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Determinants and impact of microvascular obstruction in successfully reperfused ST-segment elevation myocardial infarction. Assessment by magnetic resonance imaging

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Abstract Microvascular obstruction (MVO) is an important and independent determinant of post-infarct remodeling. Fifty-two patients with a successfully reperfused ST-segment elevation acute myocardial infarction (MI) were studied with MRI in the first week and at 4 months post-infarction. On early (i.e., 2–5 min) post-contrast MRI, MVO was detected in 32 patients with an MVO to infarct ratio of 36.3±24.9%. On late (i.e., 10–25 min) post-contrast MRI, MVO was detected in only 27 patients, with an MVO to infarct ratio of 15.9±13.9%. MVO infarcts (n=32) were associated with higher cardiac enzymes (troponin I, P=0.016), and lower pre-revascularization thrombolysis in myocardial infarction (TIMI) flow (P=0.018) than non-MVO infarcts (n=20). Infarct size was larger in MVO infarcts (25.0±14.3 g) than non-MVO infarcts (12.5±7.9 g), P=0.0007. Systolic wall thickening in the infarct and peri-infarct area, and left ventricular (LV) ejection fraction (EF) were worse in MVO (46.1±7.2%) than non-MVO infarcts (50.5±6.6%, P=0.038). At 4 months, MVO infarcts showed more adverse remodeling and lack of functional improvement, whereas non-MVO infarcts improved significantly (LV EF at 4 months, MVO, 47.5±7.8%, P=0.31; non-MVO, 55.2±10.3%, P=0.0028). In the majority of patients with successfully reperfused ST-segment elevation MI, MVO is observed, whose present and maximal extent can be best evaluated on early post-contrast MRI. Presence of MVO is associated with more extensive infarctions, and characterized by greater adverse LV remodeling and lack of functional recovery.

Keywords Heart · Magnetic resonance imaging · Myocardial infarction · Coronary artery disease

Introduction

The extent of myocardial necrosis in acute myocardial infarction (MI) is not the sole determinant of post-infarct left ventricular (LV) remodeling. Other important determinants are the patency of infarct-related artery, infarct location, and the concomitant presence of microvascular obstruction (MVO) in the necrotic myocardium [1–5]. It was found in the early 1990s that a significant number of acute infarct patients, showed a lack of reperfusion at tissue level despite the presence of a normal thrombolysis in myocardial infarction (TIMI) flow after revascularisation of the infarct-related artery [4]. Since then, several studies have shown that presence of MVO or so-called no- or slow-reflow phenomenon is associated with a worse outcome with greater adverse LV remodeling, and a higher mortality than non-MVO infarcts [4–13]. Already after 90 min of occlusion, important damage associated with interstitial and myocardial cellular edema occurs in the ischemic myocardium. More prolonged periods of coronary occlusion result in severe inflammation and disruption of the capillary network [14, 15]. In addition to microcirculatory damage caused by ischemia-reperfusion injury, stent placement may result in distal embolization, spasm, and

release of vasoactive mediators. When the pathophysiologic response is severe, MVO occurs defined as adequate restoration of epicardial flow but poor or no distal tissue-level perfusion [15, 16].

Although initially studied by means of contrast echocardiography [4], it has been shown that the new contrast-enhanced magnetic resonance imaging (MRI) techniques using an additional inversion pulse to create a higher contrast between normal and infarcted myocardium, are also well suited to demonstrate the presence of MVO [9, 11, 13, 17, 18]. In order to better understand the impact of MVO on post-infarct remodeling and LV recovery, we evaluated in this study the underlying determinants of MVO using serial MRI in patients in the early phase (i.e., within 1 week and at 4 months after a successful revascularization of an ST-elevation acute MI.

