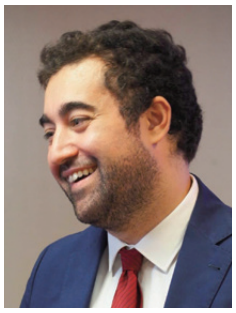
WHITE PAPER

Cardiac MRI to Assess the Acute Phase of Myocardial Infarction: Applications of new 2020 SCMR Guidelines



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Introduction

Myocardial infarction (MI) remains a leading cause of mortality worldwide[1]. In fact, it can cause an irreversible myocardial damage, unless coronary reperfusion is restored sufficiently early. Thanks to its ability to accurately identify the MI area[2], cardiac MRI is the gold standard to assess the patient prognostic after MI[3,4]. MRI can be also helpful to identify residual viability, stunning, and microvascular damage[5]. In addition, post-MI sequelae, including left ventricle (LV) thrombus, LV aneurysm or pseudo-aneurysm formation, pericarditis, can be easily identified[6].

This article will focus only on the challenging evaluation of the MI acute phase (first 7 days) with:

- the assessment of the myocardial viability, in order to determine if a coronary revascularization could restore a contractile function in the MI area.
- the evaluation of prognostic factors predicting ventricular remodeling: MI size[7], presence of microvascular obstruction (MVO) or presence of hemorrhagic component in the MI area[8].

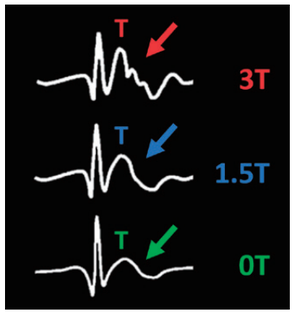
The Society for Cardiovascular Magnetic Resonance (SCMR) has very recently published the latest guidelines on validated protocols to follow in clinical routine for the "Acute MI or Acute coronary syndromes"[6].

MRI safety precautions

The examination room has to be equipped with a nearby safety equipment (emergency resuscitation cart, defibrillator). In addition, if the patient had a recent coronary angiography with iodine contrast agent, a beforehand serum creatinine verification has to be done before doing a gadolinium injection to ensure a glomerular filtration rate > 30 ml/min. Finally, performing an MRI in the hours following a stent implantation is allowed without any risk for the patient[9].

> *"In case of inferior myocardial infarction, an apnea test with ECG has to be performed before cardiac MRI to eliminate a severe paroxysmal atrioventricular block suspicion, which is an exam contraindication."*
>
> **Théo Pezel, MD**

Tips and tricks: How to interpret ECG tracing during MRI exam?



- During cardiac MRI exam, a 2-3 lead ECG tracing is performed to allow the acquisition synchronization with the cardiac cycle. The R wave, and only the R wave, is detected by the system and used to locate a new heart cycle.
- When patient is immediately scanned following a MI, it may be tempting to use the MR ECG trace to detect an arrhythmia or some ischemia signs. However, it is important to know that magnetic field is responsible for ECG signal distortion, leading to a pseudo-growth of the T wave in particular. This is caused by blood flows, and particularly the aortic flow near the electrodes. Therefore, any diagnosis cannot be made from an MR ECG trace. Its only exploitable parameter is the heart rate to identify possible arrhythmia.

Recommended Cardiac MRI sequence protocol (SCMR guidelines): Acute MI or Acute coronary syndromes

