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Cardiac MRI in the diagnosis of complications of myocardial infarction

A. Flavian a,*, F. Carta b, F. Thuny c, M. Bernard b, F. Kober b, G. Moulin a, A. Varoquaux a, A. Jacquier a

a Service de radiologie adulte générale et vasculaire, hôpital de la Timone, 264, rue Saint-Pierre, 13385 Marseille cedex 5, France
b CNRS-CRMBM, UMR 6612 CNRS, faculté de médecine Timone, université de la Méditerranée, 13385 Marseille cedex 5, France
c Service de cardiologie et pathologies valvulaires, hôpital de la Timone, 264, rue Saint-Pierre, 13385 Marseille cedex 5, France

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Abstract The improvement in revascularization techniques and medicine treatment during infarction has substantially reduced mortality during the acute phase of this condition. Since the advent of kinetic sequences and the concomitant development of gadolinium chelates and delayed enhancement sequences, cardiac MRI has become the second-line reference examination for ischemic heart disease. The technique of delayed enhancement with the inversion recovery sequence performed after injection has been validated for numerous indications in ischemic disease. Delayed enhancement sequences make it possible in particular to look for “no-reflow” areas (microvascular obstructions), to quantify the infarction area, and to assess prognosis. MRI also allows us to define the area at risk, that is, the area with edema, and to look for and assess the mechanical complications of the infarction. The aim of this review is to summarize current knowledge about: the pharmacokinetic principles that regulate myocardial enhancement; the different sequences available to acquire delayed enhancement images, and; the value of cardiac MRI in the diagnosis of complications of myocardial infarction.

Cardiovascular diseases are the principal cause of death in industrialized countries [1]. In France, they accounted for 28.4% of all deaths in 2005, that is, 150,000 individuals. The improvement in revascularization techniques and in medicine treatment during infarction has substantially reduced mortality during the acute phase of this condition. Nonetheless, the chronic complications of this disease are a public health problem, and ischemic heart disease is responsible for 5% of the deaths among these patients. The concomitant development of gadolinium chelates and of delayed enhancement MRI sequences led to the

* Corresponding author.
E-mail address: antonin.flavian@orange.fr (A. Flavian).
demonstration of myocardial enhancement after gadolinium injection during myocardial infarction (MI) in the mid 1980s. Gadolinium chelates have changed very little if at all since then, unlike delayed enhancement MRI, which has undergone many substantial improvements. Numerous studies have also shown the fundamental importance of quantifying the enhanced area. Cardiac MRI has thus acquired an important role in the diagnosis, evaluation and treatment follow-up of coronary artery disease.

Nonetheless, the use of MRI for this indication varies between centers across France. The role of slice imaging in assessment of the complications of MI was reported as early as 2004 [2]. The aim of this review is to summarize current knowledge about:

- the pharmacokinetic principles that regulate myocardial enhancement;
- the different sequences available to acquire delayed enhancement images;
- the value of cardiac MRI in the diagnosis of MI complications.

### Physiopathology of delayed myocardial enhancement

The gadolinium chelates used in clinical practice are extracellular contrast agents, that is, they diffuse freely between the vascular and interstitial sectors but never penetrate the cell sector. Remember that approximately 5% of the myocardium is vascular, 15% interstitial, and 80% cellular.

