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Cardiovascular Magnetic Resonance of Acute Myocardial Infarction at a Very Early Stage

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OBJECTIVES Very early changes in myocardial tissue composition during acute myocardial infarction (AMI) are difficult to assess in vivo. Cardiovascular magnetic resonance (CMR) imaging provides techniques for visualizing tissue pathology.

BACKGROUND The diagnostic role of CMR in very acute stages of myocardial infarction is uncertain. We investigated signal intensity changes beginning within 60 min after acute coronary occlusion in patients undergoing therapeutic septal artery embolization.

METHODS We investigated eight patients with hypertrophic obstructive cardiomyopathy undergoing interventional septal artery embolization by applying microparticles to reduce left ventricular outflow tract obstruction. In a clinical 1.5-tesla (T) CMR system, we visualized infarct-related myocardial signal by T_1-weighted sequences before and 20 min after administration of contrast media (delayed enhancement) and edema-related signal by T_2-weighted spin-echo sequences before and 58 ± 14 min after the intervention as well as on days 1, 3, 7, 14, 28, 90, and 180 during follow-up.

RESULTS Infarct-related changes as defined by contrast enhancement were observed as early as 1 h after the intervention and during six months of follow-up. In contrast, infarct-related myocardial edema, as visualized by high signal intensity in T_2-weighted spin-echo sequences, was not consistently detectable 1 h after acute arterial occlusion; this was possible in all subsequent studies until day 28.

CONCLUSIONS Contrast-enhanced magnetic resonance imaging detected infarct-related signal changes as early as 1 h after AMI in humans, whereas the sensitivity of edema-related signal changes was not sufficient during this very early stage. (J Am Coll Cardiol 2003;42:513–8) © 2003 by the American College of Cardiology Foundation

The early and reliable diagnosis of acute myocardial infarction (AMI) has a strong impact on clinical outcomes (1). Current clinical routine is mostly based on medical history, physical status, the electrocardiogram (ECG), and serologic findings. Although the overall sensitivity of these parameters is good (2,3), it is less favorable early after the onset of ischemia (4). However, in the diagnosis of myocardial infarction (MI), speed is essential, and in selected patients with equivocal results, the knowledge of very early changes could be helpful in therapeutic decision-making.

Cardiovascular magnetic resonance (CMR) is able to differentiate infarcted from normal myocardium (5–7) and also detects myocardial edema by T_2-weighted pulse sequences (8,9). However, in most animal and human studies (10,11), the changes were assessed at least 24 h after the acute injury. We investigated the signal intensity changes in the very early stages of MI after acute occlusion of a coronary artery.

METHODS

Patients. We studied eight patients (5 men and 3 women; 41 to 78 years old) with hypertrophic obstructive cardiomyopathy scheduled for interventional septal artery embolization. All patients had severe symptoms with dyspnea (New York Heart Association [NYHA] functional class III or IV), despite optimized pharmacologic treatment, and fulfilled the accepted criteria for undergoing this intervention. Significant coronary artery disease was excluded by cardiac catheterization. All patients gave written, informed consent. One patient was embolized a second time three months after the first procedure because severe symptoms and a high pressure gradient persisted.

Septal artery embolization. The intervention was performed by infusion of a solution containing microparticles (Contour Emboli; Target, Boston Scientific Corp., Boston, Massachusetts) into the septal artery supplying the basal septum as identified by intra-procedural contrast-enhanced echocardiography. The infusion resulted in acute embolic occlusion of the artery with complete cessation of flow, thus closely matching the pathophysiology of thrombotic occlusion in atherosclerotic AMI.

CMR imaging. We used a dedicated CMR system (Signa CV/*i*; 1.5 T, GE Medical Systems, Milwaukee, Wisconsin) located close to the cardiac catheterization laboratory and intensive care unit. Patients were continuously monitored during the examination by ECG, repeated blood pressure measurements, and pulse oximetry.