Sequence	Technical description	Clinical aim
SSFP localizer (steady-state free precession imaging)	Real time apnea sequence in the three orthogonal planes	Obtaining the anatomical axes of the heart
STIR (short TI inversion recovery) (optional)	T2-weighted sequence triple-inversion: dark blood recovery with fat saturation	Identification of **myocardial edema**
T2 mapping (optional)	T2 mapping sequence short axis ± long axis (recently integrated into routine imaging protocols)	
T2* mapping (optional)	T2* mapping sequence (short axis) before contrast agent injection Please note that T2* mapping is only available at 1.5T (cannot be used at 3T)	Identification of **hemorrhagic MI component**
Cine SSFP	Cine sequence performed in short and long axis (2, 3 and 4 chambers view) before or after contrast agent injection	Assessment of **segmental kinetics** and **myocardial thickness** Assessment of **Left ventricular ejection fraction (LVEF)**
T1 mapping pre/post Gadolinium (optional)	T1 mapping sequence before and after gadolinium (short axis) (recently integrated into routine imaging protocols)	Identification of **hemorrhagic MI component** **Extracellular volume assessment (ECV)**
First pass perfusion (optional)	Saturation-recovery sequence, single-shot, gradient echo, at rest Done after an intravenous 0.05-0.10 mmol/kg gadolinium bolus with a 3-7 ml/s flow rate	Identify **a myocardial perfusion defect** (identification of MI area, presence of low-flow/no-flow)
Late gadolinium enhancement (LGE) (magnitude)	Sequence acquired 10 minutes after gadoliniu injection Gradient echo sequence with inversion-recovery in short and long axis (2 and 4 chambers view), 2D or 3D An inversion time (TI) has to be determined to null healthy myocardial signal ± early enhancement imaging at 1-3 min in order to visualize the MVO	Identification of **myocardial scar ± edema** Identification of **MVO** Eliminate **left ventricular thrombus suspicion** (visible in PSIR especially) Look for an associated **pericardial effusion**
PSIR (phase sensitive inversion recovery) (optional)	Sequence acquired 10 minutes after the gadolinium injection Inversion-recovery sequence with a PSIR reconstruction, to be insensitive to the inversion time choice	

Note: "Optional" sequences are recommended for specific indications (Cf "Clinical aim" column)

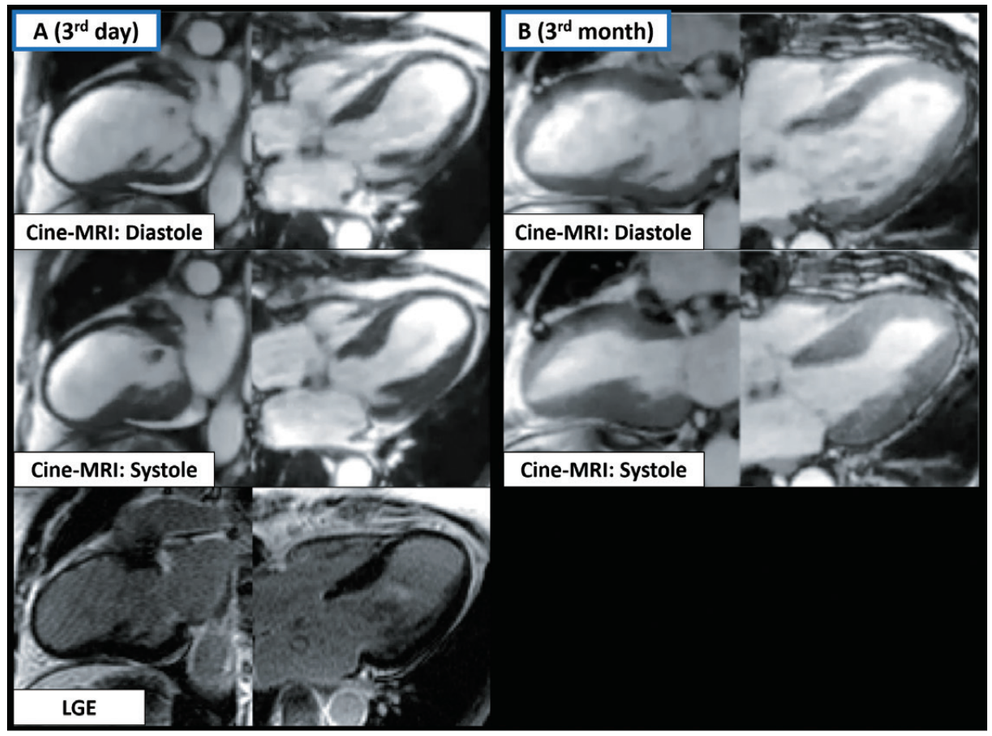


Figure 1 Cardiac MRI performed 3 days and 3 months after a myocardial infarction (MI) in a 68-year-old man with diabetes mellitus. The patient had a proximal left anterior descending coronary (LAD) occlusion treated with a stent during a coronary angiography.

