverage	Each slice acquired in one breath-hold; whole ventricle covered in 7–10 breath-holds	Entire left ventricle acquired in one breath-hold	Entire left ventricle covered in one acquisition during free breathing	Left ventricle acquired either in multiple or in one breath-hold
CNR	High CNR for normal and infarcted myocardium	Lower CNR than reference standard of IR-GRE; however, adequate image quality can be obtained	Higher CNR than standard of reference of IR-GRE	No significant loss of CNR compared with standard of reference of IR-GRE
Inversion time	Inversion time needs to be adjusted for each slice to avoid contrast change over time	No contrast change over time in the myocardium	Optimal inversion time to null the myocardium may change significantly over the acquisition period	No contrast change over time in the myocardium in one breath-hold technique
Mean imaging time	5–8 min	16 s	10–15 min	2.4 min (multibreath-hold); 20 s (single breath-hold)
Notes		Reduced respiratory artefacts in uncooperative patients; reduced cardiac motion artefacts in patients with arrhythmias	Image quality reduced if the efficiency of navigator gating decreased	

3D, three dimensional; CNR, contrast-to-noise ratio; IR-GRE, inversion recovery segmented gradient recalled echo; PSIR, phase-sensitive inversion recovery; SSFP, steady-state free precession.

Figure 2. Normal post-contrast segmented inversion recovery spoiled gradient echo delayed enhancement image in a 38-year-old male. Increased signal intensity in liver (white arrow) and kidney (black outlined notched arrow) confirm presence of gadolinium-based contrast in the delayed enhancement MRI.

(LV) mass and the scar size as a percentage of LV mass. For calculation of myocardial scar size, hyperenhanced areas are delineated by a reader using the manual segmentation method, which is observer dependent and time-consuming. Automated segmentation methods use either a threshold of signal intensity from 2 to 6 standard deviations above the remote myocardium or a threshold of full-width at half-maximum to calculate infarct size.

Alternatively, visual scoring is a fast and semi-quantitative method to detect infarct size based on 17 segments of the American Heart Association heart model, which can be used in daily clinical practice. In this method, a visual score ranging from 0 to 4 was attributed to each of the 17 segments according to the transmural extent of the hyperenhancement (0, no hyperenhancement; 1, 1–25%; 2, 26–50%; 3, 51–75%; and 4, 75–100% transmural). All scores are summed and divided by the maximal possible score ($17 \times 4 = 68$) and then multiplied by 100 to express global infarct size as a percentage [9].

Pitfalls and artefacts

Differentiation of subendocardial infarct from blood pool

The hyperintense blood between myocardium and papillary muscles can mimic myocardial scar (Figure 7).

Figure 3. Appropriate suppression of myocardial signal by inversion time adjustment in a 56-year-old male with myocardial infarction. Myocardial signal was suppressed successfully using an appropriate inversion time and myocardium was seen as hypointense in the segmented inversion recovery spoiled gradient echo magnitude image (a). Both magnitude (a) and phase-sensitive reconstructed (b) images in the long-axis plane clearly demonstrate subendocardial myocardial scar affecting the anterior wall of the left ventricle (arrowhead). Note the linear motion artefact (*) that is accentuated on the phase-sensitive image (b).

Distinguishing small subendocardial myocardial infarctions from blood pool can also be difficult since both are hyperintense in the image. As an aid, myocardial thickness and morphology can be compared with cine MRI to help determine whether the enhancement occurs in the myocardium or in the LV cavity (Figure 8).

Epicardial and pericardial fat mimicking scar

Epi- and pericardial fat often show high signal intensity using the inversion recovery technique. This can mimic epicardial hyperenhancement (Figure 9).

Figure 4. Normal enhancement of valves and their annuli in a 54-year-old male. Four-chamber segmented inversion recovery spoiled gradient echo image shows contrast enhancement in mitral and tricuspid valves (arrowheads) and their annuli due to accumulation of contrast material in these highly collagenous tissues.

Checking the continuity of myocardial scar area in adjacent slices and in other imaging planes is helpful to identify epicardial fat.

Partial volume effects

Partial volume effects typically cause problems near the aortic outflow tract (Figure 10a–c) and epicardial locations due to right ventricular septomarginal trabeculation, as well as the right ventricular insertion site near the apex (Figure 11a–c). Myocardial hyperenhancement is considered to represent myocardial scar if it can be seen continuously in two consecutive slices (Figure 11d–g).

