Quantitative Measurement of Infarct Size by Contrast-Enhanced Magnetic Resonance Imaging Early After Acute Myocardial Infarction

Comparison With Single-Photon Emission Tomography Using Tc\textsuperscript{99m}-Sestamibi

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OBJECTIVES

The aim of this research was to evaluate kinetics and extent of myocardial contrast enhancement (CE) in comparison with single-photon emission computed tomography (SPECT) early after acute myocardial infarction (AMI).

BACKGROUND

Quantification of infarct size serves as a surrogate end point in evaluating new therapies of AMI. Contrast-enhanced magnetic resonance imaging (CeMRI) of the myocardium is a promising new method for identification of irreversible tissue injury.

METHODS

A total of 33 patients were examined by CeMRI and SPECT 7±2 days after AMI and successful coronary intervention. After gadolinium-diethylenetriamine pentaacetic acid injection (0.2 mmol/kg), continuous short-axis slices of the left ventricle (LV) were acquired every 7 min up to 42 min using different inversion times (TI). Myocardial CE at each imaging time point was quantified and compared with corresponding SPECT perfusion defect.

RESULTS

All patients showed myocardial CE in the infarct region. A constant TI for CeMRI resulted in a decrease of signal intensity and extent of CE on late acquisitions. With TI adjustment, infarct image intensity peaked at 21 min with a contrast of 478% of remote myocardium and remained at this level up to 42 min after contrast injection (437%); CE extent was stable over time and agreed well with SPECT within an average difference of 3% of the LV myocardium, yielding the best correlation at 28 min (r = 0.86).

CONCLUSIONS

In patients after AMI and successful reperfusion, CE is stable over time and matches well with SPECT perfusion defect; CeMRI under standardized conditions can accurately assess myocardial infarct size in vivo and may be attractive for serving as a surrogate end point early after AMI. (J Am Coll Cardiol 2005;45:544–52) © 2005 by the American College of Cardiology Foundation

Noninvasive assessment of myocardial infarct size is an important clinical goal in patients with coronary artery disease because of its known prognostic value (1). After acute myocardial infarction (AMI) it allows risk stratification and evaluation of myocardial salvage, which has an important impact for patient management due to the differentiation of reversible from nonreversible tissue injury. Several methods have been introduced for measurement of infarct size in humans including electrocardiography, cardiac enzyme levels, and noninvasive imaging techniques such as echocardiography (2). However, all of them provide indirect parameters that may not specifically delineate nonreversible tissue injury.

Nuclear imaging techniques are currently accepted and validated clinical tools for the quantification of infarct size (3). Several animal and clinical studies have shown that \textsuperscript{99m}Tc-sestamibi single-photon emission computed tomography (SPECT)-assessed myocardial perfusion defects during coronary occlusion provides an estimate of myocardium at risk. Subsequently, injection of \textsuperscript{99m}Tc-sestamibi several days after coronary reperfusion allows delineation of the “final” infarct size in close correlation to pathology specimens from both laboratory animals and humans (4,5); SPECT is being used as an end point in various clinical trials as a measure of the efficacy of reperfusion therapy (6). Quantitative infarct size measured by \textsuperscript{99m}Tc-sestamibi has also been shown to predict subsequent mortality after reperfusion therapy (7).

Recently, contrast-enhanced magnetic resonance imaging (CeMRI) has been applied to identify myocardial necrosis, which represents a promising alternative to established nuclear methods providing a high-resolution delineation of infarct extent (8–17). However, the clinical role of delayed enhancement in acute and subacute myocardial infarction (MI) is much less defined than it is in patients with chronic MI. Studies in animals performed one to two days after

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reperfusion infarction showed that regions of enhancement after administration of extracellular contrast agents such as gadolinium-diethylenetriamine pentaacetic acid (Gd-DTPA) are associated with irreversible ischemic injury (8,9), while other studies showed that myocardial enhancement overestimates the infarct zone by 10% to 20% (10). Experimental comparison of Gd-DTPA with necrosis-specific porphyrin-based contrast agents have produced controversial results for the quantitative assessment of infarct size (11,12).

Conflicting results were also reported in patient studies conducted within one week after AMI, which have shown that regions of enhancement can recover contractile function two months later, whereas other investigators observed no improvement of enhanced regions (13,14).