A: Long axis cine-MRI and LGE (2 and 4 chambers view) at the 3rd day after MI
Large antero-septo-apical akinesia (absence of myocardial thickening between diastole -above image - and systole -middle image-) and 6 mm parietal thinning measured. Presence of a minimal non-transmural subendocardial anterior IDM with LGE <25% thickness (bottom image) in favor of a good myocardial viability.
B: Long axis cine-MRI (2 and 4 chambers view) at the 3rd month after MI revascularization
Significant functional recovery of segmental kinetics at the antero-septo-apical wall (marked myocardial thickening between diastole -top image- and systole -bottom image-). This functional LV recovery at 3 months after the LAD revascularization confirms the good viability of the antero-septo-apical territory diagnosed on the LGE sequences at the 3rd day, despite the presence of an initial parietal thinning due to myocardial stunning.

Cardiac MRI images analysis

Cine-MRI sequence

Cine-MRI sequences are essential to assess the left ventricular ejection fraction (LVEF), segmental kinetic abnormalities and myocardial thickness. However, a recent work has shown that parietal thinning is not specific enough to conclude on a myocardial viability[10] (Figure 1).

"The parietal thinning of the myocardium is not a sufficient sign to conclude on myocardial viability."

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First-pass perfusion sequence

Myocardial perfusion imaging is based on assessing the contrast agent delivery to the myocardium during the first pass following a bolus injection. In the case of a MI followed by a revascularization, infarct regions could appear hypo-intense, due to a low flow into the scar tissue. Thus, perfusion images interpretation usually also takes into account the viability assessment by LGE[11].

In addition, another goal to the first pass sequence is to detect MVO, which can help to distinguish a chronic MI to an acute one, since MVO exclusively occurs in acute MI and disappears progressively over time[12].

Late gadolinium enhancement (LGE) sequence

During MI acute phase, two pathophysiological phenomena can lead to a local extracellular volume: myocardial necrosis and edema. In fact, extracellular myocardial edema can persist for one month after MI[13].

Myocardial viability refers to the myocardium part that lives and contributes (or has the potential to contribute) to the systolic blood ejection. To assess this myocardial viability in cardiac MRI, LGE sequence can be used to study the extracellular gadolinium retention, 10-15 minutes after injection. The concept is based on the delayed wash-in and wash-out in tissues with an increased proportion of extracellular space. In the case of an acute MI, this is caused by edema and by cellular necrosis and lysis, while in chronic MI, the extracellular space increase is caused by fibrosis[14].

In clinical practice, LGE is qualitatively evaluated with visual estimation, establishing a thickness percentage of the infarcted myocardium in relation with the global wall, in order to define the transmural LGE extent. A semi-quantitative approach has also been described, using the American Heart Association (AHA) 17 segment model and giving for each segment a score from 0 to 4 depending on the scar extent (0: no scar, 4: 100% scar). In addition to this visual estimation, some companies have proposed post-processing solutions like Medis® (Medis Medical Imaging Systems, Netherlands).

In clinical practice, myocardial viability can be concluded as long as **LGE transmurality is <25%**[3].

"Myocardial edema can persist up to one month after myocardial infarction (MI), leading to an over-estimation of the MI size and a difficulty to conclude about myocardial viability. You cannot eliminate myocardial viability the first month after an MI! You have to repeat the cardiac MRI one month after!"