Materials and methods

Patients were included if they presented with acute MI with cumulative ST-segment elevation ≥6 mm and had undergone successful revascularization by percutaneous coronary intervention (PCI) of the infarct-related artery, defined as TIMI-epicardial flow ≥2 at the end of the procedure. Only patients presenting within 2–12 h of symptom onset were included in the study. Patients referred from secondary centers were allowed to receive thrombolysis prior to transfer to the University Hospital. Patients with prior coronary artery bypass grafting, pulmonary edema, cardiogenic shock or significant co-morbidities were excluded from the study. The ethics review board of the University Hospital of the University of Leuven, Belgium, approved the protocol, and the study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from each patient.

Protocol for MRI

All MRI studies were performed on a 1.5-T system (Intera, Philips Medical Systems, Best, Netherlands) using commercially available cardiac MRI software, electrocardiographic triggering and cardiac-dedicated surface coils. All patients were positioned in supine position. Studies were performed in the 1 week and 4 months follow-ups. First, after determination of the cardiac axes with localizers, the myocardium at risk was defined using a T2-weighted short-tau inversion-recovery (STIR) fast spin-echo (FSE) MRI in the cardiac short-axis. Next, global and regional LV function were assessed using breath-hold, steady-state free-precession (SSFP) cine MRI in the cardiac short-axis, vertical- and horizontal long-axis. In the cardiac short-axis direction, the left ventricle was completely encompassed by contiguous 8-mm-thick slices. The myocardial perfusion at rest was evaluated using a first-pass imaging technique with four to five short-axis levels (saturation-recovery spoiled-gradient-echo technique) and a contrast-dose of 0.05 mmol/kg gadopentetate dimeglumine. Immediately following the first-pass perfusion sequence, an additional dose of 0.15 mmol/kg of gadopentetate dimeglumine was administered. Imaging was continued to evaluate the presence of myocardial necrosis and concomitant MVO by means a breath-hold T1-weighted three-dimensional inversion-recovery gradient-echo technique. The inversion time was individually adapted to suppress signal of normal myocardial tissue. The presence of MVO was evaluated on both the early (i.e., 2–5 min) and late (i.e., 10–25 min) images following contrast-injection, while myocardial necrosis was quantified on the late images, i.e., 10–25 min following contrast. MVO was defined as a dark zone in the area at risk, usually located in the subendocardium and showing a variable degree of transmurality. An MVO infarct was defined on the early post-contrast images. In practice the MVO location always corresponded with the center of the infarct location. The infarct area was defined as the zone of bright signal on the late enhanced images, in contrast with the dark-gray signal of the normal myocardium.

Image analysis

All MRI studies were analyzed on an off-line workstation (Philips ViewForum). The myocardial area at risk on T2-weighted-STIR FSE MRI, visible as an area of increased myocardial signal intensity in the perfusion territory of the infarct-related coronary artery, was manually traced and the volume quantified. For evaluation of global and regional function and calculation of LV mass, the endocardial and epicardial borders were manually traced in the end-diastolic and end-systolic short-axis slices. Papillary muscles were not included in the myocardium. LV end-diastolic volume (EDV), LV end-systolic volume (ESV) and LV mass were determined and indexed to body surface area (BSA). Volumes at end systole were corrected for longitudinal ventricular shortening. The first-pass perfusion MR images were visually assessed, and scored as normal or as subendocardial or transmural perfusion defect in one of the coronary artery perfusion territories. Areas of MVO and myocardial necrosis (including eventual residual MVO) were manually traced.

Regional morphological and functional analysis was performed using a three-compartment model. The infarct area was defined by the presence, location and extent of myocardial enhancement on the delayed enhancement images. Next, a *peri-infarct* area was defined as a rim of myocardium completely surrounding the infarct area. This was achieved by using in the short-axis direction a 30° sector of the LV cirucmference adjacent to both sides of the infarct area, while in longitudinal direction one slice of nonenhanced myocardium in immediate contact with the

