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Correlation of Serial Cardiac Magnetic Resonance Imaging Parameters With Early Resolution of ST-Segment Elevation After Primary Percutaneous Coronary Intervention

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Background The aim of the present study was to determine whether the parameters of cardiac magnetic resonance imaging (CMRI) might correlate with early ST-segment resolution (STR) after primary percutaneous coronary intervention (PCI) in ST-elevation myocardial infarction (STEMI).

Methods and Results CMRI was performed in 45 STEMI patients (age: 56.6±13.0 years) at 8.2±8.0 days (early phase) and 3.3±1.1 months (late phase) after successful PCI. CMRI parameters were compared between 2 groups: ≥70% STR (group 1, n=21) and <70% STR (group 2, n=24). Both groups had similar baseline characteristics, except for a higher frequency of ≥2 myocardial blush grade and shorter pain-to-balloon time in group 1. Early-phase CMRI showed that persistent microvascular obstruction (PMO) (38.1% vs 91.7%, p<0.001) occurred less frequently and the percent infarct mass against total left ventricular (LV) mass (17.7±8.7% vs 29.1± 13.4%, p=0.001) was smaller in group 1. Late-phase CMRI revealed a significant increase in LV end-diastolic volume (–1.5±8.7 vs 14.5±25.5 ml, p=0.026) and reduced ejection fraction (55.0±9.9% vs 47.8±11.1%, p=0.027) in group 2.

Conclusions CMRI demonstrated that early STR might be related to PMO and infarct size, and predicts LV dysfunction and adverse LV remodeling. Also, early-phase CMRI findings are comparable to late-phase CMRI in association with early STR. (*Circ J* 2008; **72:** 1621 – 1626)

Key Words: Magnetic resonance imaging; ST-elevation myocardial infarction; ST-segment resolution

Among the many markers used to assess successful myocardial reperfusion after primary percutaneous coronary intervention (PCI) or thrombolysis therapy, the extent of resolution of ST-segment elevation is a simple noninvasive indicator of the outcome of infarcted myocardium[1] Previous studies using myocardial contrast echocardiography[2,3] or radioisotope scintigraphy[4–6] have demonstrated that early ST-segment elevation resolution (STR) after primary PCI or thrombolysis is associated with better salvage of reperfused myocardium. However, both the pathologic correlates and the mechanisms of early STR are relatively unknown. Recent advances in cardiac magnetic resonance imaging (CMRI) allow relatively precise assessments of myocardial perfusion, infarct size, left ventricular (LV) remodeling and function, with excellent correlation with histology in animal models of acute and chronic myocardial infarction (MI)[7,8] However, there are little data on the CMRI findings related to early STR after successful primary PCI. Therefore, we performed CMRI serially in the early and late phases after successful revascularization by primary PCI for treatment of MI. We sought to determine whether the morphologic and functional parameters of CMRI correlated with early STR after primary PCI for ST-elevation myocardial infarction (STEMI).

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Method

Subjects

We investigated 45 patients with acute STEMI in whom primary PCI was performed successfully within 12 h of symptom onset. Diagnosis of acute MI (AMI) was based on the presence of acute ischemic chest pain, electrocardiogram (ECG) findings (ST-segment elevation ≥0.1 mV in 2 or more contiguous leads), elevated serum cardiac biomarkers (cardiac troponin T >0.1 μg/L, creatine kinase-MB (CK-MB) fraction enzyme level >10 μg/L), and angiographic significant stenosis (≥50%) of a coronary artery. Exclusion criteria were unstable hemodynamic status, any history of previous MI, or contraindications to CMRI (eg, pacemaker implantation or claustrophobia). We also excluded patients with recurrent chest pain or anginal equivalent associated with ST-segment changes during the time period between primary angioplasty and second CMRI. All participants gave written informed consent to the study protocol.