The intensity of tissue enhancement by gadolinium depends on two factors: tissue perfusion and; the volume of gadolinium distribution in the tissue. That is, the blood brings the contrast product to the capillary network, where exchanges with tissue take place. Next, the volume of gadolinium distribution (interstitial sector) in the tissue determines the intensity of the enhancement compared with adjacent tissue. During MI, MRI with delayed enhancement sequences differentiates between three different types of myocardial tissue: viable myocardium (the signal of which is cancelled by inversion recovery), necrotic myocardium (enhanced compared with normal myocardium) and lesions of microvascular obstruction (“no-reflow” areas, which appear dark within the enhanced area and demonstrate the complete absence of perfusion). After total coronary occlusion, myocytes are the first cells to die, and the first manifestation of this cell death is the destruction of the cell membrane and the consequent disappearance or reduction of the cellular sector and increase in the interstitial sector. The volume of gadolinium distribution in the infarcted myocardium thus increases. When the ischemic lesions are very deep, tissue edema, inflammatory reactions, endothelial necrosis and microthrombotic phenomena cause microvascular obstruction. This obstruction prevents the blood from perfusing this area of the myocardium and thus also prevents its enhancement by gadolinium. The kinetics of extracellular contrast product between the blood, an exchange compartment, and the myocardium, a distribution compartment, follows a two-compartment model, as described by Kety [3]. Two earlier reviews have explained this equation in detail [4,5].

### Different delayed enhancement sequences

Simonetti et al. [6] developed the 2D and then 3D gradient IR TurboFlash sequence that subsequently became the reference sequence for exploring myocardial enhancement in clinical practice [7]. Briefly, this sequence begins by a magnetization preparation pulse of 180° inversion recovery (IR), followed by a variable time (inversion time, TI) before image acquisition. The relaxation speed of various tissues differs especially according to their concentrations of gadolinium. The inversion time must be chosen so that the normal myocardium signal is cancelled out and all the areas with higher gadolinium loads than normal myocardium (area of infarction, for example) appear bright (hyperintense signals) [8]. The optimal inversion time is chosen based on specific sequences (Look-Locker or TI Scout, depending on the manufacturer) that make it possible to analyze the contrast rapidly for different inversion times. The myocardial relaxation time depends on blood levels of gadolinium and therefore, varies over time as a function of renal clearance. The inversion time must therefore increase constantly during the examination. It must also be chosen very carefully for it can lead to artefacts or errors of interpretation. Besides these reference sequences, other sequences have been developed to assess viability: “steady state free precession inversion-recovery” (depending on the manufacturer: 2D or 3D MDE, TrueFisp IR 2D or 3D single or multishot, and TurboFlash or TrueFisp PSIR, phase sensitive inversion recovery, at Siemens only). Viallon et al. [9] compared all of the delayed enhancement sequences to the reference ultra-fast gradient echo sequence (TurboFlash IR 2D for Siemens, 2D MDE for General Electric, TFE prepulse invert for Philips). They showed that 3D acquisitions allow complete coverage of the left ventricle in one breath hold with a better contrast to noise ratio (Fig. 1a). The PSIR sequences do not require adaptation of the inversion time and allow recognition of the enhancement of artefacts due to a poor TI setting. The spatial resolution of the single shot sequences (one slice to each heart beat) is lower but they nonetheless produce interpretable images, even for patients with arrhythmia or during free breathing.

### How to perform the delayed enhancement sequences during MRI

The major criteria for the precision of the assessment of the delayed enhancement area are:

- the selection of the time between the injection and the performance of the sequence;
- the selection of an appropriate inversion time, as explained above;
- a fine slice thickness (6 mm) and a sufficient quantity of contrast product (0.2 mmol/kg).

For acute infarction, Wagner et al. [8] found no differences in the evaluation of the extent of necrosis between 5 and 30 min after injection of 0.1 or 0.2 mmol/kg of gadolinium chelate. They underline that the accuracy of the measurement is related to the correctness of the inversion time setting. Although most authors agree that delayed enhancement sequences should be acquired between 10
and 15 min after the injection of contrast product, shorter delays can be used [10,11]. In a multicenter study of 282 patients with acute infarction and 284 patients with chronic infarction, Kim et al. [12] showed that the sensitivity of delayed enhancement sequences was highest (99% for acute and 91% for chronic infarction, with angiography as the reference), with gadolinium doses greater or equal to 0.2 mmol/kg. The authors also concluded that there was no significant difference for the gadolinium doses greater or equal to 0.2 mmol/kg between 10 and 30 min after injection.

