Signature: © Pol J Radiol, 2015; 80: 536-543
DOI: 10.12659/PJR.895396



Polish Journal of Radiology
www.PolRadiol.com
ORIGINAL ARTICLE

Received: 2015.07.20
Accepted: 2015.07.27
Published: 2015.12.13

Microvascular Obstruction Evaluation Using Cardiovascular Magnetic Resonance (CMR) in ST-Elevated Myocardial Infarction (STEMI) Patients

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

Dorota Piotrowska-Kownacka[1ABCDEF], Łukasz Kownacki[2ABDE], Janusz Kochman[3BD], Agnieszka Kołodzińska[3BD], Małgorzata Kobylecka[4EF], Leszek Królicki[4AEF]

[1] 1st Department of Radiology, Medical University of Warsaw, Warsaw, Poland
[2] Department of Radiology, European Health Center, Otwock, Poland
[3] Department of Cardiology, Medical University of Warsaw, Warsaw, Poland
[4] Department of Nuclear Medicine, Medical University of Warsaw, Warsaw, Poland

Author's address: Dorota Piotrowska-Kownacka, 1st Department of Radiology, Medical University of Warsaw, Warsaw, Poland, e-mail: dorota.piotrowska@mrlab.pl

Source of support: The study was supported by grant N402 043 32/1326

Summary

Backround: Restoration of blood flow in epicardial coronary artery in patients with acute myocardial infarction can, but does not have to restore efficient blood flow in coronary circulation.

The aim of the study was a direct comparison of microvascular obstruction (MVO) detected by rest and stress perfusion imaging and gadolinium enhancement obtained 2 min. (early MVO) and 15 min. (delayed MVO) post contrast.

Material/Methods: 106 patients with first anterior myocardial infarction were studied. Cardiovascular magnetic resonance (CMR) was performed 5±2 days after primary percutaneous coronary intervention (pPCI). Stress and rest perfusion imaging was performed as well as early and delayed gadolinium enhancement and systolic function assessment.

Scoring of segmental function, perfusion defect, MVO and scar transmurality was performed in 16 segment left ventricular model.

Results: The prevalence of MVO varies significantly between imaging techniques ranging from 48.8% for delayed MVO to 94% with stress perfusion. Median sum of scores was significantly different for each technique: stress perfusion 13 (7; 18), rest perfusion 3 (0.5; 6), early MVO 3 (0; 8), delayed MVO 0 (0; 4); $p<0.05$.

Infarct size, stress and rest perfusion defects were independent predictors of LV EF at discharge from hospital.

Conclusions: Imaging protocol has a significant impact on MVO results. The study is the first to describe a stress-induced MVO in STEMI patients. Further research is needed to evaluate its impact on a long term prognosis.

MeSH Keywords: Adenosine • Anterior Wall Myocardial Infarction • Magnetic Resonance Imaging • No-Reflow Phenomenon

PDF file: http://www.polradiol.com/abstract/index/idArt/895396

Background

The goal of primary percutaneous intervention (pPCI) in patients with ST segment elevation myocardial infarction (STEMI) is the restoration of blood flow in an infarct-related coronary artery. This approach leads to a reduction of infarct size and to lower mortality [1].

However, reopening of an artery does not ensure successful perfusion at the tissue level. This phenomenon is related to absent or impaired perfusion at the tissue level. Such a condition of coronary microcirculation is called "no reflow" and is caused by microvascular obstruction (MVO) due to a very complex mechanism. The causes of "no reflow" include swelling of the endothelial cells and cardiomyocytes, microembolisation with thrombotic or atherosclerotic debris as well as infiltration of neutrophiles and platelets and a hemorrhage [2–4].

It is established that presence of microvascular obstruction is related to a poor short-term and long-term prognosis [5]. Microvascular obstruction is a dynamic condition that increases in size within the first 48 hours after reopening of the infarct-related artery and remains stable between 48h and 10 days [6,7]. Therefore, absence of no-reflow immediately after pPCI does not exclude the presence of no-reflow 2–10 days later.

It is well known that cardiovascular magnetic resonance (CMR) is a good diagnostic tool for determining microvascular obstruction [8,9].There are at least two CMR methods suitable for MVO assessment. The first method uses "first-pass" perfusion imaging and is based on observation of slower wash-in of gadolinium-based contrast agent [10]. The second approach is based on gadolinium enhancement imaging obtained early (1–2 min.) [11,12] or later (more than 10 min.) after contrast infusion [12–14].

It is important to notice that the mentioned acquisition time reflects only the start point of the imaging sequence. Depending on the patient's condition and imaging parameters the scanning can last even 10 minutes and this can decrease the true MVO size [11,12].

Persistent or delayed MVO (dMVO) generally presents as a dark area surrounded by enhanced myocardium on delayed gadolinium-enhanced CMR images obtained at least 10 minutes after contrast administration.

On the other hand, "early" MVO can be seen as enhancement deficit on first pass perfusion images at rest [10] or as dark spots surrounded by enhanced myocardium on "early" gadolinium-enhanced images obtained 1–2 min. after contrast administration. Both methods are often used interchangeably.

The CMR imaging of first-pass perfusion under stress conditions early after STEMI has been published more recently in limited literature [15–18]. The data based on a limited number of patients and only few articles described patients treated with primary PCI [16,18]. Until now no literature has been available comparing all 4 methods of MVO imaging by CMR.

Therefore, we aimed to assess the prevalence and size of MVO imaged by 4 CMR methods: first-pass stress perfusion for stress MVO (sMVO), first-pass rest perfusion for rest MVO (rMVO), early gadolinium enhancement for early MVO (eMVO) imaging and delayed gadolinium enhancement for delayed MVO (dMVO) imaging.