ECG Analysis

A 12-lead ECG was performed before and 90 min after primary PCI. The sum of ST-segment elevation was mea-

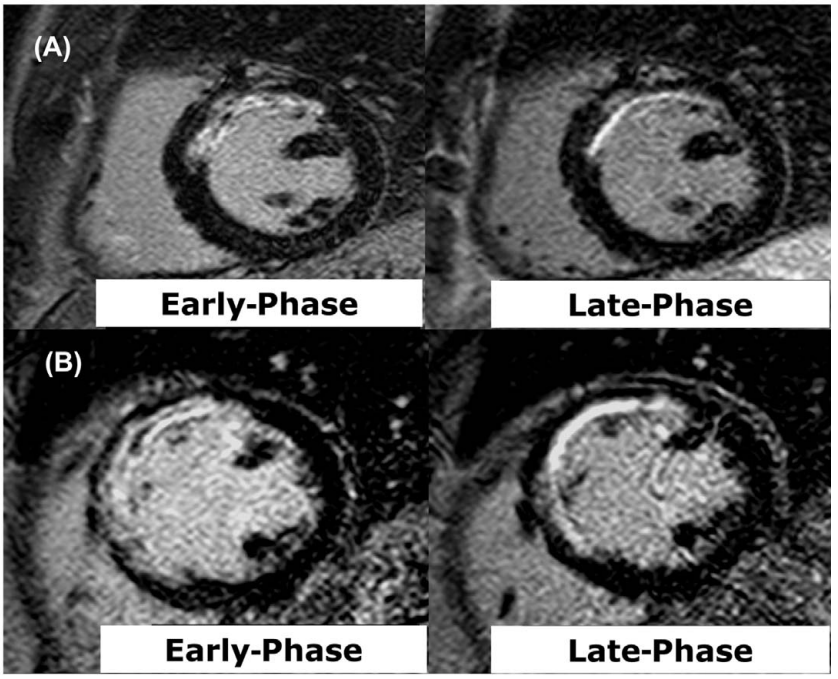

Fig 1. Non-transmural (<75%) anteroseptal hyperenhancement revealed in early-phase and late-phase study of group 1 (≥70% ST segment resolution, A). Transmural anterior and anteroseptal hyperenhancement (≥75%) revealed in early-phase and late-phase study of group 2 (<70% ST segment resolution, B).

Table 1 Baseline Patient Characteristics

	ST-segment resolution ≥70% (group 1, n=21)	*ST-segment resolution <70% (group 2, n=24)*	*p value*
Age (years)	*55.2±12.4*	*57.8±13.8*	*0.412*
Gender (M/F)	*17/4*	*23/1*	*0.169*
Diabetes mellitus	*5 (23.8%)*	*6 (25.0%)*	*0.926*
Hypertension	*12 (57.1%)*	*13 (54.2%)*	*0.841*
Pain-to-balloon time (min)	*207.3±81.9*	*340.4±279.9*	*0.033*
Multivessel disease	*17 (85.7%)*	*18 (70.8%)*	*0.296*
Infarct location			*0.213*
Anterior	*12 (57.1%)*	*18 (75.0%)*	–
Lateral	*2 (9.5%)*	*0 (0.0%)*	–
Inferior	*7 (33.4%)*	*6 (25.0%)*	–
Initial CK-MB (mg/dl)	*9.7±23.6*	*55.2±91.7*	*0.027*
Peak CK-MB (mg/dl)	*239.7±147.5*	*347.1±260.1*	*0.111*
hsCRP (mg/L)	*6.4±5.9*	*13.9±13.5*	*0.073*
TIMI 0 before PCI	*14 (70.0%)*	*19 (73.1%)*	*0.576*
MPG (≥2)	*18 (90.5%)*	*10 (41.7%)*	*0.001*
Medication			
Aspirin	*21 (100.0%)*	*24 (100.0%)*	*1.000*
Clopidogrel	*21 (100.0%)*	*24 (100.0%)*	*1.000*
Abciximab	*2 (9.6%)*	*4 (16.7%)*	*0.670*
ACE inhibitor	*14 (66.7%)*	*13 (54.2%)*	*0.393*
ARB	*5 (25.0%)*	*6 (23.8%)*	*0.926*
β-blocker	*21 (100.0%)*	*24 (100.0%)*	*1.000*
CCB	*7 (33.3%)*	*2 (8.3%)*	*0.061*
Statin	*19 (90.5%)*	*21 (87.5%)*	*1.000*

Data are mean value ± SD.
CK-MB, creatine kinase MB isoenzyme; hsCRP, high-sensitivity C-reactive protein; TIMI, Thrombolysis In Myocardial Infarction; PCI, percutaneous coronary intervention; MPG, myocardial blush grade; ACE, angiotensin-converting enzyme; ARB, angiotensin-receptor blocker; CCB, calcium-channel blocker.

