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Myocardial Salvage by CMR Correlates With LV Remodeling and Early ST-Segment Resolution in Acute Myocardial Infarction

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OBJECTIVES The purpose of this study was to assess the association of myocardial salvage by cardiac magnetic resonance (CMR) with left ventricular (LV) remodeling and early ST-segment resolution in patients with acute myocardial infarction (MI).

BACKGROUND Experimental studies revealed that MI size is strongly influenced by the extent of the area at risk (AAR), limiting its accuracy as a marker of reperfusion treatment efficacy in acute MI studies. Hence, an index correcting MI size for AAR extent is warranted. T2-weighted CMR and delayed-enhancement CMR, respectively, enable the determination of AAR and MI size, and the myocardial salvage index (MSI) is calculated by correcting MI size for AAR extent. Nevertheless, the clinical value of CMR-derived MSI has not been evaluated yet.

METHODS In a prospective cohort of 137 consecutive patients with acutely reperfused ST-segment elevation MI, CMR was performed at 1 week and 4 months. T2-weighted CMR was used to quantify AAR, whereas MI size was detected by delayed-enhancement imaging. MSI was defined as AAR extent minus MI size divided by AAR extent. Adverse LV remodeling was defined as an increase in LV end-systolic volume of ≥15%. The degree of ST-segment resolution 1 h after reperfusion was also calculated.

RESULTS AAR extent was consistently larger than MI size (32 ± 15% of LV vs. 18 ± 13% of LV, $p < 0.0001$), yielding an MSI of 0.46 ± 0.24. MI size was closely related to AAR extent ($r = 0.81$, $p < 0.0001$). After correction for the main baseline characteristics by multivariate analyses, MSI was a major and independent determinant of adverse LV remodeling (odds ratio: 0.64; 95% confidence interval: 0.49 to 0.84, $p = 0.001$) and was independently associated with early ST-segment resolution (*B* coefficient = 0.61, $p < 0.0001$).

CONCLUSIONS In patients with reperfused ST-segment elevation MI, CMR-derived MSI is independently associated with adverse LV remodeling and early ST-segment resolution, opening new perspectives on its use in studies testing novel reperfusion strategies. (J Am Coll Cardiol Img 2010;3:45–51) © 2010 by the American College of Cardiology Foundation

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Mortality is the best parameter for measuring the efficacy of reperfusion strategies in patients with acute myocardial infarction (MI) (1). Using this hard end point, a large sample is necessary to test novel treatments in combination with the existing reperfusion strategies that are already highly effective. Thus, other markers of reperfusion treatment efficacy are needed, and infarct size has often been used as a surrogate end point for mortality (2). This parameter, however, is influenced by several factors (3–6): 1) the size of the area at risk (AAR) (myocardium supplied by the culprit vessel); 2) residual flow to the ischemic territory (e.g., collateral flow); 3) myocardial metabolic demand; and 4) the duration of coronary occlusion. Small differences in the AAR may result in a significant variation of infarct size (5,6), underscoring the fact that most of the infarct size variability is due to the extent of the myocardium at risk. The myocardial salvage index (MSI) is calculated by correcting the amount of necrotic myocardium for the AAR extent, and it may be a better surrogate end point than infarct size (7–9).

ABBREVIATIONS AND ACRONYMS

AAR = area at risk
CMR = cardiac magnetic resonance
DE = delayed enhancement
ECG = electrocardiogram
FOV = field of view
LV = left ventricular
MI = myocardial infarction
MO = microvascular obstruction
MSI = myocardial salvage index
SI = signal intensity

In patients with MI, delayed-enhancement (DE) cardiac magnetic resonance (CMR) is a well-validated technique for the determination of the necrotic (acute phase) and scarred (chronic phase) myocardium (10). In experimental models, T2-weighted CMR enabled depiction of the myocardial edema in the salvageable AAR (11,12), and Friedrich et al. (13) reported promising data on myocardial salvage determination in acute MI patients. The purpose of this study was to investigate the clinical value of CMR-derived MSI by testing its association with 2 important clinical and prognostic parameters: left ventricular (LV) remodeling and early ST-segment resolution.