Myocardial cleft

Myocardial clefts present as a discrete pouch of blood signal penetrating more than half the thickness of the myocardial wall in the basal inferior wall of the left ventricle or the mid- to apical segments of the interventricular septum. They typically narrow or occlude during systole [10]. The blood inside the cleft can simulate myocardial scar in delayed enhancement images. Differentiation can be accomplished by evaluating the pouch and wall morphology on cine MRI (Figure 12).

Myocardial clefts also need to be differentiated from non-compacted myocardium that is characterized by the diastolic ratio of non-compacted to compact layers of myocardium >2.3 at diastole [11].

Septal perforator branch of the left anterior descending artery

The left anterior descending artery gives rise to a variable number of septal perforator branches. Approximately 15% of subjects have a large first septal perforator branch, which may mimic linear hyperenhancement in a short-axis delayed enhancement image at the base of the heart (Figure 13).

Figure 5. Unsuccessful suppression of myocardial signal due to suboptimal inversion time adjustment in a 45-year-old male with a history of myocardial infarction. Segmented inversion recovery spoiled gradient echo magnitude image (a) in the short-axis plane demonstrates inadequate suppression of normal myocardial signal due to shorter inversion time than optimal. Both normal myocardium and myocardial scar area (arrow) in the inferior wall of the left ventricle appear as intermediate signal intensity, making recognition of infarct very difficult. Hyperintense myocardial scar (arrow) is better demonstrated by applying phase-sensitive reconstruction (b).

Figure 6. Selection of optimal inversion time in a patient with a history of myocardial infarction. One short-axis slice of the left ventricle at the level of inferior papillary muscle insertion point is repeatedly obtained using different inversion times varying from 120 to 330 ms with low-resolution inversion recovery prepared steady-state free precession look locker sequence in one breath-hold. There is a large myocardial infarction area in the anterolateral wall of the left ventricle (arrow). The optimal inversion time in this case is visually selected as 300 ms, which maximises the contrast between the infarcted and the normal myocardium.

Figure 7. Subendocardial pseudoinfarct appearance due to blood in 41-year-old male. Short-axis segmented inversion recovery spoiled gradient echo image at the base of the left ventricle (a) shows a suspicious hyperintense area (arrow) for myocardial scar. However, the cine steady-state free precession image from the same slice location at end diastole (b) demonstrates that this hyperintense area represents blood between myocardium and papillary muscle (arrow). The slight difference in the slice location between delayed enhanced and cine image is due to through plane motion of the heart. As a note in this study, cine images are obtained immediately after contrast administration.

Figure 8. Subendocardial myocardial infarction in a 56-year-old female without a history of heart attack. Short-axis segmented inversion recovery spoiled gradient echo image at the mid-ventricular level (a) shows a hyperintense area reflecting subendocardial myocardial infarction (arrow) in the inferoseptal wall. Cine steady-state free precession image at the same level (b) confirms that infarcted area is within the myocardium (arrow). The infarct is hard to identify in (a) owing to poor contrast resolution between the infarct and blood pool. When practical, longer delay times may improve this contrast differential.

Figure 9. Epicardial fat mimicking myocardial scar in a 41-year-old female. Short-axis segmented inversion recovery spoiled gradient echo image at the mid-ventricular level (a) shows a pericardial hyperintense area suspicious for myocardial scar (arrow). The hyperintense signal is on the anatomical trajectory of the left anterior descending artery and can be due to epicardial fat. The long-axis view (b) confirms that this hyperintense signal belongs to epicardial fat (arrow).

Figure 10. (a–c) Pseudoinfarct appearance due to partial volume effect of aortic outflow tract in a 60-year-old male. Short-axis segmented inversion recovery spoiled gradient echo image at the basal level of the left ventricle (b) shows a hyperintense area in the septum due to partial volume effect of aortic outflow track (arrow). This area is continuous with the aortic outflow tract superiorly (*) (a), and ventricular septum inferiorly. Three-chamber image (c) confirms that the hyperintense area in the septum is due to partial volume effect of aortic outflow tract. Both short-axis (b) and three-chamber (c) images also demonstrate a hyperintense area in the lateral wall of the left ventricle (arrowhead) consistent with myocardial scar.