sured 20 ms after the J point in leads $V_{1–4}$ for anterior, leads V_5, V_6, I and aV_L for lateral, and leads II, III, aV_F for inferior infarction. The ECG analysis was performed by 2 investigators who were unaware of the clinical and CMRI findings. Resolution of ST-segment elevation was calculated as the sum of ST-segment elevation on initial ECG minus the sum of ST-segment elevation on the second ECG, divided by the sum of ST-segment elevation on initial ECG, and expressed as a percentage. Based on the degree of STR, enrolled patients were assigned to either group 1 with complete early STR (n=21, STR ≥70%) or group 2 without complete early STR (n=24, STR <70%).[5,9]

Coronary Angiography

All angiographic findings were interpreted by 2 interventional cardiologists who were unaware of the CMRI and

Table 2 LV Volumes, LVEF, Infarcted Wall Thickness and Mass, and Persistent Microvascular Obstruction on CMRI According to the Extent of ST-Segment Resolution

	ST-segment resolution ≥70% (group 1, n=21)	*ST-segment resolution <70% (group 2, n=24)*	*p value*
LVEDV (ml)			
Acute phase	*157.1±30.5*	*149.3±29.9*	*0.391*
Chronic phase	*155.6±35.9*	*163.8±36.5*	*0.454*
ΔLVEDV	*–1.5±18.8*	*14.5±25.5*	*0.026*
LVESV (ml)			
Acute phase	*75.8±25.5*	*82.6±23.1*	*0.353*
Chronic phase	*71.9±25.8*	*87.3±30.7*	*0.076*
ΔLVESV	*–3.8±20.3*	*4.7±20.3*	*0.101*
LVEF (%)			
Acute phase	*52.2±9.9*	*45.0±9.7*	*0.020*
Chronic phase	*55.0±9.9*	*47.8±11.1*	*0.027*
ΔLVEF	*2.9±7.6*	*2.8±9.9*	*0.891*
Total wall thickness of infarcted LV segment (mm)			
Acute phase	*9.5±1.5*	*9.8±2.1*	*0.517*
Chronic phase	*7.7±1.4*	*6.5±1.6*	*0.009*
ΔLV wall thickness	*–1.8±1.1*	*–3.3±2.3*	*0.013*
Infarcted wall thickness (mm)			
Acute phase	*5.6±1.4*	*6.9±1.8*	*0.012*
Chronic phase	*3.8±1.1*	*3.8±1.0*	*0.716*
ΔInfarcted wall thickness	*–1.9±1.1*	*–3.1±1.9*	*0.019*
Relative infarcted wall thickness (%)			
Acute phase	*60.2±13.5*	*70.4±18.5*	*0.017*
Chronic phase	*49.4±12.9*	*62.3±16.1*	*0.008*
ΔInfarcted wall thickness	*–10.8±11.2*	*–11.2±17.0*	*0.936*
Total LV mass (g)			
Acute phase	*126.2±24.8*	*123.4±25.0*	*0.524*
Chronic phase	*113.5±21.8*	*109.9±24.8*	*0.426*
ΔLV mass	*–12.7±10.3*	*–13.6±21.7*	*0.682*
Relative infarct LV mass (%)			
Acute phase	*17.7±8.7*	*29.1±13.4*	*0.001*
Chronic phase	*12.5±6.3*	*22.6±12.3*	*0.001*
ΔInfarcted LV mass	*–5.2±4.9*	*–6.6±7.7*	*0.539*
Presence of PMO (n, %)			
Acute phase	*8 (38.1%)*	*22 (91.7%)*	*<0.001*
Chronic phase	*3 (14.3%)*	*9 (37.5%)*	*0.079*

Data are mean value±SD.
LV, left ventricular; LVEF, LV ejection fraction; CMRI, cardiac magnetic resonance imaging; LVEDV, LV end-diastolic volume; Δ, difference between acute and chronic phases; LVESV, LV end-systolic volume; LV mass, end-diastolic LV mass; PMO, persistent microvascular obstruction.
Relative infarcted wall thickness (%)=infarcted wall thickness/total wall thickness of infarcted LV segment.
Relative infarct LV mass (%)=infarcted LV mass/total LV mass.