Many factors may contribute to the disparate results published so far including species differences, methodological aspects, or timing of image acquisition after contrast injection, thus suggesting the need for standardized protocols for the assessment of infarct size (18). The purpose of this study was to examine the time course of myocardial contrast enhancement (CE) in patients with reperfused AMI seven days after myocardial injury and to compare the extent of CE with the size of perfusion defects determined by Tc-99m-sestamibi SPECT at the same time.

METHODS

Patient population. A total of 33 consecutive patients (61 ± 12 years of age; 26 men) with first time AMI (onset of symptoms <48 h) and successful coronary reperfusion therapy by coronary stenting on the day of admission were included in the study. Diagnosis of infarction was based on acute chest pain, characteristic electrocardiogram changes, elevated creatine kinase (CK), and angiographically demonstrated partial or complete occlusion of the infarct-related artery. Patients with previous MI or contraindication to MRI (pace-maker or claustrophobia) were excluded from the study. All subjects were examined 7.0 ± 1.5 days after reperfusion therapy. The study protocol was reviewed and approved by the local ethics committee. Written informed consent was obtained before inclusion in the study.

Imaging protocols. MRI. Patients were examined in a supine position with a 1.5-T tomograph (Siemens Sonata, Erlangen, Germany) equipped with a dedicated cardiac phased-array surface coil. Contiguous end-diastolic short-axis slices of the left ventricle (LV) were acquired from base to apex using an electrocardiogram-gated segmented TrueFISP inversion recovery sequence for sensitive assessment of inversion time (TI) changes. Three slices (slice thickness: 8 mm) per breath hold were acquired at end expiration (6 to 10 s), altogether 12 to 15 slices per study to cover the entire LV. Image parameters were as follows: repetition time (TR) 2.3 ms, excitation time (TE) 1.4 ms, image matrix 256 × 128 interpolated to 256 × 256, field of view 450 mm, flip angle 60°.

After a baseline acquisition, a bolus of 0.2 mmol Gd-DTPA per kg body weight (Magnevist, Schering-AG, Berlin, Germany) was injected into a peripheral vein. Complete LV image sets were acquired every 7 min until 42 min after injection (imaging time points: 7', 14', 21', 28', 35', 42' min), using four different TIs (250, 300, 350, 400 ms) at every time point. In order to null normal myocardium, all datasets were analyzed on a patient-by-patient basis to define the best TI at every imaging time point. Based on this adjustment, the mean TI increased with duration of examination (7': 290; 14': 297; 21': 331; 28': 344; 35': 375; 42': 390 ms).

SPECT. Resting SPECT studies were performed within 24 h of CeMRI. All patients received an intravenous injection of 250 MBq of technetium-99m-sestamibi; SPECT acquisitions were performed with a triple-head camera system (MultiSPECT 3, Siemens Medical Systems, Erlangen, Germany) equipped with low-energy, parallel-hole collimators 30 min after tracer application. Images were acquired in a 64 × 64 data matrix with an acquisition time of 40 s per projection. The image data were reconstructed over 180° from 45° right anterior oblique to 45° left posterior oblique by use of a Butterworth filter with a cutoff frequency of 0.45, order 5.

Data analysis. MRI. Quantitative analysis of MRIs were performed by a computer program developed at our institution (MunichHeart/MRI). Epip- and endocardial contours of the entire LV were manually traced. Subsequently, the mean transmural circumferential signal intensity of every short-axis slice—excluding the LV outflow tract—was automatically calculated within 36 segments (10° spacing). The circumferential profiles were transformed into a polar map (432 sub-segments) for all acquired image sets. To avoid partial volume effects, the most apical slice was excluded from analysis. Signal intensity of remote and enhanced myocardium was determined in a representative slice over time. The size of myocardial enhancement at each time point was quantified using a threshold analysis with normalization to remote myocardium, which was set to

### Abbreviations and Acronyms
- AMI = acute myocardial infarction
- CE = contrast enhancement
- CeMRI = contrast-enhanced magnetic resonance imaging
- CK = creatine kinase
- EF = ejection fraction
- Gd-DTPA = gadolinium-diethylenetriamine pentaacetic acid
- LV = left ventricle/ventricular
- MI = myocardial infarction
- MRI = magnetic resonance imaging
- SPECT = single-photon emission computed tomography
- TI = inversion time
Thresholds of >160%, >175%, >200%, >225%, >250% were applied to quantify the extent of hyperenhancement as percentage of the LV.