**Diagnosis of the acute or chronic character of the MI**

Progress has been made in adapting T2-weighted sequences in cardiac MRI, to improve the search for intramyocardial edema in order to differentiate acute and chronic infarction; [13—16]. The presence of myocardial edema in the ischemic area of T2-weighted sequences with fat saturation is a robust marker of the acute nature of an infarction (Fig. 1b). On the other hand, dyskinesia and thinning of the wall indicate that it is chronic. Extracellular contrast products enhance the myocardium but cannot distinguish between acute and chronic infarction. Saeed et al. [17] described the possibility of using the combination of an intravascular contrast product and a standard contrast product to define the age of the infarction, for the intravascular product will enhance only the acute infarction [17]. This phenomenon is explained by the great microvascular permeability during the acute phase of infarction, which disappears as it heals.

**Evaluation of MI severity**

**The extent of delayed enhancement**

Delayed enhancement after gadolinium injection is a prognostic marker for ventricular remodeling, as Nijveldt et al. showed after MI among 63 patients; delayed enhancement was the item most often correlated with increased telesystolic and telediastolic volumes and thus with left ventricular (LV) dysfunction [18].

Wu et al. [19] used delayed enhancement sequences to show that the size of the infarction at the acute phase is correlated with its long-term prognosis. Over the follow-up period (16 ± 5 months), the number of cardiovascular event was 30% for the patients whose infarction took up less than 18% of the LV myocardium, 43% for the patients whose infarct ranged from 18 to 30% of the LV myocardium, and 71% for the patients whose infarction took up more than 30%. Choi et al. [20] conducted explorations of 24 patients during the first week and then 3 months after their first infarction. They observed that the best predictor of overall contractile recovery was the presence of stunned unenhanced myocardium or of myocardium with enhancement involving less than 25% of its thickness. Other reports have confirmed these data [21]. Delayed enhancement expresses the presence of fibrosis. Enhancement can only be shown when fibrosis exceeds 15% of the area in an abnormal region and this region is adjacent to a normal region. That is, the interpretation of these images is essentially qualitative and is based on comparison with a myocardial parenchyma considered healthy.

Several studies have confirmed that the size of the delayed enhancement and the presence of microvascular obstruction ("no reflow" lesions) at the acute phase of MI are correlated with LV remodeling [22—27].

The presence of delayed enhancement expresses the myocardial scar after infarction, and marks a risk of mortality independent of other prognostic markers. In 2009, Benjamin et al. followed a series of 857 patients who had had a cardiac MRI, including delayed enhancement sequences and a median follow-up of 4.4 years [28]. A myocardial scar and the existence of a coronary abnormality were associated with risk of death and necessitated consideration of the need for recourse to cardiac transplantation, independently of the LV ejection fracion (LVEF), compared with patients without myocardial scars, who had a relative risk of requiring cardiac transplantation of 1.33 (95% confidence interval: [1.05 to 1.68], P=0.018).
Cardiac gadolinium: the phenomenon; ages are (Figs. 2 and 3). They occur exclusively during the first 2 to 3 weeks after infarction. The exact origin of these blockages is probably multifactorial and the lesions can be due to the presence of: tissue edema; impairment of endothelial vascular cells, or; migration of atheromatous debris after angioplasty. Microvascular obstruction appears as a subendocardial hypoperfused area within the necrotic area and is found in 37 to 50% of patients with ST segment elevation MI [29,30]. The no-reflow lesions occur in larger infarctions (measurement based on enzyme assays and MRI) and those with longer occlusion time before revascularization. Visualization of microvascular obstruction is a dynamic phenomenon: these areas are enhanced by the distribution of gadolinium from the periphery to the center of the lesion. Bogaert et al. recommended that delayed enhancement sequences be used to look for them in the 2 to 3 first minutes after the injection of contrast product [31]. MRI repeated at 4 months postinfarction in this series also showed poorer functional recovery in the microvascular obstruction group than the group without it [31]. Wu et al. [19] showed that microvascular obstruction is associated with a higher risk of long-term myocardial complications. The larger the enhanced area, the more frequent the onset of cardiovascular complications in this long-term follow-up (25 months). The presence of microvascular obstruction after infarction was an important prognostic factor due to its association with a larger infarction area and worse impairment of LV function during the first week postinfarction. Others later confirmed these results [29]. In 2009, Cochet et al. [24] distinguished the microvascular obstruction visible on first-pass perfusion sequences (early after gadolinium injection) from the persistent microvascular obstruction corresponding to the no-reflow areas on delayed enhancement sequences. Microvascular obstruction that is persistent may thus be a more negative prognostic marker than that seen only on first-pass sequences for comparing the onset of major cardiac events during a one-year postinfarction follow-up of patients with these lesions on MRI.