ECG findings. In all patients, the infarct-related artery was treated with stent implantation and primary angioplasty was successful with final Thrombolysis In Myocardial Infarction (TIMI) grade 3 flow in the infarct-related artery and residual stenosis <30%. Myocardial blush grade (MBG) was assessed by 2 persons who were unaware of the clinical data (κ value of interobserver variation=0.90). MBG was identified according to a dye density score (0=no myocardial blush or persistent blush; 1=minimal blush; 2=moderate blush, but less than obtained during angiography of the contralateral or ipsilateral noninfarct-related artery; 3=normal myocardial blush)![10] All patients received aspirin 250 mg, clopidogrel 300 mg and intravenous heparin 70 IU/kg before PCI and were maintained on aspirin 100 mg and clopidogrel 75 mg for at least 6 months following PCI. Abciximab was administered during the procedure at the discretion of the interventional cardiologist.

Protocol of CMRI

CMRI was performed using a 1.5-T imaging unit (Gyroscan Intera, Philips Medical Systems, Best, The Netherlands) equipped with a dedicated cardiac software package, the synergy cardiac coil and a vectorcardiogram. Cine images were acquired in sequential short-axis slices covering the entire ventricles, using steady-state free precession technique (balanced turbo field echo sequence, TR=3.3 ms, TE=1.7 ms, flip angle=50°, field of view (FOV) 36 cm, matrix size=256×256, slice thickness=10 mm with no gap, 25 phases/cardiac cycle).

Delayed enhancement images were obtained by acquiring an inversion-recovery segmented gradient echo T1 weighted sequence (TR=2 heart beats, TE=1.5 ms, flip angle=15°, FOV=36 cm, matrix size=512×512, no. of signal average=2) 10–15 min after intravenous injection of 0.2 mmol/kg of gadolinium DTPA. Sequential short-axis slices of 10 mm in thickness were obtained and the presence of significant delayed hyperenhancement was assessed in 6 segments per slice corresponding to the coronary territory, with the exception of the most apical slice and the basal slice 10 mm below the aortic outflow tract.

Magnetic Resonance Imaging Data Analysis

LV ejection fraction (LVEF), LV end-diastolic wall thickness (LVEDWT), volume and mass were measured on

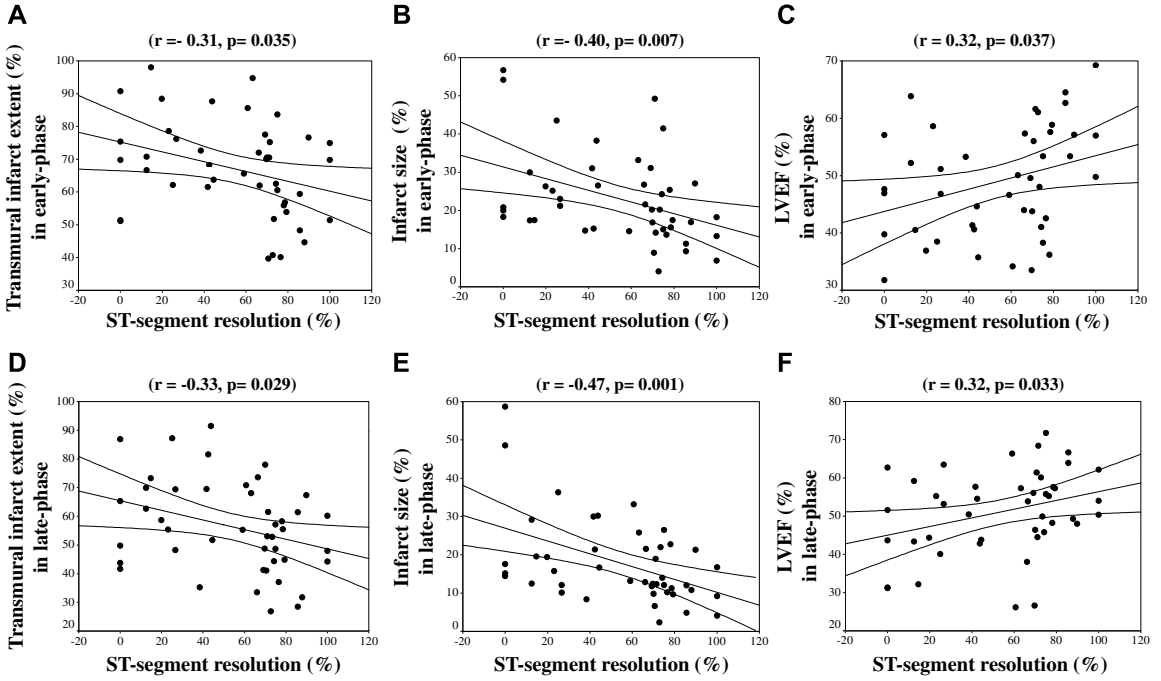

Fig 2. Correlations between the extent of ST-segment resolution and cardiac magnetic resonance imaging parameters of acute (A, B, C) and chronic phase (D, E, F); transmural infarct extent (A, D), infarct size (B, E) and left ventricular ejection fraction (LVEF; C, F). Infarct size, infarcted mass per total left ventricular mass in percent.