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In the case of acute MI, it's very challenging to distinguish myocardial necrosis and edema on LGE, which is a major limitation in the myocardial viability assessment within the first month following the MI[15]:

- In case of transmural LGE (> 50-75%): myocardial viability cannot be evaluated with consistency because of the difficulty to quantify the "peri-lesional edematous component" with this transmurality. To assess the edema presence, T2 STIR or T2 mapping sequence can be used[16].
- In case of non-transmural LGE (<25%): MI is viable[3].

T2-STIR and T2 mapping sequences

Myocardial edema can be detected on T2 images (T2-STIR or T2 mapping)[16]. Moreover, myocardial edema assessment can guide the operator to date the MI age. In fact, edema visualization corresponds to a recent MI, less than one month old.

Focus: How to understand microvascular obstruction (MVO)?

Definition

Revascularization of epicardial coronary arteries by angioplasty is not sufficient to confirm the myocardial micro-vascularization integrity. No-reflow histologically corresponds to a non-viable area linked to the persistence of a microvascular obstruction (MVO) which can paradoxically be aggravated by coronary reperfusion[8]. In fact, a brutal restoration of a normal blood supply to damaged micro-vessels could lead a reperfusion injury, leading a myocardial swelling acceleration, tissue edema, endothelial disruption and inflammation. Production of oxygen-free radicals is enhanced by this reperfusion within the first few minutes of reflow and also takes part in reperfusion injury[17].

MVO diagnosis

Two criteria allow to make the MVO diagnosis[8] (Figure 2):

1) Delay (slow flow) or absence of subendocardial ± transmural perfusion (no flow/no reflow), observed with the first-pass sequence.
2) Persistence of a low signal intensity in the subendocardial area, surrounded by high and delineating signal.

Finally, it is important to remember that MVO signs progressively disappear over time after MI, replaced by myocardial scar.

MVO hemorrhagic component

A MI with MVO can be complicated by a local hemorrhagic component following the reperfusion, after microvascular obstruction. This hemorrhage can be identified with iron-sensitive MRI sequences like T1, T2 and T2* mapping[16]. T2* quantification is currently the method of choice for this indication.

MVO prognostic value

The MVO detection has prognostic implications as well, since correlations have been demonstrated with cardiovascular adverse events (heart failure, arrhythmia and death), bigger infarct size, aneurysm and LV remodeling[8,18].

"Microvascular obstruction signs progressively disappear over time after the myocardial infarction, replaced by myocardial scar."

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Case report: patient in MI acute phase after revascularization

68-year-old diabetic patient performing a cardiac MRI in the acute phase of an revascularized anterior MI (Figure 2). In the case of a revascularized patient, the challenge is not to assess myocardial viability, because the patient is already revascularized, but rather to look for the poor MI prognostic factors. Therefore, the report should specify in this situation the presence or absence of the following prognostic factors: MI size, MVO, hemorrhagic component and intra-LV thrombus.