SPECT. Image analysis was performed by operators who were unaware of clinical information and MRI results. A polar map approach developed in our laboratory and validated with phantom measurements was employed (19).

For quantitative assessment a volumetric sampling tool was applied to create polar maps of identical size representing the relative distribution of activity throughout the LV. The spatial sampling of the LV from apex to base was matched to the selection of the MRI short-axis slices. Each polar map was normalized to its own maximal value. The size of defect was calculated with the use of a threshold of 50% as derived from phantom studies and was quantified as percentage LV (6).

Statistics. Mean and SD are given for all continuous data. To compare differences between signal intensity and enhancement size for different TIs as well as CK levels depending on prior fibrinolysis, a two-tailed t test was used. Linear regression analysis was used for the comparison of enhancement size by CeMRI with SPECT perfusion defect, peak CK, and LV ejection fraction (EF). Further comparison of CeMRI and SPECT was performed by Bland-Altman analysis. A value of p < 0.05 was considered statistically significant.

RESULTS

Study population. Table 1 summarizes the clinical data of the patient population. A total of 15 patients had anterior, 16 had inferior, and 2 had lateral MI; 8 patients underwent intravenous fibrinolysis before catheterization. Successful coronary intervention including stent implantation of the infarct-related artery (left anterior descending artery n = 15, right coronary artery n = 15, left circumflex artery n = 3) was performed in all 33 patients on the day of admission; 13 patients had one-vessel, 6 had two-vessel, and 14 had three-vessel disease. Left ventricular EF, assessed by ven-

Table 1. Patients’ Characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yrs)</th>
<th>Gender</th>
<th>Infarct Location</th>
<th>Coronary Artery Disease</th>
<th>Infarct-Related Artery</th>
<th>Peak CK (U/l)</th>
<th>EF (%)</th>
<th>Infarct Size SPECT (%LV)</th>
<th>Infarct Size MRI (%LV)</th>
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All (mean ± SD) 61 ± 12 867 ± 477 48 ± 12 15 ± 15 15 ± 13

Magnetic resonance imaging infarct size was measured at 28 min after contrast using an adjusted inversion time and a threshold of >200% remote.

ANT = anterior; CK = creatine kinase; EF = ejection fraction; INF = inferior; LAD = left anterior descending artery; LAT = lateral; LCX = left circumflex artery; LV = left ventricle; MRI = magnetic resonance imaging; RCA = right coronary artery; SPECT = single-photon emission computed tomography.
triculography during reperfusion therapy, was 48 ± 12%.
Peak CK level after infarction averaged 867 ± 477 U/l in all
patients (744 ± 395 U/l without, 1,223 ± 512 U/l with
prior fibrinolysis; p = 0.01).

CeMRI. SIGNAL INTENSITY. Figure 1 shows CeMRI ex-
amples of a representative slice in different infarct regions
over time. Myocardial infarction was correctly localized by
CE in all patients. A total of 7 min after injection of contrast
agent, infarct regions already displayed a clear increase of signal
intensity compared with remote myocardium. Using different
TI strategies showed a moderate effect on signal intensity in
remote myocardium. A marked signal decrease in enhanced
regions on later acquisitions was observed if the TI was held
constantly at 300 ms over time (Fig. 2A). As compared with
remote, the signal intensity for both strategies peaked at 21
min after contrast (478% vs. 477%) and remained at 437% after
42 min for TI adjustment but decreased to 358% for a constant
TI (Fig. 2B, Table 2).

MYOCARDIAL CE. Figure 1 displays the extent of hyperen-
hancement within infarcted myocardium of three patients
over time. Enhancement was detectable 7 min after injec-
tion of contrast agent. Extent of enhancement varied over
time independent of the chosen threshold (Fig. 3A). The
mean extent peaked at 14 min after contrast and subse-
quently decreased. Figure 3B shows the influence of TI on
the enhancement extent for a threshold of >200% of remote
myocardium. For MRI performed using an adjusted TI, the
extent of enhancement varied from 18.2 at 14 min to 12.7%
LV at 42 min. In contrast, when TI was held constant at
300 ms, the enhancement extent significantly decreased
after the 28th min.

Comparison of MRI CE with measures of infarct size.
SPECT. Figure 4 shows examples of the extent of myo-
cardial CE and the corresponding SPECT perfusion defect
in different infarct regions.