cine images by commercially available software (MASS, Medis, The Netherlands). Papillary muscles and pericardial fat were excluded from the calculations. LVEDWT was measured at the center of the infarct region. The early-phase and late-phase CMRI protocols consisted of cine and delayed enhancement imaging (Fig 1). Infarcted wall thickness was measured with enhanced segment of delayed enhancement imaging in the end-diastolic phase. Relative infarcted wall thickness (%) was defined as infarcted wall thickness/total wall thickness of infarcted LV segment.

To quantify the infarct size, hyperenhanced areas were manually traced on short-axis delayed hyperenhancement images, except for the most basal and apical slices, and measured by 2 experienced cardiologists and radiologists. Total mass was calculated as: myocardial area × sliced thickness × myocardial specific gravity of 1.05. Relative infarct LV mass (%) was defined as sum of the infarcted LV area/total LV area. Persistent microvascular obstruction (PMO) was defined as the hypoenhanced area within the hyperenhanced area.[11]

Statistical Analysis

Values are expressed as mean ± standard deviation. Comparison of the discrete variables was performed using the chi-square test or Fisher's exact analysis. Because all the variables showed skewed distribution, nonparametric tests were used to compare the differences between 2 groups. Also, Spearman's correlation test was used to identify the relationship of STR and CMRI parameters. Multiple linear regression was performed to identify the independent predictor(s) of change in the LV end-diastolic volume (LVEDV) using the variables with $p<0.1$ on simple linear regression and crucial variables. Statistical analysis was performed with SPSS 11.0 (SPSS Inc, Chicago, IL, USA). A p value <0.05 was considered statistically significant.

Results

Forty-five patients (mean age 56.6±13.0 years, 40 males) diagnosed with acute STEMI underwent primary PCI within 12 h (pain-to-balloon time 4.6±3.7 h) after the onset of chest pain. Baseline characteristics, shown in Table 1, were similar in both groups, except for a higher frequency of ≥2 MBG, shorter pain-to-balloon time and lower level of initial CK-MB at hospital arrival in group 1. The extent of STR at 90 min correlated inversely with the pain-to-balloon time ($r=–0.548$, $p<0.001$) and the initial CK-MB serum level at hospital arrival ($r=–0.443$, $p=0.009$). There was no difference in the extent of ST segment resolution according to the location of the STEMI (anterior wall 49.3±30.1% vs lateral wall 74.3±6.1% vs inferior wall 64.1±31.0%, $p=0.287$).

Relationship Between CMRI Parameters and Early STR in the Early-Phase

CMRI was performed in all patients at 8.2±8.0 days after primary PCI (early-phase) and again at 3.1±1.1 months (late-phase). All patients showed delayed contrast-enhancement on early phase CMRI. Immediately after infarction, delayed contrast-enhanced CMRI revealed significantly smaller infarcted myocardial mass and transmural infarct extent in group 1 (Table 2). However, LVEF was better preserved in group 1 (Table 2). PMO occurred less frequently in group 1 (Table 2). The extent of STR correlated with the transmural infarct extent ($r=–0.31$, $p=0.035$, Fig 2A), the infarct size expressed as percent infarct mass per total LV mass ($r=–0.40$, $p=0.007$, Fig 2B) and LVEF ($r=0.32$, $p=0.037$, Fig 2C).

Relationship Between CMRI Parameters and Early STR in the Late-Phase

Late-phase CMRI showed a decreased total wall thickness of the infarcted segment in all patients; however, the

reduction was significantly greater in group 2 (1.8±1.1 vs 3.3±2.3 mm, p=0.013, Table 2). The infarct size on late-phase CMRI was reduced in both groups, compared with early-phase CMRI. On late-phase CMRI, group 2 showed a significantly increased LVEDV, whereas it was unchanged in group 1. The LVEF was significantly lower in group 2 than in group 1. The extent of ST resolution showed correlations with the transmural infarct extent (r=–0.33, p=0.029, Fig 2D), the infarct size (r=–0.47, p=0.001, Fig 2E), the LVEF (r=0.32, p=0.033, Fig 2F) and extent of LVEDV (r=–0.31, p=0.044, Fig 3) on late-phase CMRI.