enhanced myocardium was considered as peri-infarct area. The remaining of the myocardium was defined as *remote myocardium* (Fig. 1). Regional morphological analysis included measurement of wall thickness at end diastole and end systole, allowing to calculate regional function, i.e., systolic wall thickening, in the three areas. The infarct volume was obtained as the sum of areas of myocardial enhancement on the cardiac short-axis images, and a similar approach was used to quantify the MVO volume. The mean infarct transmurality was obtained by tracing the area of enhancement across the myocardial wall, and dividing it by the total area of myocardium encompassed by the enhanced myocardium. A similar approach was used to define the mean transmurality of MVO. Dividing the volume of MVO by the infarct volume yielded the MVO/infarct volume ratio. All images were analyzed by consensus of two experienced observers (J.B., M.K.).

Statistical analysis

All continuous data were reported as mean±SD. Paired Student's *t*-tests were applied to evaluate differences in EF, EDV, ESV, mass, infarct size and transmurality, MVO size and transmurality between the first week and 4 months post-infarction. Unpaired Student's *t*-tests were used to evaluated the above parameters for differences between the MVO and non-MVO group. An ANOVA test with the post-hoc Scheffé test was used to evaluate the morphological and functional differences in the infarct, peri-infarct, remote myocardium between the 1 week and 4 months study, and between MVO and non-MVO infarctions. All tests are two-tailed, and statistical significance was accepted at $P<0.05$. Except when stated otherwise, the MVO data and results are those obtained from the early enhanced images.

Results

Patient data

Between May 2003 and December 2004, 52 patients with an acute ST-elevation MI, agreed to participate in the study (see Table 1). All patients had a successful revascularisation (primary PCI: 45, intravenous thrombolysis: seven). All patients successfully underwent both MRI examinations at the first week (mean 4±1 days) and 4 months (mean 121±6 days). At coronary angiography, the culprit lesion was located in the left anterior descending coronary artery (27 patients), right coronary artery [21] and left circumflex coronary [4]. Forty-nine of the 52 culprit lesions were located in the proximal or mid portions of the coronary arteries. Three patients had a history of a previous MI. There was no significant difference in time from symptom onset to revascularization between MVO and non-MVO patients. However, MVO patients showed significantly higher maximal levels of troponin I ($P=0.016$). Moreover, the pre-revascularization TIMI flow was significantly lower in the MVO-infarct group (0.6±1.1 versus 1.4±1.3, $P=0.018$). No differences were found in post-revascularization TIMI flow. No cardiac deaths or major adverse events were noticed during the 4-months follow-up period.