The SPECT perfusion defect within infarct regions
averaged 15.2 ± 15% of the LV with a mean Tc-99m-
sestamibi uptake of 40 ± 9%. Quantification of the extent
of myocardial CE based on a threshold of >160% and >175%
of remote myocardium overestimated, whereas a threshold
of >225% and 250% underestimated, the mean SPECT
perfusion defect. Using a threshold of >200% showed the
best agreement of enhancement extent with measurements
of perfusion defect by SPECT (Figs. 3A and 3B).
Contrast-enhanced magnetic resonance imaging (CeMRI) was performed with an adjusted inversion time and quantified based on a threshold of about 200% of remote myocardium. Over time, peak creatine kinase (CK) and left ventricle ejection fraction (EF) as measures of infarct size yielded lower correlations than SPECT perfusion defect (Table 2). For CK and EF, the correlation coefficients ranged from $r = 0.57$ to 0.71 and $-0.25$ to $-0.58$, respectively. Figure 6 shows the correlation for both parameters with the myocardial enhancement size 28 min after contrast injection using a threshold of about 200% of remote myocardium.

### DISCUSSION

All patients with reperfused AMI displayed myocardial CE within infarcted regions, and all infarcts were correctly localized by CeMRI seven days after the acute event. The results of this study demonstrate that the signal intensity and extent of enhanced regions varies with time after contrast injection and with the chosen TI. Using an adjusted TI, CeMRI infarct size was stable over time after contrast injection and matched well with the myocardial defect size by Tc$^{99m}$-sestamibi SPECT for a threshold of about 200% of remote myocardium. A fixed TI of 300 ms allowed comparable assessment until 21 min after contrast injection but underestimated SPECT perfusion defect on later acquisitions; CeMRI can accurately assess myocardial infarct size, but standardization of the imaging protocol is required.

**Pathophysiology of CE in MI.** Myocardial enhancement by extracellular contrast agents like Gd-DTPA have been shown to occur in different conditions of myocardial tissue damage. The exact relationship of the observed hyperenhanced regions to the underlying pathophysiology has been a subject of debate; Gd-DTPA is biologically inert and passively diffuses throughout the extracellular space with a half-time in blood of approximately 20 min (20). In chronic MI, CE represents irreversible injured tissue, which has been shown in animal and patient studies (8,21). However, the mechanism of delayed enhancement in acute and subacute MI is less defined.

Myocardial enhancement is a function of delivery, distribution, and washout of contrast agent within infarcted tissue. The delivery to areas of MI is dependent on the

### Table 2. CeMRI Myocardial Enhancement Over Time in Comparison With SPECT Perfusion Defect and Other Parameters of Infarct Size

<table>
<thead>
<tr>
<th>Time After Contrast Injection (min)</th>
<th>7'</th>
<th>14'</th>
<th>21'</th>
<th>28'</th>
<th>35'</th>
<th>42'</th>
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<tr>
<td>Signal intensity (% remote)</td>
<td>434 ± 152</td>
<td>475 ± 156</td>
<td>478 ± 182</td>
<td>474 ± 151</td>
<td>447 ± 198</td>
<td>437 ± 146</td>
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<td>Enhancement size (% LV)</td>
<td>17.5 ± 18</td>
<td>18.2 ± 15</td>
<td>16.6 ± 13</td>
<td>14.7 ± 13</td>
<td>12.9 ± 10</td>
<td>12.7 ± 10</td>
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<td>CeMRI-SPECT (% LV)</td>
<td>2.3 ± 13</td>
<td>3.0 ± 9</td>
<td>1.4 ± 9</td>
<td>-0.6 ± 8</td>
<td>-0.8 ± 7</td>
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<td>CeMRI vs. SPECT ($r$ value)</td>
<td>0.70</td>
<td>0.84</td>
<td>0.82</td>
<td>0.86</td>
<td>0.77</td>
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<td>Enhancement size vs. CK ($r$ value)</td>
<td>0.57</td>
<td>0.61</td>
<td>0.60</td>
<td>0.60</td>
<td>0.71</td>
<td>0.70</td>
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<td>Enhancement size vs. EF ($r$ value)</td>
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<td>-0.58</td>
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Contrast-enhanced magnetic resonance imaging (CeMRI) was performed with an adjusted inversion time and quantified based on a threshold of about 200% of remote myocardium. CK = creatine kinase; EF = ejection fraction; LV = left ventricle; SPECT = single-photon emission computed tomography.
The patency of the infarct-related artery and on the microvascular perfusion. Once the contrast media reaches the infarcted tissue, it distributes into the extravascular space. Evidence suggests that concentrations of contrast agents and the partition coefficient are increased in regions of acute infarction (22). This can be explained by an increase of distribution volume for the contrast agent, which may be caused by myocyte necrosis, edema, or inflammation with increased capillary permeability. In a previous study, it has been demonstrated that regions of hyperenhancement were associated with sarcomere membrane rupture examined by electron microscopy (15). After infarction, the loss of sarcomere membrane integrity, which is thought to be tightly related to cell death (23), allows Gd-DTPA to passively distribute into previous intracellular space. This affects myocardial Gd-DTPA kinetics because additional time is required for contrast molecules to diffuse in and out of isolated breaks in the cellular membrane. This has been shown for other markers with similar molecular weight as Gd-DTPA after myocyte death (24) as well as in AMI, where marked changes of wash-in and washout kinetics of Gd-DTPA within rim and core regions of infarcted tissue were observed as compared with normal myocardium (15).