ST-Segment Change and LV Remodeling

Neither LVEDV nor LV end-systolic volume (LVESV) was statistically different between the 2 groups in the early-phase. On late-phase CMRI, group 2 showed a significantly increased LVEDV, whereas it was unchanged in group 1.

More than 70% ST resolution was an independent predictor for preventing adverse LV remodeling (R^2=0.427, p=0.036) after controlling for age, gender, hypertension, diabetes, multivessel disease, pain-to-balloon time and MBG and GpIIbIIIa inhibitor (Table 3).

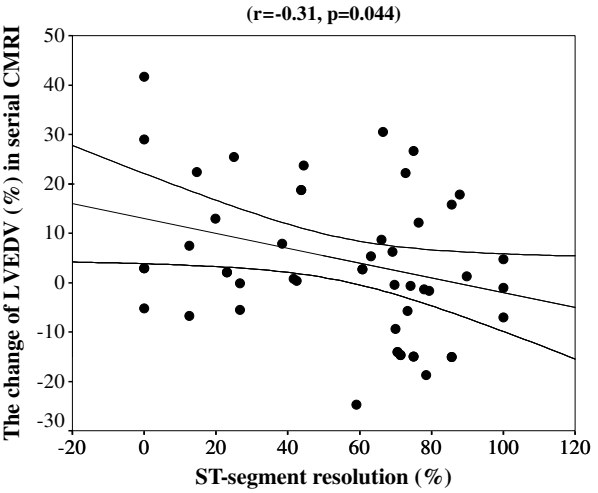

Fig 3. Correlation between the extent of ST-segment resolution and the relative left ventricular end-diastolic volume (LVEDV) change between the acute and chronic phases. CMRI, cardiac magnetic resonance imaging.

Discussion

In this study, early resolution of ST-segment elevation after primary PCI was shown to be a simple, clinically useful indicator for predicting infarct size and LV function. We found that patients with early STR ≥70% achieved greater myocardial salvage immediately after AMI in terms of less infarcted myocardial mass with less microvascular obstruction. In the late-phase, patients with early STR ≥70% showed less LV remodeling and LV dysfunction.

Early ST-Segment Resolution

Early resolution of ST-segment elevation is related to restoration of myocardial perfusion, less myocardial damage, and better prognosis after PCI for AMI[1,5,12] However, some patients with partial STR (<70%) had a TIMI III flow in the infarct-related artery[13] therefore, the angiographic findings may not reflect the status of microvascular perfusion. But, partial or absent ST-segment resolution indicates impaired microvascular perfusion, even in patients who have patent epicardial blood flow. Our study confirmed the finding that analysis of ST-segment changes on CMRI after primary PCI for STEMI could provide information about epicardial patency, as well as effective reperfusion at the tissue level.

CMRI Findings

Contrast-enhanced, high-resolution CMRI is a highly sensitive and reliable method of detecting the morphologic and functional sequelae of AMI[7,14] There have been various studies using CMRI for assessment of infarct size, LV function and LV remodeling in patients with AMI[7,15–17] but there has been little data on the correlation between serial CMRI parameters and the extent of early STR. The only existing clinical study addressing that issue was recently published by Thiele et al[9] However, their study was focused only on the CMRI findings in the late-phase of MI, whereas we investigated the morphologic and functional parameters of both early- and late-phase CMRI that correlate with the extent of STR after primary PCI.

LV remodeling after AMI is known to be a predictor of major adverse cardiac events and is related to various factors[6,18] Hypoenhancement within the hyperenhanced areas on delayed imaging is considered to be related to microvascular obstruction caused by endothelial damage, occluding thrombi and the influx of inflammatory cells[19] Microvascular dysfunction is known to be associated with greater myocardial damage and contributes to LV remodeling, together with infarct size.[3,15,16,20]