We used a four-element, phased-array coil or body coil. A T_2-weighted spin-echo sequence (short T_1 inversion recovery [STIR], repetition time [TR] 2 RR interval, echo time [TE] 64 ms, slice thickness 10 mm, matrix 256 × 256, field

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Abbreviations and Acronyms

AMI = acute myocardial infarction
CMR = cardiovascular magnetic resonance
ECG = electrocardiogram/electrocardiographic/electrocardiography
MI = myocardial infarction
MR = magnetic resonance
NYHA = New York Heart Association
STIR = short T_1 inversion recovery
TE = echo time
TI = inversion time
TR = repetition time

of view 34 × 34 or 36 × 36 mm) was used to visualize myocardial edema. Irreversible myocardial injury was visualized by T_1-weighted sequences (TE 23 ms, slice thickness 10 mm, matrix 256 × 160, field of view 36 × 36 cm) 20 min after intravenous application of 0.2 mmol/kg gadolinium–pentetic acid (Magnevist, Schering AG, Berlin, Germany). In three patients, we also assessed myocardial necrosis, as defined by "delayed enhancement," using a T_1-weighted, inversion-recovery prepared gradient echo sequence (TR 5.5 ms, TE 1.4 ms, inversion time [TI] 220 to 250 ms, slice thickness 10 mm).

We calculated the signal intensity ratio of the infarcted myocardium compared with remote myocardium and the proportion of the infarcted tissue related to the entire myocardium in the infarct-containing slice.

The left ventricular ejection fraction was quantified in a set of contiguous gradient-echo images (TE 4.8 to 6.1 ms, slice thickness 10 mm) in the true short-axis orientation.

Cardiovascular magnetic resonance was performed before and 58 ± 14 min after the intervention as well as on days 1, 3, 7, 14, 28, 90, and 180. One patient (who underwent 2 interventions) had an additional study 7 h after the intervention.

Care was taken to reproduce the same slice position in the follow-up studies, using anatomic landmarks.

Statistics. The infarct area during follow-up was compared with remote areas using an analysis of variance (Statview, SAS Inc., Cary, North Carolina) and as a post hoc analysis Fisher exact test.

RESULTS

All patients could be investigated without complications in a routine clinical setting.

Septal artery embolization procedure. All septal ablation procedures were successful, as defined by an immediate drop in the pressure gradient and subsequent enzyme release. No major complications occurred. Creatine kinase activity peaked at 570 ± 213 U/l (normal range <170 U/l for women and <200 U/l for men) after the intervention and normalized in all subjects within five days. In four patients, the ECG demonstrated signs of anteroseptal MI as defined by ST-segment elevations ≥0.2 mV in at least two adjacent chest wall leads (generally leads V_2 to V_4).

At the end of the follow-up period, the clinical status had improved in all patients (mean NYHA class 3.1 ± 0.1 vs. 1.3 ± 0.2, $p < 0.0001$).

T_2-weighted CMR. Before the intervention, the T_2-weighted images did not show any myocardial signal intensity abnormalities in the basal septal region. Also, within 1 h after the intervention, there were no relevant intramyocardial signal intensity changes in T_2-weighted images. In some patients it was necessary to differentiate intramyocardial signal from slow flow (Fig. 1). Therefore, a gradient-echo sequence was used to verify the endocardial border. Also, in the one patient with an additional CMR study 7 h after the intervention, no significant edema was detectable at that time point. However, 24 h after the intervention, a signal increase in the infarct area was visible in seven of eight patients. On days 3, 7, 14, and 28, these signal changes were visible in all patients, whereas after 90 and 180 days they had disappeared.

Contrast-enhanced CMR. The course of signal intensity changes in late contrast-enhanced T_1-weighted CMR images differed from that of T_2-weighted CMR images. The investigated regions had no contrast enhancement before the intervention. The signal intensity ratio between septal and remote myocardium increased from 1.01 ± 0.01 before the intervention to 1.84 ± 0.19 1 h after the intervention ($p < 0.05$). A localized enhancement persisted during the entire follow-up period (Figs. 1 and 2). The intensity and extent of the localized signal increase varied between the patients.

The area of the infarcted region during follow-up was significantly larger between days 1 and 14 than at all other time points ($p < 0.02$) in the late contrast-enhanced images. Furthermore, significant changes in the T_2-weighted CMR images were observed. The area of the increased STIR signal was larger than that of the late contrast-enhanced images, but this difference did not reach statistical significance (Fig. 2). After 180 days, the area of the signal increase in the late contrast-enhanced images appeared more sharply defined and smaller than on day 7.

Functional analysis. In all patients, a persisting regional hypokinesia in the infarcted region was observed, as quantified in gradient-echo CMR image series. The global left ventricular function did not change (ejection fraction: 68 ± 4% vs. 69 ± 4%, p = NS).

DISCUSSION

Our data show that contrast-enhanced CMR is able to visualize even small myocardial infarcts in vivo at a very early stage, whereas T_2-weighted CMR may not be sufficiently sensitive.