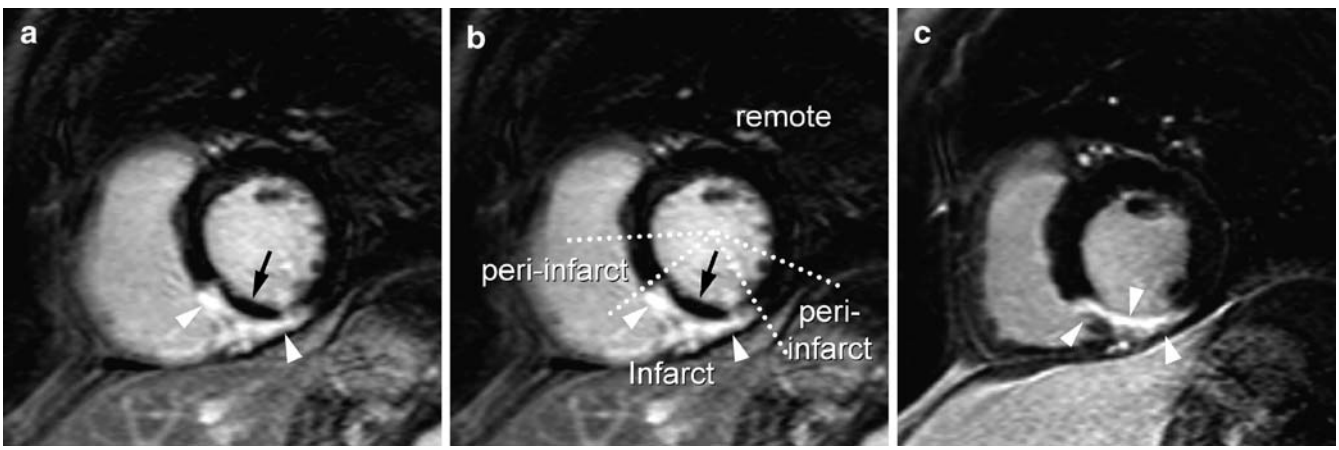

Fig. 1 **a** Example of three-compartment analysis to assess regional morphology and function in a patient with an extensive, transmural infarct in the LV inferior wall (*arrows*) with a concomitant important zone of MVO as shown on this contrast-enhanced inversion-recovery MR image (*arrowhead*). **b** In the short-axis images, the enhanced area is defined as *infarct* area, a surrounding rim of myocardium (30° angle) on both sides as *peri-infarct* area, while the remaining myocardium is defined as *remote* myocardium. **c** Same short-axis slice at 4 months follow-up. Note the thinning of the infarcted area (*arrowheads*)

Table 1 Patient characteristics

	MVO infarcts (n=32)	Non-MVO infarcts (n=20)	P value
Patient age (years)	55.7±1.7	54.9±10.7	
Risk factors and history			
Male/female	27/5	19/1	
Current smoker	16	12	
Diabetes mellitus	5	3	
Hypertension	11	7	
Body mass index (kg/m^2)	26.1±3.1	27.3±4.5	
Previous myocardial infarction	3	0	
Primary treatment			
PCI	28	17	
Thrombolysis	4	3	
Infarct-related artery			
LAD coronary artery			
Proximal	9	4	
Mid	5	9	
RCA coronary artery			
Proximal	8	4	
Mid	6	1	
Distal	1	1	
LCx coronary artery			
Proximal	2	1	
Distal	1	0	
Time to revascularization (min)	257±128	298±180	0.38
TIMI-flow pre-revascularization	0.6±1.1	1.4±1.3	0.018
TIMI-flow post-revascularization	2.8±0.4	2.9±0.3	0.62
Troponin I in hospital (μg/l)	127±124	55±54	0.016

Cardiac morphology and function at 1 week post-infarction

Data on global and regional LV morphology and function are shown in Tables 2, 3 and 4 (see also Figs. 1, 2, 3). In all patients, myocardial edema was found in the perfusion territory distal to the culprit lesion at coronary angiography. Thirty-two of them showed MVO at early contrast-enhanced MRI, which was always located in the area of myocardial edema. The ratio of MVO to non-MVO infarct was 14/13 for LAD, 15/6 for RCA, and 3/1 for LCx coronary artery. Although no differences were found in extent of area at risk, the ratio of infarct to area at risk was significantly larger in MVO infarcts than in non-MVO infarcts (P<0.0001) (Table 2). Mean MVO volume was 7.5±6.5 g (range 1–24), corresponding to 36.3±24.9% of the infarct volume (range 8.3–78.6). The MVO was subendocardially located, with a maximal transmural spread of 51±18% (range 25–91%). On late-enhancement MRI, MVO was detected in 27 patients with significantly lower MVO volumes (4.1±4.8 g, P=0.03), lower MVO/infarct volume ratios (15.9±13.9%, P=0.0003), and lower MVO transmurality (32.4±17.8%, P=0.0002) than on the early-enhanced images. Comparison of the first-pass perfusion MRI and early post-contrast MRI studies yielded a match in 48/52 patients about the presence or absence of a MVO/myocardial perfusion defect. In the remaining four patients, a subendocardial perfusion defect was detected without evidence of MVO on the early post-contrast MR images.