Baks et al reported that LVEF and LVESV in the chronic

Table 3 Multiple Linear Regression Analysis for Independent Clinical Determinants of Adverse LV Remodeling

	t	*Standardized coefficient*	*p value*
Adverse LV remodeling (R^2=0.427)			
Male	*–3.227*	*–0.533*	*0.003*
≥70% ST resolution	*–2.183*	*–0.372*	*0.036*
Age	*–1.647*	*–0.272*	*0.109*
Tertiles of peak CK-MB	*1.529*	*0.218*	*0.136*
≥2 MBG	*–1.350*	*–0.214*	*0.186*
Hypertension	*1.041*	*0.155*	*0.305*
Multivessel disease	*0.978*	*0.140*	*0.335*
Diabetes	*–0.714*	*–0.103*	*0.480*
LAD	*0.370*	*0.051*	*0.714*
Tertiles of pain-to-balloon time	*0.008*	*0.054*	*0.958*

MBG, myocardial blush grade; LAD, left anterior descending artery. Other abbreviations see in Tables 1,2.
Adverse LV remodeling was defined as increase in LVEDV between early-and late-phase CMRI.

stage were related to acute infarct size and the perfusion score based on the extent of hypoenhancement at baseline![16] Similarly, Hombach et al demonstrated that infarct size, PMO, and the extent of transmural infarction were predictive of adverse LV remodeling![21] Our study results showed that the extent of STR might reflect the infarct size and the presence of PMO, and was also correlated with LV remodeling and LVEF, as shown in previous studies![1,12]

Study Limitations

First, the sample size was relatively small, which limits statistical power. Second, the duration of early-phase CMRI was 8.2±8.0 days after primary PCI, which might affect the morphologic and functional parameters of CMRI. Third, the assessment of CMRI data had several technical problems. We excluded the most apical slice and the basal slice 10 mm below the aortic outflow tract to minimize the partial volume effect. Both the area and transmural extent of contrast-enhanced myocardium were assessed visually. Quantification of hypoenhanced areas was not performed because of the relatively low spatial resolution of CMRI. Minimal to mild microvascular obstruction also could have been missed because of the limited visualization. Despite these technical limitations, the present study demonstrated significant correlations between the CMRI parameters and the extent of STR that concur with findings of previous studies. Furthermore, in our opinion, this study is of important clinical significance, because it showed for the first time morphologic and functional correlates in serially performed CMRI for early STR.

Conclusion

The extent of STR is determined by the presence of microvascular obstruction and infarct size, and early complete resolution of ST-segment elevation predicts the improvement of LV dysfunction and prevention of adverse LV remodeling in STEMI. Also, early-phase CMRI data are comparable to late-phase CMRI in association with early STR. Our study confirms that early resolution of ST-segment elevation after primary PCI is a simple, clinically useful indicator of infarct size and LV function.