Microvascular obstruction

Diminished arterial flow can persist after recanalization of an epicardial artery, due to microvascular obstacles (in the arterioles, veins, and capillaries). These lesions prevent reperfusion, to a greater or lesser degree; they are called microvascular obstruction or no-reflow areas (Figs. 2 and 3). They occur exclusively during the first 2 to 3 weeks after infarction. The exact origin of these blockages is probably multifactorial and the lesions can be due to the presence of: tissue edema; impairment of endothelial vascular cells, or; migration of atheromatous debris after angioplasty. Microvascular obstruction appears as a subendocardial hypoperfused area within the necrotic area and is found in 37 to 50% of patients with ST segment elevation MI [29,30]. The no-reflow lesions occur in larger infarctions (measurement based on enzyme assays and MRI) and those with longer occlusion time before revascularization. Visualization of microvascular obstruction is a dynamic phenomenon: these areas are enhanced by the distribution of gadolinium from the periphery to the center of the lesion. Bogaert et al. recommended that delayed enhancement sequences be used to look for them in the 2 to 3 first minutes after the injection of contrast product [31]. MRI repeated at 4 months postinfarction in this series also showed poorer functional recovery in the microvascular obstruction group than the group without it [31]. Wu et al. [19] showed that microvascular obstruction is associated with a higher risk of long-term myocardial complications. The larger the enhanced area, the more frequent the onset of cardiovascular complications in this long-term follow-up (25 months). The presence of microvascular obstruction after infarction was an important prognostic factor due to its association with a larger infarction area and worse impairment of LV function during the first week postinfarction. Others later confirmed these results [29]. In 2009, Cochet et al. [24] distinguished the microvascular obstruction visible on first-pass perfusion sequences (early after gadolinium injection) from the persistent microvascular obstruction corresponding to the no-reflow areas on delayed enhancement sequences. Microvascular obstruction that is persistent may thus be a more negative prognostic marker than that seen only on first-pass sequences for comparing the onset of major cardiac events during a one-year postinfarction follow-up of patients with these lesions on MRI.

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Intramyocardial hemorrhage

Intramyocardial hemorrhage after infarction is secondary to the suffusion of blood within the infarction area, associated with profound impairment of the vascular endothelium following damage to the cells of the vascular wall. It demonstrates an irreversible stage of myocardial ischemia. Its occurrence is correlated with the size of the infarction and the duration of the occlusion [32]. It can be seen on MRI by T2-weighted sequences, where the hypointense signal expresses an artefact of magnetic susceptibility corresponding to the presence of iron in the blood in the hemorrhagic area. It can be interpreted as a hemorrhage only within the infarction area. In a recent publication, Ganame et al. used T2 STIR sequences to detect bleeding in the infarction area, with a minimum volume of 1 mL. MRI took place on two occasions in 88 patients who had undergone MI revascularization; the first was the week after the acute phase, and the second 4 months later. These authors reported that myocardial hemorrhage in the infarction after revascularization, regardless of the size of the infarction, is a risk factor independent of ventricular remodeling [33]. Nonetheless, our understanding of intramyocardial hemorrhage remains sketchy. That is, hemorrhage has been described within lesions showing microvascular obstruction, but data to affirm that these are different entities remain sparse. It is possible that intramyocardial hemorrhage occurs in the patients with more substantial and larger microvascular lesions.