To our knowledge, this is the first report on CMR in patients early after an embolic event. There is a case report using CMR to visualize microvascular obstruction after

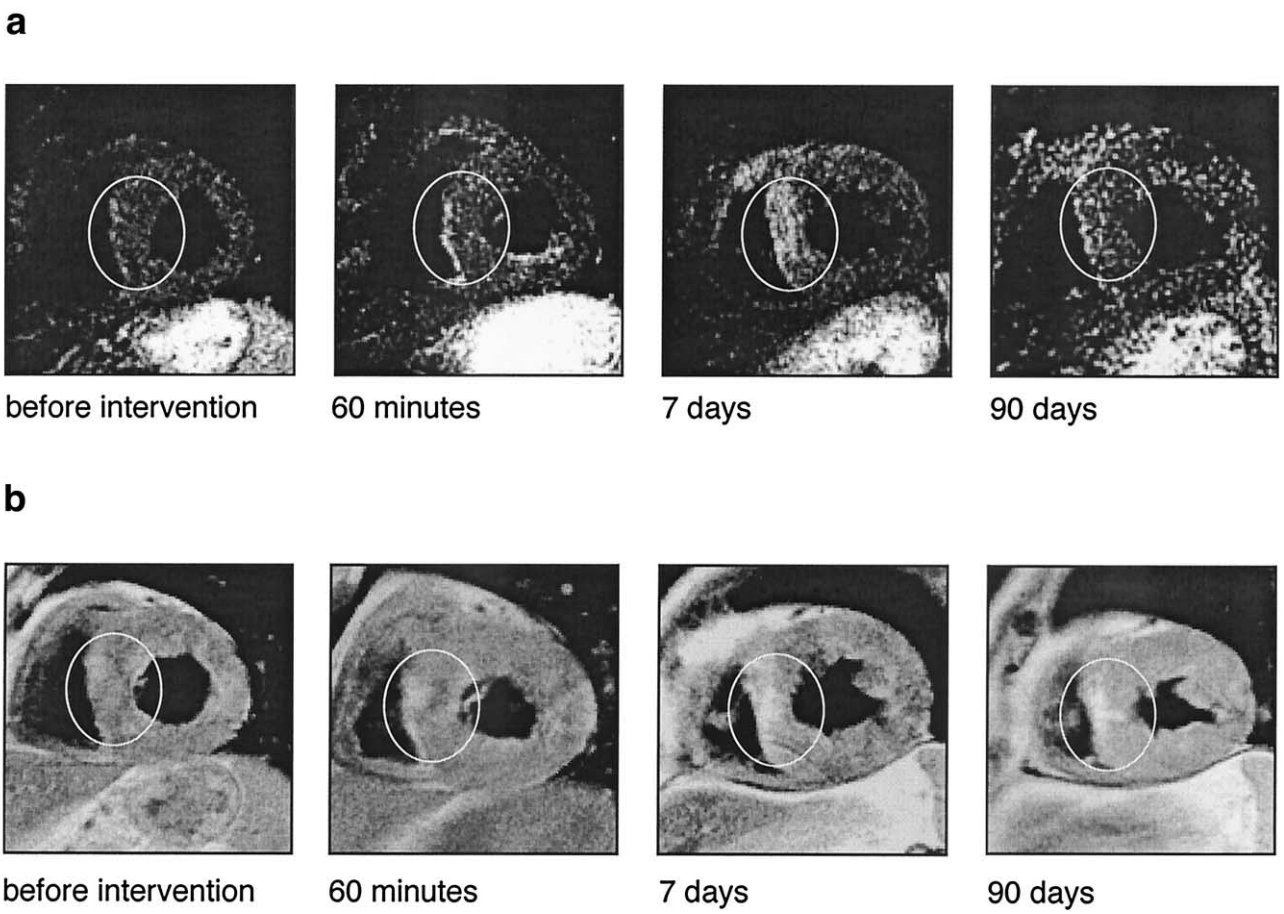

Figure 1. Signal changes in the septal myocardial infarction in a follow-up detected by magnetic resonance imaging. **(a)** T_2-weighted spin-echo sequences visualizing the edema. Sixty minutes after acute occlusion of the septal artery, there was no signal increase in the myocardium; the streaky artifacts visible adjacent to the septum and in the inferior parts in both ventricles are most likely due to slow flowing blood. **(b)** T_1-weighted spin-echo imaging after gadolinium. Enhancment was detectable within 60 min of infarction.

septal artery embolization (12). However, the ablation method in that case differed from ours by the use of alcohol.