Infarct volumes and degree of transmurality were significantly larger in MVO than in non-MVO infarcts. As shown in Table 5, the presence of MVO and the degree of MVO transmurality were related to infarct size. While at 1 week, no differences were shown in global LV volumes, LV EF was significantly lower in MVO infarcts (46.1±7.2%) than in non-MVO infarcts (50.4±6.5), P=0.038 (Table 2). Regional analysis showed no differences between groups in end-diastolic wall thickness (Table 3). However, systolic wall thickening was significantly lower

Table 2 Data on global LV morphology and function, and data on infarct characteristics

	MVO infarcts (n=32)	Non-MVO infarcts (n=20)	P value
LV EDV (ml)			
1 week	158.4±34.2	155.9±24,6	0.78
4 months	169.9±43.2	148.0±32.2	0.06
Diff 1 week–4 months	+ 11.5±29.6*[1]	−7.9±27.3	0.02
LV EDV/BSA (ml/m^2)			
1 week	83.0±15.2	79.4±10.1	0.35
4 months	89.1±21.7	75.2±13.3	0.013
Diff 1 week–4 months	+ 6.1±15.3*[1]	−4.2±13.9	0.018
LV ESV (mL)			
1 week	86.0±23.8	77.6±17.4	0.18
4 months	90.9±31.4	66.7±24.6	0.0052
Diff 1 week–4 months	+4.9±21.8	−10.8±19.9*[1]	0.012
LV ESV/BSA (ml/m^2)			
1 week	45.0±11.0	39.5±7.8	0.06
4 months	47.6±16.3	34.0±12.6	0.0025
Diff 1 week–4 months	+2.6±11.5	−5.5±10.3*[1]	0.013
LV EF (%)			
1 week	46.1±7.2	50.4±6.5	0.038
4 months	47.5±7.8	55.2±10.3	0.0036
DD	+ 1.3±7.4	+ 4.8±6.3*[2]	0.09
LV mass (g)			
1 week	118.1±30.0	111.7±21.7	0.41
4 months	105.6±22.0	105.0±27.0	0.93
Diff 1 week–4 months	−13.0±18.0*[3]	−5.9±13.4	0.13
LV mass/BSA (g/m^2)			
1 week	61.6±13.1	56.8±9.2	0.15
4 months	54.8±12.3	53.6±8.4	0.68
Diff 1 week–4 months	−6.8±9.3*[3]	−3.2±6.7*[1]	0.14
Area at risk (g)	38.9±19.1	34.0±18.8	0.37
Infarct volume (g)			
1 week	25.0±14.3	12.5±7.9	0.0007
4 months	15.2±7.6	7.9±6.5	0.0009
Diff 1 week–4 months (%)	−36.1±17.7*[4]	−41.5±21.4*[4]	0.33
Infarct volume/LV mass (%)			
1 week	20.7±10.8	11.3±7.5	0.0012
4 months	14.4±7.7	7.5±6.0	0.0014
Diff 1 week–4 months	−28.3±18.4*[4]	−37.8±20.8*[4]	0.09
Infarct volume/area at risk (%)	65.6±18.6	40.1±22.0	<0.0001
Infarct transmurality (%)			
1 week	83.3±16.0	74.7±16.1	0.04
4 months	71.3±16.3	67.6±12.6	0.40
Diff 1 w–4 months	−12.0±8.1*[4]	−7.1±8.2*[3]	0.04

*[1]P<0.05 for the difference between 1 week and 4 months, *[2]P<0.01 for the difference between 1 week and 4 months, *[3]P<0.001 for the difference between 1 week and 4 months, *[4]P<0.0001 for the difference between 1 week and 4 months

Table 3 Regional end-diastolic myocardial wall thickness

	MVO infarcts (n=32)	Non-MVO infarcts (n=20)	P value
Infarct area			
1 week	7.9±1.9	7.5±1.4	0.41
4 months	6.3±1.4*	6.7±1.6**	0.33
Peri-infarct area			
1 week	7.7±1.5	7.4±1.3	0.43
4 months	7.1±1.2**	6.9±1.3	0.64
Remote myocardium			
1 week	7.1±1.6	7.0±1.1	0.96
4 months	6.8±1.3	7.0±1.2	0.68

All values are expressed in mm
*P<0.0001 for the difference between 1 week and 4 months,
**P<0.05 for the difference between 1 week and 4 months

in the MVO group in the infarct (17.4±14.9% versus 33.6±20.2%, P=0.0018) and peri-infarct area (32.1±13.2% versus 43.3±20.1%, P=0.02) (Table 4).