Nonetheless, Basso et al. used histology and MRI to explore the hearts of two patients who died of cardiogenic shock after MI [34]. Both had large regions of T2-weighted hypointense signals, which were hypoperfused on the delayed enhancement sequences. The histopathology tests showed a large area of intramyocardial hemorrhage in both, confirming the correlation between T2 hypointense signals and hemorrhage on the histologic study.

Figure 2. 3D TurboFlash IR sequence, small axis view: late myocardial enhancement and no-reflow area with hypointense signal of the interventricular septum, one week after infarction.

Figure 3. Same sequence, long axis view: late myocardial enhancement corresponding to a lower infarction with a small subendocardial no-reflow band (arrow).
Infarction complications identifiable by MRI

Ventricular remodeling and cardiac dysfunction

Major changes to the myocardium after MI include:

• the transformation of the necrotic area into a fibrous scarred area with the risk of ventricular remodeling when scarring is extensive (i.e., 15–20% of the total LV myocardial mass);
• myocardial hypertrophy in the peri-infarction area;
• angiogenesis of the fibrous area as well as modifications of the extracellular matrix [19].

Moreover, we also know that the size of the infarction is a good predictor of development of dilated ischemic cardiomyopathy (Fig. 4). When the infarction involves between 17 to 20% or more of the left ventricle, myocardial remodeling evolves toward increased telediastolic and telesystolic volumes and dilated heart failure. The prognosis of these patients is linked to the extent of the LV dysfunction, which can best be analyzed by MRI, in view of the morphologic modifications associated with episodes of MI.

Thrombus of the cardiac cavity

Patients with ischemic dilated cardiomyopathy have an increased risk of thrombosis in the dilated cavity, associated with reduction of their LVEF and related to kinetic disorders of the infarcted and peri-infarction area. This reduction causes stasis of the blood in the left ventricle, which can activate coagulation processes. Mollet et al. [35] used MRI to study 57 patients with an acute or chronic infarction or ischemic heart disease and showed that the delayed enhancement sequences permitted identification of more and smaller thrombi than transthoracic echocardiography. These investigators [35] described the thrombi of the delayed enhancement sequences as intraventricular formation on hypointense signals, well defined, surrounded by well-enhanced blood (Fig. 5). They stressed that thrombi were significantly more frequent in the regions where the segmental myocardial contractility was the most abnormal and/or in the areas of delayed enhancement. Finally, they showed that the delayed enhancement sequences were more sensitive for the detection of thrombi and allowed visualization of smaller thrombi than the images of cine-MRI SSFP or transthoracic echocardiography. Recently, Weinsaft et al. [36] used cardiac MRI to assess the prevalence and risk factors of thrombus formation in 784 patients with systolic dysfunction (LVEF < 50%). In this series, the prevalence of LV thrombi was 7%. This prevalence was five times higher in patients with ischemic cardiomyopathy than in those with non-ischemic cardiomyopathy. Moreover, this prevalence was proportional to the reduction of LVEF in patients with both ischemic and non-ischemic heart disease. They [36] identified a low LVEF and myocardial scarring (with transmurality greater than 50%) as independent risk factors of intracardiac thrombus. During the 6-month follow-up of this study, the incidence of embolic cerebrovascular events was 5.6% in patients with a LV thrombus compared with 2.1% in patients without such a thrombus.