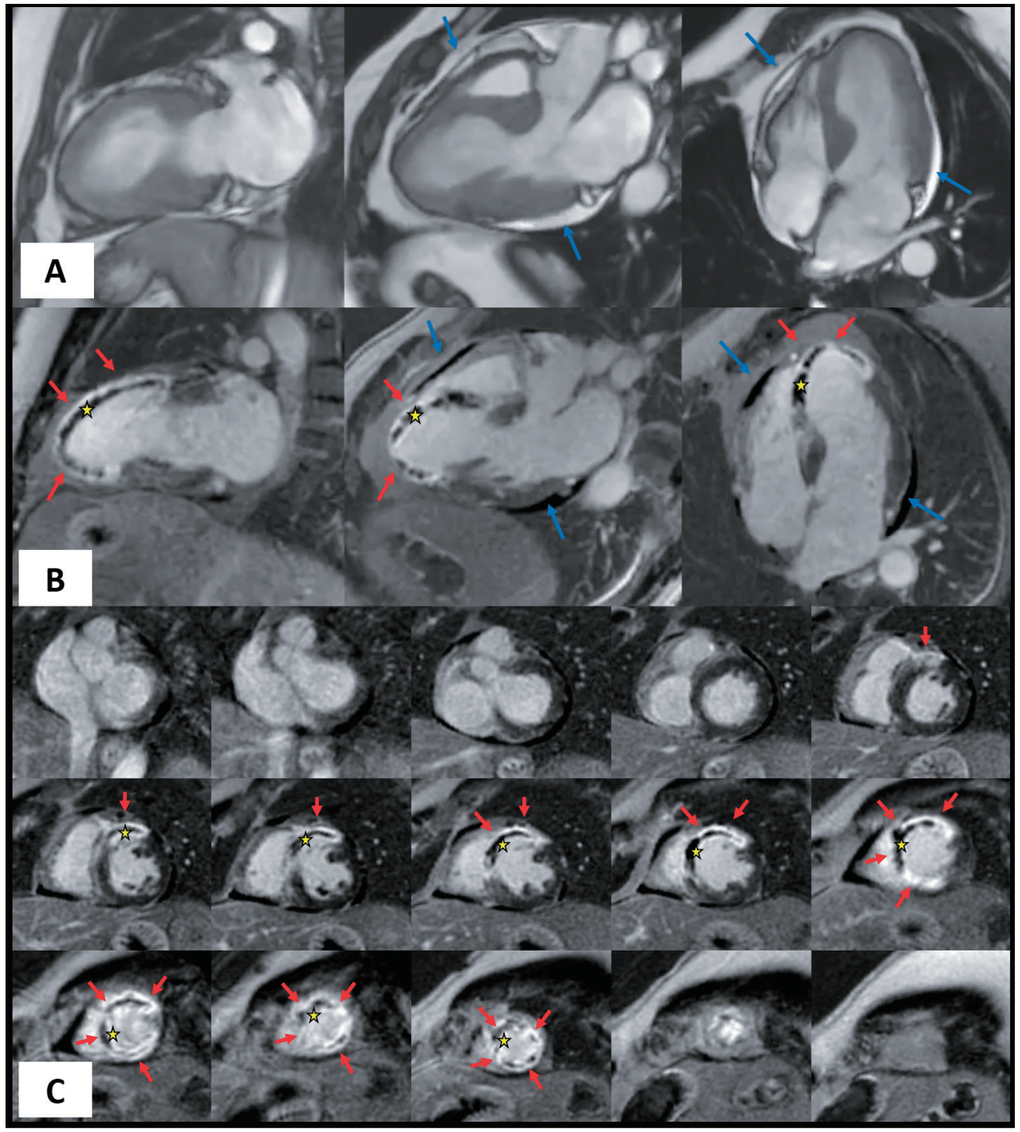

Figure 2 Cardiac MRI performed 96 hours after an anterior myocardial infarction (MI) in a 42-year-old male smoker, following a coronary angiography revealing an ischemic heart disease.

A : SSFP scan - Long axis cine-MRI (2, 3 and 4 chambers view)
Large antero-septo-apical akinesia with the presence of a moderate pericardial effusion which appears in hypersignal (blue arrows).
B, C : LGE scan - Long axis (2, 3 and 4 chambers view) and short axis
Severe anterior MI with visualization of transmural LGE (hypersignal) in the antero-septo-apical area (red arrows). MI is complicated by microvascular obstruction (MVO) corresponding to the areas in hyposignal (yellow stars).

Conclusion

This article presented the cardiac MRI protocol recommended by SCMR guidelines to assess the patient prognostic after a MI. Cardiac MRI is the gold standard for evaluating post-MI prognosis. In fact, it allows to associate a morphological and functional analysis with first pass perfusion and LGE sequences to directly quantify the MI transmurality. However, this viability assessment remains challenging during the MI acute phase. In this case, the evaluation corresponds to the search of prognostic factors predictive of left ventricular remodeling: MI size, presence of MVO and MI hemorrhagic component. Finally, a major point to keep in mind is that a one-month follow-up cardiac MRI is often requested.

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