METHODS

Study population. Between May 2006 and September 2007, 137 consecutive acute MI patients (55 patients at the Monasterio Foundation, Pisa, Italy [Center A] and 82 patients at Gasthuisberg Hospital, Leuven, Belgium [Center B]), presenting with cumulative ST-segment elevation of ≥6 mm and treated with percutaneous coronary intervention within 12 h from symptom onset were prospectively studied by CMR at 1 week and 4 months. Exclusion criteria were critical stenosis (i.e., lumen narrowing of ≥75%) in vessels other than the infarct-related artery, previous MI or revascularization, atrial fibrillation, cardiogenic shock, or contraindication to CMR. The local ethics review boards approved the protocol, and written informed consent was obtained from each patient.

CMR protocol. Fifty-five patients were examined at Center A with a 1.5-T unit (CVi, GE Healthcare, Milwaukee, Wisconsin), and 82 patients at Center B with a 1.5-T unit (Intera CV, Philips Medical Systems, Best, the Netherlands). All studies were performed using dedicated cardiac software, phased-array surface receiver coil, and vectocardiogram triggering. LV volumes, mass, and function were assessed by breath-hold steady-state free-precession cine CMR. In the short-axis orientation, the left ventricle was completely encompassed by contiguous slices. The sequence parameters were field of view (FOV): 350 to 400 mm, repetition time (TR)/echo time (TE): 3.2/1.6 ms, flip angle: 60°, matrix: 224 × 192, slice thickness: 8 mm (CVi, GE Healthcare) and FOV: 350 to 400 mm, TR/TE: 3.6/1.8 ms; flip angle: 60°, matrix: 256 × 160, slice thickness: 8 mm (Intera CV, Philips Medical Systems).

AAR was determined using breath-hold T2-weighted short-TI inversion-recovery fast spin echo pulse sequence. In short-axis orientation, the left ventricle was entirely encompassed by contiguous slices. The sequence parameters were FOV: 380 to 400 mm; TR: 2 R-R intervals, TE: 100 ms, TI: 150 ms, matrix: 256 × 192, slice thickness: 8 mm (CVi, GE Healthcare) and FOV: 380 to 400 mm, TR: 2 R-R intervals, TE: 100 ms; TI: 150 ms, matrix: 256 × 256, slice thickness: 8 mm (Intera CV, Philips Medical Systems). After administration of 0.2 mmol/kg of gadolinium-tetraazacyclododecanetetraacetic acid, DE imaging was used to quantify infarct size and concomitant microvascular obstruction (MO) by breath-hold 3-dimensional (Intera CV, Philips) or 2-dimensional (CVi, GE Healthcare) segmented inversion-recovery gradient-echo pulse sequence. DE imaging was performed 8 to 20 min after contrast administration, and the inversion time was individually adapted to suppress the remote myocardium signal (typical range from 200 to 300 ms). Sequence parameters were FOV: 350 to 400 mm, TR/TE: 4.6/1.3 ms, flip angle: 20°, matrix: 256 × 192, slice thickness: 8 mm (CVi, GE Healthcare) and FOV: 350 to 400 mm TR/TE: 4.5/1.3 ms, flip angle: 15°, matrix: 256 × 128, slice thickness: 5 mm (Intera CV, Philips Medical Systems).

Image analysis. All CMR studies were analyzed offline using inhouse-developed cardiac software (CardioViewer, UZ Leuven, Belgium) by consensus of 2 experienced operators (J.B. and P.G.M.) who were

blinded to clinical data. Cine CMR was used to derive LV volumes, mass, and global function. Adverse LV remodeling was defined as an increase in LV end-systolic volume of ≥15% during follow-up.

On T2-weighted images, infarct-related edema was considered present when the signal intensity (SI) of the myocardium was >2 SD of the mean SI of the contralateral remote region. The AAR extent was obtained by manually tracing the hyperintense region on T2-weighted, short-axis images and expressed as LV percentage. When present, a hypointense area within the hyperintense myocardium was considered a hemorrhagic component of infarcted myocardium (14) and included in the AAR computation. The signal-to-noise ratio of injured and remote myocardium and the contrast-to-noise ratio were calculated by locating same-size regions of interest in injured and remote myocardium and in background noise, respectively (13). On DE imaging, MI was considered present if the SI of hyperenhanced myocardium was >5 SD of the mean SI of the remote region (15), and MO was defined as a hypoenhanced region within infarcted myocardium. MI size and, if present, MO extent were obtained by manually drawing the regions of interest and expressed as LV percentage. MI transmurality was computed by dividing the DE area by the total area of the corresponding myocardial wall and expressed as a percentage.