Postinfarction arrhythmias

The heterogeneity around the infarction area itself has been studied in patients who were to receive implantable cardioverter defibrillators after an infarction. This study showed that the size of this peri-infarction area is predictive of ventricular arrhythmias [37]. The existence and enhancement of the peri-infarction area have been controversial in the literature. Nonetheless, a recent study showed the critical prognostic role of the extent of this infarction area in the onset of arrhythmia. The authors defined the peri-infarction area as the area where the signal measured on delayed enhancement sequences was from two to four standard deviations of that of normal myocardium [38]. The identification of the peri-infarction area is important for guiding treatment aimed at increasing vascularization and for preventing myocardial remodeling [39]. Similarly, the presence around the infarction of a gray border area on the delayed enhancement sequences and its extent are also major prognostic factors for postinfarction mortality in these patients [38].
Ventricular aneurysms

The true aneurysm is a subacute complication of infarction, occurring most often after an extensive and transmural infarction around the anterior interventricular artery. We talk of aneurysm when the fibrous wall of the infarcted area is the site of dyskinesia with parietal dilatation. It is composed of the endocardium, fibrous tissue that replaces the myocardium, and the pericardium. It has a wider neck than pseudoaneurysms (Fig. 6). The wall of the true aneurysm is enhanced after the injection of contrast product. It may benefit from surgical treatment that consists of inserting a prosthetic patch in the contractile area, which would simultaneously exclude the aneurysm and restore the physiological LV form and volumes. MRI is the examination of choice for pretreatment assessment before this type of intervention. Nonetheless, in 2009, Jahnke reported that MRI and transesophageal ultrasound were similarly effective for assessing ventricular mass and volumes and for this presurgical assessment [40]. MRI remains the most effective imaging modality for this indication, because of its capacity to provide pertinent information about the aneurysm for the surgeon, but also to assess LV function with good reproducibility before and after surgery [41–43].

Pseudoaneurysms

Myocardial free wall rupture is a catastrophic complication of infarction that is responsible for 4% of postinfarction deaths and found in 23% of patients who die from infarction [44]. The pseudoaneurysm occurs in the rare cases where the ventricular rupture is contained by adherent pericardial wall or scar tissue: the rupture of the pericardial partition (Fig. 7a, b). It occurs in the acute postinfarction phase, and the neck is very narrow. Contrast product does not enhance the wall of a pseudoaneurysm. These lesions may be complicated by rupture, by heart failure when the pseudoaneurysm volume is large, or by thrombosis/embolism. Treatment of a pseudoaneurysm requires surgery and the placement of a Dacron patch. In 2005, Mousavi et al. reported that the sensitivity and specificity of MRI was superior to that of transthoracic echocardiography for the diagnosis of a ventricular pseudoaneurysm. MRI thus occupies a place of choice in the study of this complication [45].

Finally, MRI is an effective tool in the case of postinfarction mitral insufficiency, for it facilitates the study of ischemia and of the rupture of the papillary muscles.

Interventricular communication

Interventricular communication (IVC) is an acute and serious complication of MI. The mortality rate in patients with a postinfarction IVC treated medically is assessed at 80 to 90% in the first two months after occurrence. Surgical treatment is delicate since this lesion occurs in an area of myocardial necrosis. MRI has a major interest in the management of these patients because it allows the demonstration of both IVC and the extent of the necrotic area. That is, MRI makes it possible to analyze the edges of the IVC, for surgical sutures are particularly difficult when necrotic. In such a case, surgery is delayed if possible to await fibrosis of the necrotic tissue. These data are essential for management and treatment decisions. Several teams use MRI for pretreatment assessment of septal ruptures [46].
Figure 8. 3D TurboFlash sequence at a late time after injection, 4-chamber view. Pericardial effusion and contrast uptake (arrow).

Dressler syndrome

It consists of a pericardial inflammation several days postinfarction in 3 to 4% of cases, which can be accompanied by pericardial and sometimes also pleural effusion, with chest pain. Besides the effusion, MRI shows contrast uptake in the pericardium (Fig. 8) [47].

Lipomatous metaplasia

It corresponds to the aging of the scar, which gains fat over time. It is not a complication but rather an evolution of the infarcted area, corresponding to the infiltration of fat into the infarction scar. Its frequency increases with age and infarction size, and is higher among men and patients with a coronary bypass [48]. It can be shown easily by MRI, especially by fat saturation, or by CT.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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