Cardiac morphology and function at 4 months follow-up

Although MVO and non-MVO infarcts showed a similar reduction in infarct size, due to the larger infarct size of MVO infarcts, the impact on LV mass reduction was greater in MVO infarcts than in non-MVO infarcts (Table 2; see also Figs. 1, 3). A greater extent of wall thinning in the infarct area, as well as a greater reduction in infarct transmurality were found in MVO infarcts compared with non-MVO infarcts (Table 3). Moreover, a significant wall thinning was found in the peri-infarct area too in the MVO infarct group. Non-MVO infarcts showed a significant improvement in LV ejection fraction (EF) (50.4±6.5% to 55.2±10.3%, P=0.0028), which was related to a significant decrease in LV ESV (Table 2). In contrast, MVO infarcts did not show a functional improvement but a significant increase in LV EDV. Subgroup analysis of the four patients with first-pass perfusion defect but without MVO on early post-contrast imaging showed an LV EF of 51.1% (range 44.6–54.2) at 1 week, and 57.0% (range 44.4–63.4). Mean infarct size at 1 week was 7.6 g (range 1–15.9).

Table 4 Regional systolic wall thickening

	MVO infarcts (n=32)	Non-MVO infarcts (n=20)	P value
Infarct area			
1 week	17.4±14.9	33.6±20.2	0.0018
4 months	19.7±17.5	40.8±20.9	0.0003
Peri-infarct area			
1 week	32.1±13.2	43.3±20.1	0.02
4 months	34.1±15.0	53.7±21.9	0.0004
Remote myocardium			
1 week	58.2±22.2	53.6±18.2	0.44
4 months	53.7±19.6	57.3±25.3	0.57

All values are shown as percentages

Discussion

Microvascular reperfusion damage is found in the majority of patients with a ST-segment elevation MI, despite a successful reperfusion of the epicardial coronary artery. Both the presence and severity of MVO are clearly related to more extensive MIs (based on enzyme levels and infarct size determined on contrast-enhanced MRI) presenting a more severely impaired pre-revascularization TIMI flow. Presence of MVO is characterized by lack of functional recovery and adverse remodeling at 4 months follow-up.

Depiction of MVO on contrast-enhanced MRI is a dynamic phenomenon with significant changes in size and transmurality over time after contrast administration. Both the absolute MVO size and the ratio of MVO to infarct size, as well as the MVO transmurality, show a dramatic reduction (i.e., in the order of 40–50%) between early and late imaging. Five patients with MVO would have been missed if only late enhancement images were obtained. These findings suggest that the full extent of MVO can be best appreciated very soon after contrast administration. Diffusion of contrast agent into the MVO is the most likely explanation for the shrinkage of the MVO area between early and late post-contrast imaging. This phenomenon usually occurs simultaneously on both the endo- and epicardial sides. Because timing is critical to visualize MVO, this may explain some of the differences on the reported MVO incidence, as well as of MVO size literature [7, 11, 13, 19–22]. At present, it is unknown whether the speed of fill-in of the MVO area is prognostically important.