Electrocardiography. A 12-lead surface electrocardiogram (ECG) was obtained at hospital admission and 1-h after infarct-related artery recanalization. The sum of ST-segment elevation was measured 20 ms after the end of the QRS complex J point in leads I, aVL, and V_1 to V_6 for anterior MI, and leads II, III, aVF, V_5, and V_6 for nonanterior MI. ST-segment resolution was calculated as sum of ST-segment elevation on the first ECG minus the sum of the ST-segment elevation on the second ECG divided by the sum of ST-segment elevation on the first ECG and expressed as a percentage (16). Electrocardiographic analysis was performed by an operator who was blinded to clinical and CMR data.

Statistical analysis. Continuous and categorical variables were expressed as mean ± SD and frequency with a percentage, respectively. The Student-dependent *t* test and Wilcoxon test were used as appropriate to compare continuous variable differences between acute and chronic phases. Comparison of categorical variables was performed by using the chi-square test or the Fisher exact test if the expected cell count was <5. Linear regression analysis was used to test the correlation between continuous variables. Univariate linear regression and logistic regression analyses were used to determine the association of baseline variables respectively with early ST-segment resolution and adverse LV remodeling. Multivariate linear regression and logistic regression analyses with a forward selection procedure ($p < 0.05$ for entry; $p > 0.10$ for removal) were used to evaluate the influence of covariates, respectively, on early ST-segment resolution and adverse LV remodeling. Age, AAR extent, MO presence, MI transmurality, MSI, infarct location, baseline LV ejection fraction, and time to reperfusion were considered covariates. Because MI size was strongly associated with MSI ($r = -0.72$, $p < 0.0001$) and AAR extent ($r = 0.85$, $p < 0.0001$), MI size was not included in the models. Statistical analysis was performed by using SPSS software for Windows (version 12.0, SPSS Inc., Chicago, Illinois), and all tests were 2 tailed at a 5% significance level.

RESULTS

Study population. Baseline characteristics are summarized in Table 1. All infarct-related arteries were successfully stented with bare metal or drug-eluting stents. During follow-up, 2 patients were hospitalized for heart failure and 4 for recurrent angina, but no cardiac deaths or reinfarction occurred. All patients underwent CMR at 4 months.

Table 1. Baseline Characteristics (n = 137)

Age (yrs)	61 ± 12
Male	111 (81)
Cardiovascular risk factors	
Current smoker	46 (34)
Familial history of coronary artery disease	49 (36)
Diabetes mellitus	21 (15)
Hypertension	73 (53)
Hyperlipidemia	82 (60)
Time to reperfusion (min)	224 ± 123
Glycoprotein inhibitor IIb/IIIa	130 (95)
Infarct-related artery	
Left anterior descending artery	67 (49)
Right coronary artery	59 (43)
Left circumflex coronary artery	11 (8)
Maximum serum troponin I (μg/l)	92.4 ± 32.3
Medication at discharge	
Angiotensin-converting enzyme inhibitor	113 (82)
Angiotensin II inhibitor	16 (12)
Beta-blocker	119 (87)
Statin	122 (89)
Diuretics	13 (9)

Values are mean ± SD or n (%).

Table 2. Cardiac Magnetic Resonance Measurements

Variables	Acute Phase (1 Week)	Chronic Phase (4 Months)	p Value
LV end-diastolic volume (ml)	158 ± 38	167 ± 41	<0.0001
LV end-systolic volume (ml)	82 ± 24	87 ± 30	0.032
LV mass (g)	124 ± 29	111 ± 25	<0.0001
LV ejection fraction (%)	48 ± 8	50 ± 10	0.007
Myocardial infarct size (% of LV)	18 ± 13	—	—
Myocardial infarct transmurally (%)	72 ± 28	—	—
Area at risk (% of LV)	32 ± 15	—	—
Myocardial salvage index	0.46 ± 0.24	—	—
Microvascular obstruction	69 (50)	—	—
Microvascular obstruction extent (% of LV)	6 ± 9	—	—

Values are mean ± SD or n (%).
LV = left ventricle/ventricular.