**Quantification of myocardial enhancement.** For the quantitative assessment of myocardial enhancement, we developed a polar map approach similar to myocardial perfusion imaging with nuclear imaging techniques accounting for the three-dimensional geometry of the LV (19). To compare CeMRI to SPECT studies with lower spatial resolution, myocardial enhancement was only analyzed transmurally. Enhanced regions were normalized using a threshold analysis in comparison with remote myocardium, which served as a reference region in numerous other CeMRI studies (8–10,12,15). Contrast thresholds >200% of remote myocardium (100%) yielded best agreement with 50% threshold used in SPECT studies for assessment of perfusion defects. As shown in this study, the defined threshold affects the size of enhancement substantially and underlines the importance of standardization and calibration of analysis because different authors have introduced different cutoff values (8,9,12,15).

**Time course of CE early after reperfused AMI.** The results of this study show that the signal intensity and extent of hyperenhancement changed over time after contrast injection. All patients examined in this study had a successful reperfusion of the infarct-related artery ensuring comparable delivery of contrast agent. Recently, Oshinski et al. (18) demonstrated that the size of the enhanced region varies with the time imaging is performed after Gd-DTPA injection in a rat model of reperfused AMI. They found that the true infarct size as assessed by TTC-staining was overestimated by 20% to 40% immediately after contrast injection and that the time for the enhanced region to correspond to the true infarct size was \(21 \pm 4\) min. Because of the use of contrast agents such as Gd-DTPA with complex diffusion properties, myocardial enhancement as a marker of tissue injury is a time-dependent process. It reflects the underlying pathophysiology of different tissue conditions within the infarcted regions. Close inspection of the methodology of different studies reveals substantial differences in acquisition timing after injection of contrast agent (time intervals of 5 up to 60 min), which might...
explain the disparate results concerning accurate assessment of infarct size by CeMRI (8–14).

Results of this study further show that the extent of enhancement over time is dependent on the chosen TI. As the blood concentration of Gd-DTPA decreases from 2.06 to 0.77 mmol/l from 1 to 40 min after injection, the correct TI in order to null normal myocardium increases and has, therefore, to be adjusted (20). If the TI is kept constant, the periphery of the hyperenhanced region might pass through a zero-crossing, thereby affecting its apparent size. We found that if the TI was held constant, the extent of enhancement decreases on later acquisitions. However, using an adjusted TI, the signal intensity was stable until 42 min after contrast injection.

Comparison of myocardial enhancement with clinical measures of infarct size. We demonstrated that myocardial enhancement size moderately correlates with LV EF as assessed by left ventriculography during reperfusion therapy and peak CK levels. This reflects the difficulties for accurate evaluation of infarct size using methods that do not provide regional characterization of irreversible myocardial tissue injury (2).

Coronary artery reperfusion can dramatically change the washout kinetics of CK from myocardium, especially after thrombolysis as performed in a subgroup of our patients resulting in early and exaggerated peak enzyme levels and, thus, limiting the usefulness of CK curves as a measure of infarct size (25).

Single-photon emission computed tomography imaging with Tc\textsuperscript{99m}-sestamibi is currently the best available tool for infarct sizing in patients and has been used as surrogate end point in reperfusion trials after AMI (3). Tc\textsuperscript{99m}-sestamibi distributes according to myocardial blood flow and demonstrates a slow washout with minimal delayed redistribution even after flow is restored in an occluded artery (26).