Our findings of infarct-related contrast enhancement correspond to histologic evidence of the presence of myocardial necrosis as early as 40 min after arterial occlusion (13,14). The same studies showed that the infarct subsequently expands, a finding known as the "wave front phenomenon" (13). This is associated with the development of intracellular and interstitial myocardial edema.

In our approach, ablation of the septum was performed by applying micro-emboli consisting of foam gel, without any alcohol. Thus, an embolic event is created, providing a pathophysiology very similar to AMI. Thus, our model differs from classic infarcts by a more peripheral location of the occluding substrate and thus collateral flow. However, a better collateral flow, albeit reducing the infarct size, is unlikely to change the pathophysiology in the more central parts of the infarcted area. Thus, we are convinced that our model closely matches at least most parts of an atherosclerotic, but ultimately, no-flow infarction.

There are many studies on magnetic resonance (MR) findings in animal models of AMI. Although animal studies have provided significant insight into early ischemic myocardial injuries, studies in humans remain a necessity, considering the different profiles of myocardial ischemic injury in humans compared with other species. Our data may provide the information that is lacking by using a human model that closely matches the embolic event of AMI.

Magnetic resonance studies in animals showed an increase of T_1 and T_2 relaxation times 3 h after occlusion (15).

Higgins et al. (16) demonstrated in an animal model of AMI that the increase in T_2 signal of the infarcted regions reflects infarct-associated myocardial edema. Further studies on animal models confirmed the relationship between a high T_2 signal pattern and myocardial edema and further demonstrated that the spatial extent of myocardial edema exceeds that of irreversible myocardial injury (17). It was shown that diffusion-weighted MR imaging, but not T_2-weighted MR imaging, is sensitive to intracellular edema (18). Thus, it is very likely that the high signal intensity in our setting is reflective mainly of interstitial edema. In humans with subacute MI, Miller et al. (19) have shown that the function of myocardial segments showing a high STIR signal may recover, suggesting a mismatch between

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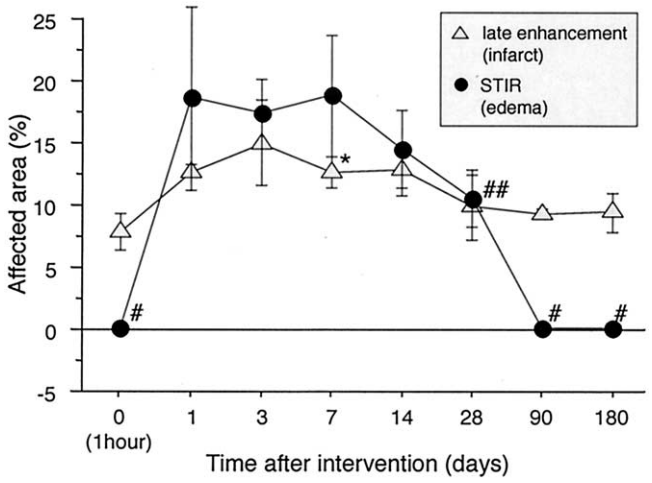

Figure 2. Changes over time in the infarcted and edematous areas. The affected area (%) = (area with signal abnormality/area of whole myocardium within slice) × 100. *Significant difference from day 0 (1 h after intervention) and days 28, 90, and 180 ($p < 0.05$). #Significant difference from days 1, 3, 7, 14, and 28 ($p < 0.05$). ##Significant difference from days 3 and 7 ($p < 0.05$). STIR = short T_1 inversion recovery.

myocardial edema and necrosis. Although myocardial edema occurs in irreversibly injured myocardium, it frequently also extends to reversibly injured myocardium and was shown to closely match the myocardial "area at risk" (9).

Our results indicate a differential time course of contrast enhancement, reflecting irreversible myocardial injury, and a high T_2 signal intensity, reflecting myocardial edema with a delay in the occurrence of T_2-related changes. Thus, for very early detection in a clinical setting, this approach does not seem to be sufficiently sensitive.

These results correspond to several animal studies showing that infarct-related myocardial edema was not detectable within the first hour before, but by 3 h after, occlusion (9,15,20). Different mechanisms may explain this. It is known that early in the course of ischemic injury myocardial edema is predominantly intracellular, and T_2-weighted CMR may not be able to detect bound water molecules in the case of intracellular edema.