CMR measurements. CMR measurements are summarized in Table 2. During follow-up, 40 patients experienced adverse LV remodeling. In all but 7 (5.1%) patients, myocardial regions with a high SI on T2-weighted images had evidence of DE on post-contrast imaging. In the 7 patients without evidence of DE, infarct-related myocardial edema involved the anterior (n = 5) and inferior (n = 2) LV walls, and ST-segment resolution was 99 ± 1% (range 96% to 100%) and troponin I peak level was 6.9 ± 5.8 μg/l (range 0.5 to 14.0 μg/l).

On T2-weighted imaging, hyperintense myocardium had invariably transmural distribution, which corresponded to diverse degrees of MI transmurality on DE images (Fig. 1). The edematous myocardium had a signal-to-noise ratio higher than that of remote myocardium (27.2 ± 11.3 vs. 12.5 ± 3.1; p = 0.013); the contrast-to-noise ratio between injured and remote myocardium was 14.7 ± 9.6. AAR extent correlated strongly with MI size (Fig. 2) and moderately with the baseline LV mass (r = 0.49, p < 0.0001).

MSI and predictors of adverse LV remodeling. MSI correlated positively with the baseline LV ejection fraction (r = 0.32, p < 0.0001) and inversely with AAR extent (r = −0.25, p = 0.008), LV end-diastolic (r = −0.18, p = 0.05) and end-systolic (r = −0.30, p = 0.002) volumes, and with LV end-systolic volume variation between the acute and chronic phases (r = −0.46, p < 0.0001). A strong inverse relationship was observed between MSI and MI size (r = −0.72, p < 0.0001), whereas MSI did not correlate with time to reperfusion (r = −0.11, p = 0.28). At univariate analysis, adverse LV remodeling was associated with anterior MI, the presence of MO, and greater AAR and infarct transmurality. Similarly, lower baseline LV ejection fraction and lower MSI predicted ventricular enlargement. At multivariate analysis, the MSI remained strongly associated with the occurrence of adverse LV remodeling (Table 3).

MSI and electrocardiographic findings. The sum of ST-segment elevations was 10.1 ± 4.3 mm and 4.9 ± 3.8 mm at admission and 1 h after reperfusion, respectively (p < 0.0001), yielding a ST-segment resolution of 5.2 ± 2.8 mm (57 ± 30%). At univariate analysis, ST-segment resolution correlated positively with baseline LV function and negatively with infarct transmurality and AAR extent. ST-segment resolution was strongly associ-

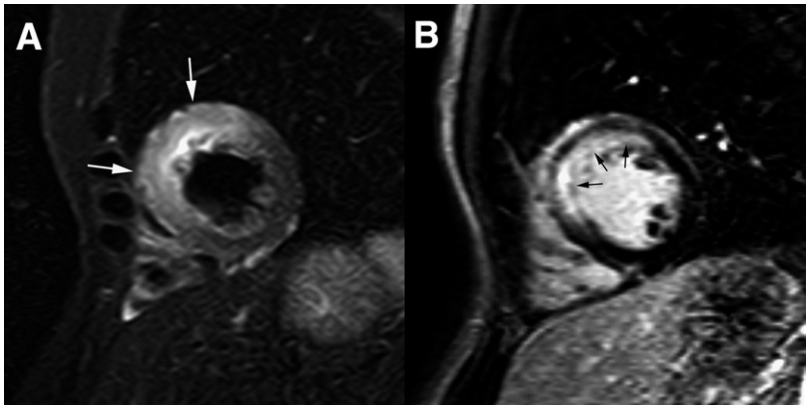

Figure 1. Infarct-Related Myocardial Edema and Delayed Enhancement in Acute Myocardial Infarction

T2-weighted short-TI inversion-recovery **(A)** and delayed enhancement **(B)** short-axis images of a reperfused anterior myocardial infarction. Infarct-related myocardial edema **(A, arrows)** has transmural extension, whereas delayed enhancement is mainly confined to the subendocardium **(B, arrows)**. In this case, the extent of the area at risk and that of myocardial infarction was 47 g and 24 g, respectively, yielding a myocardial salvage index of 0.49.

ated with MSI, and this result remained unchanged at multivariate analysis (Table 4).