Because other groups have used first-pass perfusion MRI to evaluate impaired microvascular reperfusion after angioplasty for acute MI, we compared early-enhanced contrast MRI with first-pass perfusion MRI [9, 23]. In most patients, we found a close match between both techniques concerning the presence or absence of a MVO/myocardial perfusion defect. However, first-pass perfusion MRI depicted in four patients small, subendocardial perfusion defects without evidence of MVO on early-enhanced MRI. Whether this concerns true MVO areas with limited size and very rapid fill-in, or slow-flow or fruste forms of no-reflow defects is not clear. Although the current study was not powered to perform a subgroup analysis, the behavior in terms of infarct size, impact on/and recovery of LV function at follow-up was most similar to non-MVO

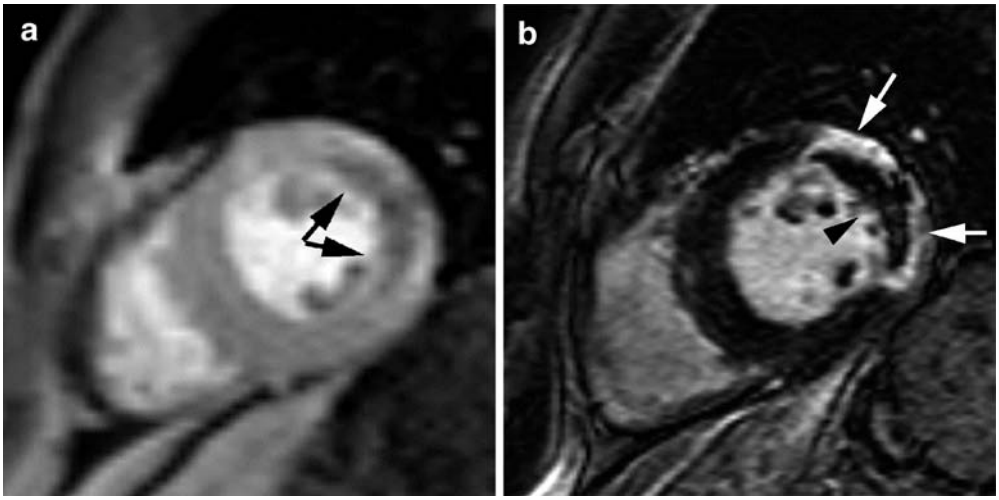

Fig. 2 Large transmural infarct in the lateral LV wall (*white arrows*) with extensive no-reflowzone. Comparison between **a** first-pass myocardial perfusion MRI and **b** post-contrast MRI obtained 15 min post-contrast. Note the close match in location and extent between the perfusion defect on the first pass perfusion images (*black arrowheads*) and hypointense area on the post-contrast MR images (*black arrowhead*)

infarcts. Moreover, because of the better spatial resolution and the full ventricular coverage, early contrast-enhanced MRI has the advantage over first-pass perfusion MRI to enable easier quantification and better appreciation of the exact degree of MVO transmurality and MVO extent.

A second important finding is the close relationship between MVO and infarct extent (i.e., infarct size and infarct transmurality). In infarcts smaller than 10% of LV mass, there is a 2/1 predominance for non-MVO infarcts, while for infarcts larger than 20% of LV mass there is a 4/1 predominance for MVO infarcts. Also the MVO severity, expressed in terms of MVO transmurality, increases with increasing infarct volume. These findings are matched with significantly higher enzyme levels and lower pre-revascularization TIMI flow in MVO-infarcts. Remarkably, there was no significant difference in time from symptom onset to revascularization between non-MVO and MVO infarcts, indicating that ischemia duration is not the sole determinant of infarct and MVO size. Due to their larger size, MVO infarcts have a more severe impact on global function and regional contractility not only in the infarct area but also in the peri-infarct area. Although the latter could be explained by a larger amount of stunned myocardium in MVO infarcts, the lack of functional recovery at 4 months does not support this hypothesis.