Later on, however, a T_2-related high signal was consistently observed. There are no published in vivo human data on patients with edema in the very early stages of MI, but an increased signal intensity in T_2-weighted images was reported for infarcted regions three days after MI, which could be correlated with myocardial edema (21,22). We also found evidence of edema one day after AMI, consistent with these results. The edema persisted for at least one month but was not detectable after three months.

In contrast to T_2 changes, T_1 abnormalities due to MI are typically not visually detectable on MR images without using contrast agents.

On the other hand, CMR imaging late after administration of gadolinium (delayed enhancement) accurately reflects irreversible myocardial injury. In contrast to T_2-weighted images, only irreversibly damaged myocardium is enhanced. Thus, it is conceivable that the spatial extent of delayed enhancement would be less than that of STIR-determined myocardial edema.

Reperfusion injury early after experimental MI has been assessed by contrast-enhanced CMR (23,24), and there are numerous studies indicating that contrast-enhanced CMR is able to differentiate between infarcted and viable myocardium (6,25,26). The agreement of gadolinium accumulation with histologic parameters of myocardial necrosis was shown in animal CMR studies as early as 6 h after AMI (27,28).

The underlying pathophysiology of gadolinium accumulation in MI, however, has not been fully elucidated. Mechanisms under discussion include a prolonged wash-in/wash-out period in the infarcted tissue due to contrast media diffusion into necrotic cells, as well as a relative increase of interstitial space and interstitial edema (29,30). Thus, the contribution of edema, by comparison with necrosis or fibrosis, to the accumulation of gadolinium is difficult to estimate.

Interestingly, we observed a transient spatial increase of the enhancing area in the contrast-enhanced T_1-weighted images during the period with edema, as defined by high signal intensity areas in the T_2-weighted images, indicating a common underlying pathophysiology. Different mechanisms may explain this finding: it has been previously shown that in humans, the time needed for the infarct to reach its maximum size is about 6 to 12 h, compared with 3 to 4 h in dogs. Furthermore, gadolinium–pentetic acid also accumulates in edema (31), and thus our finding may simply reflect the involvement of infarct-associated edema. Finally, recent reports have underlined the role of apoptosis in the pathophysiologic cascade of irreversible ischemic injury. It is assumed that the apoptotic process achieves its final spatial extent a few days after the ischemic insult.

In patients with two infarctions, the more recent acute infarct revealed more blurred edges of the infarct-related necrosis than the chronic scar. Furthermore, signal intensity increased to a lesser extent. A clear discrimination of both infarcts, however, was possible only by the presence or absence of the infarct-associated edema (Fig. 3).

Thus, a delayed contrast enhancement without concomitant edema may be a marker of either necrosis during the first hours of infarction or chronic myocardial scar (after infarct-related inflammation has disappeared).

Clinical implications. Our results underscore the potential role of CMR in a number of clinical scenarios. Verifying the diagnosis of AMI is useful in the critical care setting, when conventional methods are inconclusive or contradictory and a therapeutic decision is urgently required. In addition, differentiation between acute and chronic infarcts is also possible. In the case of known infarcts, their age can be estimated: the finding of delayed enhancement in the absence of a corresponding high STIR signal intensity would exclude an acute event.

Finally, the unique ability of CMR to provide a noninvasive, reproducible means of assessing the temporal