Acknowledgments

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References

1. de Lemos JA, Braunwald E. ST segment resolution as a tool for assessing the efficacy of reperfusion therapy. *J Am Coll Cardiol* 2001; **38:** 1283–1294.
2. Ito H, Okamura A, Iwakura K, Masuyama T, Hori M, Takiuchi S, et al. Myocardial perfusion patterns related to thrombolysis in myocardial infarction perfusion grades after coronary angioplasty in patients with acute anterior wall myocardial infarction. *Circulation* 1996; **93:** 1993–1999.
3. Bolognese L, Carrabba N, Parodi G, Santoro GM, Buonamici P, Cerisano G, et al. Impact of microvascular dysfunction on left ventricular remodeling and long-term clinical outcome after primary coronary angioplasty for acute myocardial infarction. *Circulation* 2004; **109:** 1121–1126.
4. Maes A, Van de Werf F, Nuyts J, Bormans G, Desmet W, Mortelmans L. Impaired myocardial tissue perfusion early after successful thrombolysis: Impact on myocardial flow, metabolism, and function at late follow-up. *Circulation* 1995; **92:** 2072–2078.
5. Dong J, Ndrepepa G, Schmitt C, Mehilli J, Schmieder S, Schwaiger M, et al. Early resolution of ST-segment elevation correlates with myocardial salvage assessed by Tc-99m sestamibi scintigraphy in patients with acute myocardial infarction after mechanical or thrombolytic reperfusion therapy. *Circulation* 2002; **105:** 2946–2949.
6. Kaneko H, Yaoita H, Iwai-Takano M, Watanabe N, Sakamoto K, Seino Y, et al. Prediction of remote left ventricular volumes and functions after acute myocardial infarction with successful coronary intervention. *Circ J* 2008; **72:** 226–231.
7. Kim RJ, Fieno DS, Parrish TB, Harris K, Chen EL, Simonetti O, et al. Relationship of MRI delayed contrast enhancement to irreversible injury, infarct age, and contractile function. *Circulation* 1999; **100:** 1992–2002.
8. Mahrholdt H, Wagner A, Holly TA, Elliott MD, Bonow RO, Kim RJ, et al. Reproducibility of chronic infarct size measurement by contrast-enhanced magnetic resonance imaging. *Circulation* 2002; **106:** 2322–2327.
9. Thiele H, Kappl MJ, Linke A, Erbs S, Boudriot E, Lembcke A, et al. Influence of time-to-treatment, TIMI-flow grades, and ST-segment resolution on infarct size and infarct transmurality as assessed by delayed enhancement magnetic resonance imaging. *Eur Heart J* 2007; **28:** 1433–1439.
10. van't Hof AW, Liem A, Suryapranata H, Hoorntje JC, de Boer MJ, Zijlstra F. Angiographic assessment of myocardial reperfusion in patients treated with primary angioplasty for acute myocardial infarction: Myocardial blush grade: Zwolle Myocardial Infarction Study Group. *Circulation* 1998; **97:** 2302–2306.
11. Baks T, van Geuns RJ, Biagini E, Wielopolski P, Mollet NR, Cademartiri F, et al. Effects of primary angioplasty for acute myocardial infarction on early and late infarct size and left ventricular wall characteristics. *J Am Coll Cardiol* 2006; **47:** 40–44.
12. Matetzky S, Freimark D, Chouraqui P, Novikov I, Agranat O, Rabinowitz B, et al. The distinction between coronary and myocardial reperfusion after thrombolytic therapy by clinical markers of reperfusion. *J Am Coll Cardiol* 1998; **32:** 1326–1330.
13. de Lemos JA, Antman EM, Giugliano RP, McCabe CH, Murphy SA, Van de Werf F, et al. ST-segment resolution and infarct-related artery patency and flow after thrombolytic therapy: Thrombolysis in Myocardial Infarction (TIMI) 14 investigators. *Am J Cardiol* 2000; **85:** 299–304.
14. Kim RJ, Wu E, Rafael A, Chen EL, Parker MA, Simonetti O, et al. The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. *N Engl J Med* 2000; **343:** 1445–1453.
15. Nagao M, Higashino H, Matsuoka H, Kawakami H, Mochizuki T, Murase K, et al. Clinical importance of microvascular obstruction on contrast-enhanced MRI in reperfused acute myocardial infarction. *Circ J* 2008; **72:** 200–204.
16. Baks T, van Geuns RJ, Biagini E, Wielopolski P, Mollet NR, Cademartiri F, et al. Recovery of left ventricular function after primary angioplasty for acute myocardial infarction. *Eur Heart J* 2005; **26:** 1070–1077.
17. Fieno DS, Kim RJ, Chen EL, Lomasney JW, Klocke FJ, Judd RM. Contrast-enhanced magnetic resonance imaging of myocardium at risk: Distinction between reversible and irreversible injury throughout infarct healing. *J Am Coll Cardiol* 2000; **36:** 1985–1991.
18. Tani S, Nagao K, Watanabe I, Kikushima K, Watanabe K, Anazawa T, et al. Increasingly well-preserved left ventricular function in hospital survivors with acute myocardial infarction: Effect of early and complete reperfusion strategy on left ventricular remodeling. *Circ J* 2007; **71:** 180–185.
19. Choi KM, Kim RJ, Gubernikoff G, Vargas JD, Parker M, Judd RM. Transmural extent of acute myocardial infarction predicts long-term improvement in contractile function. *Circulation* 2001; **104:** 1101–1107.
20. Ichikawa Y, Sakuma H, Suzawa N, Kitagawa K, Makino K, Hirano T, et al. Late gadolinium-enhanced magnetic resonance imaging in acute and chronic myocardial infarction: Improved prediction of regional myocardial contraction in the chronic state by measuring thickness of nonenhanced myocardium. *J Am Coll Cardiol* 2005; **45:** 901–909.
21. Hombach V, Grebe O, Merkle N, Waldenmaier S, Hoher M, Kochs M, et al. Sequelae of acute myocardial infarction regarding cardiac structure and function and their prognostic significance as assessed by magnetic resonance imaging. *Eur Heart J* 2005; **26:** 549–557.