DISCUSSION

Persistent occlusion of a coronary artery determines an irreversible injury that first involves the subendocardium and then extends as a wave front toward the subepicardium (17). Timely restoration of myocardial blood flow enables salvage of the ischemic but still viable myocardium. In concordance with earlier data (11–13), we demonstrated that CMR permits retrospective quantification of the myocardial salvage in patients with reperfused ST-segment elevation MI and that this index is a major determinant of 2 important clinical and prognostic parameters: ventricular remodeling and early ST-segment resolution.

Experimentally, post-reperfusion myocardial edema by T2-weighted CMR reproduced accurately the AAR quantified by fluorescent microspheres, the reference standard (12). In a cohort of 92 patients with acutely reperfused MI, Friedrich et al. (13) showed that comprehensive CMR permitted to derive the salvaged myocardium by combining DE and T2-weighted imaging. Our observations endorsed and expanded the previous ones by reporting that CMR-derived MSI was associated with adverse LV remodeling (18) and post-procedural ST-segment resolution (16,19,20). In particular, we found that MSI was a strong determinant of adverse LV remodeling, and this result remained unchanged at multivariate analysis after correction for important parameters, such as the occurrence of MO, MI transmurality, and baseline LV ejection fraction. Moreover, we demonstrated that MSI was closely and independently associated with early ST-segment resolution, paralleling the results of technetium-99m–sestamibi myocardial scintigraphy studies, which reported a close relationship among myocardial salvage, early ST-segment resolution (16), and cardiac mortality (9). In 5.1% of patients, we observed T2-weighted abnormalities without evidence of DE on post-contrast CMR. This result is consistent with an aborted MI, as also supported by almost complete post-procedural ST-segment resolution and a modest increase in the troponin I plasmatic level (21). The fact that T2-weighted "positive" myocardium can be DE "negative" strengthens the concept that T2-weighted and DE imaging are complementary techniques depicting, respectively, reversible and irreversible myocardial injury (10–13,22).

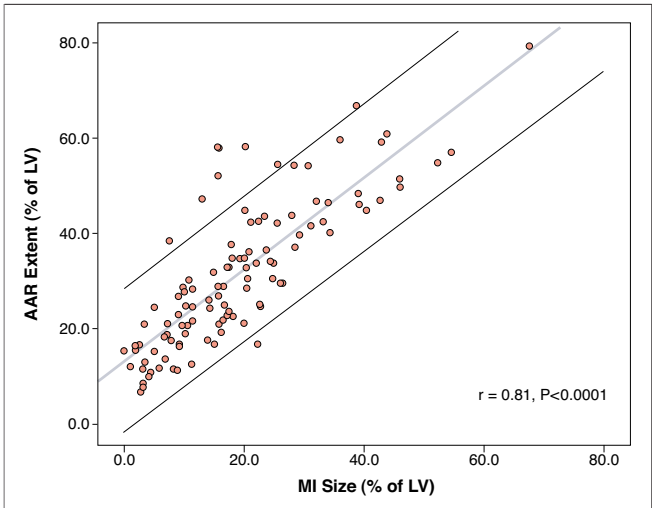

Figure 2. Scatterplot of AAR Extent and MI Size

Correlation and 95% confidence interval lines show the close linear relationship between the area at risk (AAR) extent and myocardial infarction (MI) size, underscoring that MI size is strongly influenced by the AAR extent (i.e., the amount of myocardium supplied by the MI-related artery). By extrapolation of the linear correlation line, a positive ordinate intercept is obtained implying that AAR extent exceeds MI size. LV = left ventricle.