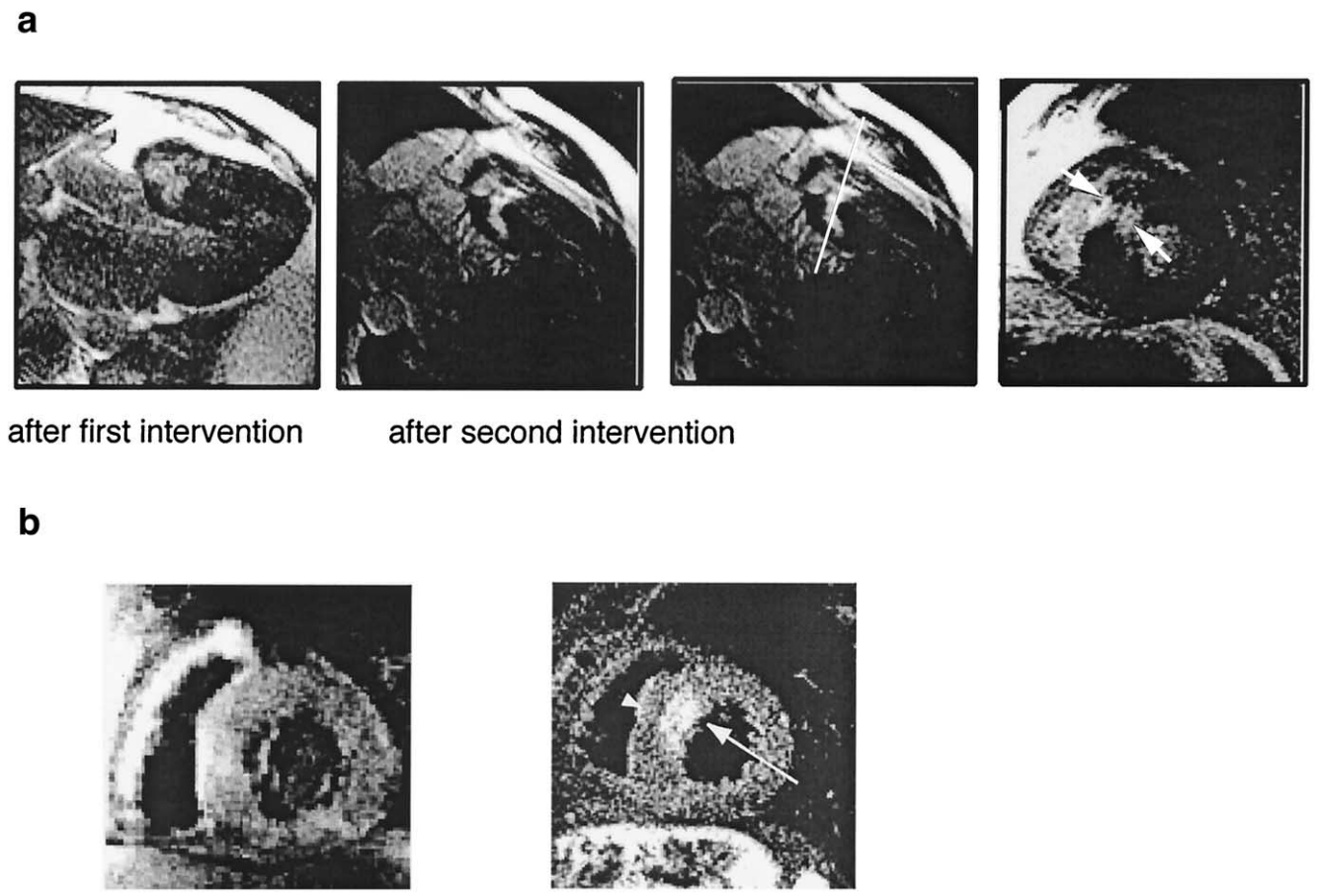

Figure 3. Contrast-enhanced CMR images. **(a)** First image = long-axis view after the first intervention; second image = long-axis view after the second intervention, with enlarged contrast accumulation; third image = long-axis view with planned short-axis orientation; fourth image = short-axis view showing a high signal intensity in the chronic infarction **(arrowhead)** and a lower, but still high, signal intensity in the acute infarction **(arrow)**. **(b)** T_2-weighted images: short-axis view after the first intervention **(left)** and short-axis view after the second intervention **(right)**. In contrast to the chronic infarction **(arrowhead)**, the acute infarction shows a very high signal **(arrow)**.

evolution and spatial extent of various myocardial ischemic injuries may justify its application as a tool for monitoring the effect of novel therapeutic approaches targeting these injuries.

Study limitations. Our study was performed on a specific patient population without chronic coronary artery disease. Although septal artery embolization produces an acute, non-reperfused MI, the injurious profile of this rather "controlled" myocardial insult may differ from the complex pathophysiology underlying clinical AMI.

We also did not register images, and despite our efforts to accurately reproduce the previous position with the help of anatomic landmarks, a slight shift of the position may have occurred.

Finally, because of the time we started our investigations, no inversion-prepared T_1-weighted gradient-echo sequence, as is usually used today for late enhancement CMR, was available. However, our experience with patients in whom both sequences were applied, did not reveal any significant differences between our and the newer techniques. Although newer techniques have a better contrast–noise relation, T_1-weighted sequences in general reveal the post-contrast signal changes we observed.

Conclusions. Contrast-enhanced T_1-weighted CMR is a sensitive method of detecting myocardial tissue damage even within 1 h after an acute embolic MI in humans, whereas T_2-weighted CMR was found to be not sufficiently sensitive at this early time point.

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