Figure 11. (a–c) Pseudoenhancement at the inferior right ventricular insertion point in a 54-year-old male with normal myocardium. Short-axis segmented inversion recovery spoiled gradient echo image with phase-sensitive reconstruction shows hyperintensity in the inferior right ventricle insertion point due to partial volume effect (arrow) (b). This finding was present only on a single slice of three contiguous slices (a–c), and a consecutive slice at the upper level of hyperintense right ventricle insertion point shows trabeculation of the right ventricle with blood pool at this site (arrow) (a). (d–g) Myocardial scar at right ventricular insertion in a 68-year-old female with pulmonary hypertension. Short-axis segmented inversion recovery spoiled gradient echo image with phase-sensitive reconstruction in two consecutive slices (d, e) shows hyperenhancement at the inferior right ventricular insertion site (arrow). This finding was present on contiguous short-axis slices (not shown), confirming the presence of myocardial scar. Corresponding short-axis cine images (f, g) confirm that hyperenhancement is in the myocardium (arrow).

Figure 12. (a,b) Myocardial cleft in a 50-year-old male without history of myocardial infarction. Four-chamber delayed enhancement image (a) demonstrates a hyperintense area in the septum that is suspicious for myocardial scar (arrow). However, the corresponding cine steady-state free precession (SSFP) image (b) proves that the hyperintense appearance is due to myocardial cleft (arrow). A supplemental movie of cine SSFP sequence (S12C) is available at http://bjr.birjournals.org/site/supplmaterial/S12C_cine_SSFP_movie.mpg

Figure 13. A pseudoenhancement due to septal perforator artery in a 50-year-old normal male. Short-axis segmented inversion recovery spoiled gradient echo image at the basal level (a) demonstrates linear mid-wall delayed enhancement in a location consistent with a large septal perforator (arrow). (b) Corresponding CT angiography shows septal perforator branch of left anterior descending artery in the septum at this location (arrow).

Figure 14. Ghosting artefacts in a 46-year-old male. In the segmented inversion recovery spoiled gradient echo short-axis image at the mid-ventricular level (a), diaphragmatic motion leads to replicated diaphragmatic contours (white arrows) and blurring which interferes with image quality in the inferior wall of the left ventricle (arrow head). One slice below this level (b) inferior wall can be seen without blurring with a better breath-hold. Four-chamber image confirms there is no myocardial scar in the inferior wall (not shown).

Figure 15. Ghosting artefact due to stomach motion in a 47-year-old female. Short-axis segmented inversion recovery spoiled gradient echo image at the mid-ventricular level shows ghosting artefact from stomach motion (thick arrows) that projects over the inferior wall of the left ventricle and mimics myocardial scar at right ventricular insertion point (thin arrow).

Ghosting artefacts

Periodic respiratory, cardiac and stomach motion lead to ghosting artefacts and blurring which may obscure or simulate lesions (Figure 14) [12].

Stomach motion artefacts simulate myocardial scar in one short axis slice, especially on the right ventricular insertion point. They reproduce the size and shape of the stomach along the phase encoding direction. This finding is the important clue to differentiate artefact from myocardial scar (Figure 15).

(a)

(b)

Figure 16. Subendocardial diffuse and global hyperenhancement due to cardiac amyloidosis. Inversion recovery delayed enhancement image with phase-sensitive reconstruction in the short-axis (a), and long-axis (b) planes demonstrates subendocardial, global and diffuse hyperenhancement (black arrows), which does not match to any coronary artery territory in a patient with biopsy-proven cardiac amyloidosis. Note that left atrial wall also enhances diffusely.

Cardiac amyloidosis

In cardiac amyloidosis, the accumulation of amyloid proteins in interstitial space increases contrast accumulation diffusely. Thus, detection of the optimal inversion time to suppress the normal myocardial signal is typically difficult. A pronounced subendocardial, global or diffuse increase in signal intensity, which does not match any coronary artery territory, is the characteristic enhancement pattern for cardiac amyloidosis [13] (Figure 16).

Conclusion

DE-CMR represents an emerging standard for identification and quantification of myocardial scar/fibrosis. The IR-GRE sequence is commonly accepted as the standard of reference. There are alternative sequences that can be performed in a single breath-hold or with free breathing in patients with breath-holding difficulty. Radiologists need to be aware of potential pitfalls and artefacts that can be seen as hyperintense areas in delayed enhancement images and may otherwise mimic myocardial scar. It is important to confirm the presence of a hyperintense area in more than one slice (two adjacent short-axis slices or one short-axis and one long-axis image at a corresponding location) and to assess the corresponding cine images when interpreting DE-CMR of myocardial scar. These cross-checks reduce the opportunity for misinterpretation; however, despite these improvements, current DE-CMR resolution is still a limitation in the detection of very small myocardial scars.

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