Interestingly, we also observed a close linear relationship between AAR extent and MI size, albeit the former was greater than the final amount of myocardial necrosis. In concordance with experimental (5,6) and clinical (3) data, this finding underscores that AAR is a major determinant of MI size. In animal models, small variations in the occluded vessel resulted in a significant change in infarct size (6), and AAR accounted for >70% of the variability in the extent of myocardial necrosis (5). The close association of infarct size with AAR may limit the usefulness of the former as an end point in studies testing myocardial reperfusion strategies. MSI corrects the infarct size for

Table 3. Univariate and Multivariate Analyses for the Prediction of Adverse LV Remodeling

	Adverse LV Remodeling			
	Univariate		Multivariate	
Baseline Variables	OR (95% CI)	p Value	OR (95% CI)	p Value
MI transmurally (%)	1.04 (1.01–1.07)	0.005	—	—
AAR (% of LV)	1.04 (1.01–1.07)	0.003	1.04 (1.01–1.08)	0.001
MSI (for 0.10 increment)	0.58 (0.46–9.75)	<0.0001	0.64 (0.49–0.84)	0.001
Presence of MO	6.79 (3.55–18.06)	<0.0001	—	—
Time to reperfusion (min)	1.00 (0.99–1.00)	0.588	—	—
Age (for 10-yr increment)	1.22 (0.87–1.72)	0.241	—	—
Anterior vs. nonanterior MI	2.27 (1.02–5.04)	0.044	—	—
LV ejection fraction	0.92 (0.87–0.97)	0.003	—	—

AAR = area at risk; CI = confidence interval; LV = left ventricle/ventricular; MI = myocardial infarction; MO = microvascular obstruction; MSI = myocardial signal intensity.

Table 4. Univariate and Multivariate Analyses for the Prediction of Early ST-Segment Resolution

	Early ST-Segment Resolution (%)			
	Univariate		Multivariate	
Baseline Variables	*B*	p Value	*B*	p Value
MI transmurality (%)	−0.79	<0.0001	−0.31	0.002
AAR (% of left ventricle)	−0.48	0.007	—	—
MSI	0.85	<0.0001	0.61	<0.0001
Presence of MO	0.14	0.371	—	—
Time to reperfusion	−0.23	0.130	—	—
Age (yrs)	0.01	0.941	—	—
Anterior vs. nonanterior MI	−0.41	0.007	−0.16	0.020
LV ejection fraction (%)	0.45	0.011	—	—

Abbreviations as in Table 3.

the amount of AAR, yielding a marker with lower inherent variability, which can be particularly attractive in studying the efficacy and safety of new reperfusion approaches before proving them in large trials. CMR determination of MSI has also considerable advantages with respect to the well-validated nuclear techniques, such as technetium 99m-sestamibi myocardial scintigraphy (2,3,7–9,16). In fact, differently from MI size, which can be quantified by predischarge scintigraphy, AAR determination necessitates radioactive tracer administration before reperfusion treatment and image acquisition within the ensuing 8 h (7). This renders technetium-99m-sestamibi myocardial scintigraphy logistically impractical and also raises concerns about radiation exposure.

Study limitations. Although this study was conducted at 2 different centers using diverse vendor CMR, both centers followed carefully the same protocol, and all data were centrally analyzed. T2-weighted spin echo technique is particularly susceptible to signal loss in LV walls distant from the surface coil. However, both CMR units used an SI correction algorithm that made the signal uniform throughout the left ventricle. Our findings must be interpreted with caution in patients with nonreperfused or non–ST-segment elevation MI, and in those with severe adverse LV remodeling because this event was uncommon in our population. Given the small sample size and short follow-up, the MSI influence on clinical outcomes was not assessed. The small sample size and the fact that only 40 patients experienced adverse LV remodeling might have influenced the multivariate analyses results.

CONCLUSIONS

In reperfused ST-segment elevation MI patients, CMR-derived MSI is a major and independent determinant of 2 important clinical and prognostic parameters, such as LV remodeling and early ST-segment resolution. These findings may open new perspectives on the use of this index as a surrogate end point in studies testing novel reperfusion strategies.

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Key Words: area at risk ■ cardiovascular magnetic resonance ■ myocardial infarction ■ myocardial salvage.