There are only a few studies in humans comparing CeMRI with SPECT perfusion imaging for the assessment of infarct size after AMI (16,17). All these studies were performed using thallium-201 and showed that regions with hyperenhancement correlate well with fixed scintigraphic defects ($r = 0.69$ and $r = 0.92$).

However, there have been no direct quantitative comparison between CeMRI and Tc\textsuperscript{99m}-sestamibi SPECT after AMI using three-dimensional co-registration of both data sets.

A major advantage of CeMRI compared with currently used SPECT is the higher spatial resolution, which makes this technique suitable to detect small areas of subendocardial infarcts (27). Furthermore, CeMRI can be combined with other magnetic resonance techniques such as estimation of contractility by cine-MRI, magnetic resonance

\begin{figure}
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\caption{Comparison of contrast-enhanced magnetic resonance imaging (CeMRI) at 28 min after contrast and single-photon emission computed tomography (SPECT) perfusion defect with corresponding polar maps. Short-axis slices are orientated from apical (\textbf{left}) to basal (\textbf{right}). (\textbf{A}) Myocardial infarction of the inferior wall (day 6) with transmural contrast enhancement. (\textbf{B}) Anterior infarction (day 8) with subendocardial enhancement.}
\end{figure}
tagging, and first-pass perfusion, offering a multifunctional approach for the assessment of myocardial viability.

**Study limitations.** In this study, a segmented inversion recovery TrueFISP sequence was used to maximize acquisition speed with high contrast for continuous imaging. The maximal signal intensity of enhanced regions was approximately 475% of remote regions, which is comparable with results of other studies using spin-echo and gradient-echo sequences (15,18). A recently introduced fast-gradient echo sequence showed an increase of contrast-to-noise ratio, which may improve differentiation between injured and normal myocardium (28). However, as contrast characteristics for the used Gd-DTPA concentration are primarily T1-dominated, the results of our study should be applicable to other imaging techniques.

It has been shown that, with the TI >275 ms the magnetization of normal myocardium, blood in the LV and in enhanced regions was above zero and differences between regions were less evident (28). The constant TI of 300 ms we used was comparable with other studies, which observed agreement of hyperenhancement with extent of myocyte necrosis irrespective of reperfusion (8,28). Although a defined TI would simplify the procedure of CeMRI, TI adjustment resulted in a smaller decrease of signal and enhancement size over time.

Our analysis approach neglected the very apical portion of the LV in order to minimize partial volume effects. However, this was performed consistently in all MRI and SPECT studies. Therefore, the “true” infarct size may have been underestimated by both techniques.

In order to assume comparable pathophysiologic conditions for the interpretation of CE in this study, only patients with successfully performed coronary intervention of the infarct-related artery were included. Results obtained may not be applicable to patients without reperfusion therapy after AMI because contrast kinetics may be different if the infarct-related artery is occluded and contrast agent cannot directly reach the infarct region.

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**Figure 5.** Comparison of contrast-enhanced magnetic resonance imaging (CeMRI) at 28 min after contrast (threshold >200% of remote myocardium) and single-photon emission computed tomography (SPECT). (A) Correlation of enhancement size with SPECT perfusion defect. (B) Agreement between both modalities depicted in a Bland-Altman graph (mean value of the differences = solid line; ±2 SD = dotted line). Average value of the two measurements is plotted along the x-axis; the difference is plotted along the y-axis. LV = left ventricle.

**Figure 6.** Myocardial contrast enhancement by contrast-enhanced magnetic resonance imaging (CeMRI) at 28 min after contrast (threshold >200% of remote myocardium) in comparison with peak creatine kinase (CK) level (A) and left ventricular (LV) ejection fraction assessed angiographically on the day of admission (B).
Conclusions. Contrast-enhanced MRI can identify and quantify the extent of infarct regions in close agreement to Tc$^{99m}$-sestamibi SPECT early after reperfused AMI. Due to the physiologic properties of extracellular contrast agents, standardized timing after contrast injection and acquisition is important for accurate assessment of infarct size. Contrast-enhanced MRI will be an attractive new method to serve as a surrogate end point in clinical studies similar to Tc$^{99m}$-sestamibi SPECT.

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