A third important finding is the morphological and functional divergence at 4 months follow-up between MVO and non-MVO infarcts. Because of their larger size MVO infarcts have a larger impact on LV mass reduction. Wall thinning is more extensive in MVO infarcts, and is not limited to the infarct area but moreover involves the peri-infarct area too. A potential explanation could be an increased wall stretch at the infarct borders. In accordance to the findings of Choi et al. [13] we found a larger reduction in infarct transmurality at 4 months in MVO infarcts. At LV level, MVO infarcts are characterized by

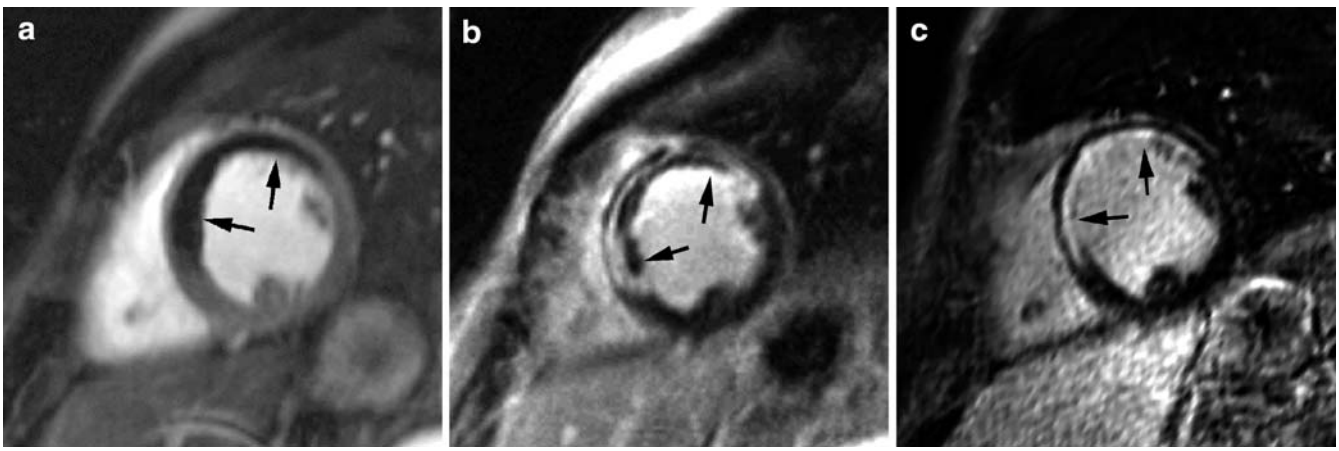

Fig. 3 Fill-in of the MVO area between the early- and late post-contrast MRI in a patient with an extensive infarction of the anteroseptal LV wall. **a** Early post-contrast MRI shows extensive dark area (*arrows*). **b** Late post-contrast MRI shows partial fill-in of the MVO area with a remaining, mainly subendocardially located hypo-intense area (*arrows*). **c** Follow-up study at 4 months shows important thinning of the infarcted area (*arrows*). Notice the presence of the remaining rim of viable subepicardial tissue

Table 5 Relationship between infarct size, presence or absence of MVO, and the degree of MVO transmurality

Infarct size[a] (%)	0–10	11–20	21–30	>31
MVO Present	6	14	8	4
Transmurality:				
1–25%	3	0	0	0
26–50%	2	8	3	1
51–75%	1	6	2	2
76–100%	0	0	3	1
MVO absent	12	6	3	0

[a]Infarct size normalized to LV mass

adverse remodeling with increased end-diastolic volumes and lack of functional recovery. In contrast, non-MVO infarcts show global functional recovery, which is mainly obtained by a reduction in end-systolic volumes.

In conclusion, presence of MVO is not only a frequent finding in successfully reperfused ST-segment elevation MI, but has important prognostic significance. Early post-contrast imaging is strongly recommended to best appreciate the presence and full extent of MVO. Because of its importance in predicting functional recovery and LV remodeling, treatment strategies should be focused to reduce microvascular damage (e.g., adenosine, nitric oxide, etc.) [24, 25]. Contrast-enhanced MRI is likely the best technique to evaluate the efficacy of novel treatment regimens.

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