We sought the differences between MVO imaged by the 4 methods and their correspondence to the left ventricular function and infarct size at the time of patient's discharge from hospital.

Material and Methods

A total of 106 patients were enrolled, with the first STEMI treated successfully with pPCI within 12 hours from the onset of chest pain. Successful pPCI was defined as a restoration of TIMI 3 flow and residual stenosis less than 10% in an infarct-related coronary artery.

The study was approved by the Local Ethics Committee. Patients with a contraindication to CMR and adenosine infusion (implantable electronic devices, claustrophobia, asthma, atrio-ventricular block of the 2nd and 3rd degree), renal failure and a history of previous MI were excluded from the study. All patients received standard treatment with aspirin, clopidogrel, beta-blockers, ACE inhibitors and statins. The application of IIb/IIIa inhibitors or aspiration trombectomy depended on the operator's choice during the procedure.

CMR protocol

All patients underwent CMR examination on a commercially available 1.5 T scanner (Siemens Avanto, Erlangen, Germany) one or two days before their discharge from hospital. CMR protocol included assessment of the myocardial function, first-pass perfusion in rest and stress conditions, early MVO imaging, persistent MVO imaging and viability assessment with delayed gadolinium enhancement method.

Left ventricular function was assessed by ECG-gated steady state free precession pulse sequence (SSFP) in parallel continuous short axis slices covering the whole left ventricle.

Typical imaging parameters were as follows: echo time (TE) 1.21 ms, slice thickness 8 mm, gap 0 mm, matrix 256×220, FOV 340–380 mm, 25–30 phases, temporary resolution below 50 ms. The imaging was performed during breath holds in expiration. Additionally, at least one horizontal and vertical long axis images were obtained for apical segment evaluation.

First-pass perfusion in rest and in pharmacological stress conditions were assessed with IR-prepared gradient echo pulse sequence. Thee short axis slices were imaged, placed at basal, middle and apical segments of the left ventricle.

Sequence parameters were as follows: echo time slice thickness 8 mm, gap 12-16 mm, matrix 256×126, FOV 340–380 mm, 50 dynamics. Parallel imaging (GRAPPA) factor up to 3 was used for high temporal resolution.

Adenosine (Adenocor, Sanofi Aventis) was infused intravenously at a dose of 140 ug/kg b.w./min. over 4.5 min. Gadopentate dimeglumine (Magnevist, Bayer Schering, Germany), 0.1 mmol/kg b.w.) was given intravenously after 4 minutes of adenosine infusion, with injection speed of 4 mL/s followed by 30 mL of 0.9% NaCl.

During the entire CMR study parameters of ECG, O_2 saturation and pulse were monitored with MRI-compatible cardiac monitor (MagLife Plus, Schiller, France). Additionally, shortly before and during adenosine infusion, blood pressure was measured continuously, and then repeated every 10 minutes until the end of the examination.

All necessary medications such as aminophiline, atropine, nitroglicerine, adrenaline as well as defibrillator were available during the entire study as a protection against possible side effects caused by adenosine infusion or late complications of STEMI.

Within 2 minutes after stress perfusion assessment early microvascular obstruction (eMVO) was imaged in a short-axis orientation by IR-prepared single-shot gradient echo sequence with fixed inversion time of 300 ms, on expiration during one or two breath holds. Sequence parameters were as follows: slice thickness 8 mm, gap 0, matrix 192×86, FOV 340–380 mm. The single-shot imaging sequence was chosen to avoid the delay between the first and the last slice scanning.

Viability and delayed microvascular obstruction (dMVO) were assessed on delayed gadolinium-enhanced images obtained with IR-prepared gradient echo sequence with high resolution in short axis 15–20 min. after contrast administration. Sequence parameters were as follows: slice thickness 8 mm, gap 0, matrix 256×220, inversion time (TI) was adapted to null the signal from normal myocardium: usually in a range of 350–400 ms, FOV 340–380 mm.

CMR analysis

Post-processing was performed using QMASS (Medis, Netherlands) software by a consensus of 2 experienced CMR readers.

For left ventricular (LV) function analysis end-diastolic and end-systolic phases were chosen. The segmentation of epicardial and endocardial borders was done automatically and then manually corrected.

For each patient, left ventricular end diastolic volume (EDV), end systolic volume (ESV), mean left ventricle mass (LV mass) and ejection fraction (EF%) were calculated.

Additionally, a detailed segmental function was assessed in 17 segments using a 5-point scale: 0 – normokinetic, 1 – hypokinetic, 2 – severe hypokinetic, 3 – akinetic, 4 – dyskinetic, in each segment.

Stress and rest MVO were defined as lower signal area compared to remote myocardium that persisted for at least 5 dynamic images. Visual analysis of stress and rest perfusion images was performed in 16 segments using the 5-point scale system corresponding to perfusion defect transmurality: 0 – without perfusion deficit, 1 – 1–25%, 2 – 26–50%, 3 – 51–75%, 4 – 76–100% of the wall thickness.

The results were measured for each segment and for the entire left ventricle as a sum of scores.

Early and delayed MVO was defined as dark regions surrounded by enhanced myocardium on early and delayed gadolinium enhancement images, respectively. Early MVO was assessed quantitatively and semi-quantitatively on early gadolinium enhancement images. Semi-quantitative analysis in 16 segments was performed using the 5-point score corresponding to eMVO transmurality: 0 – absence of eMVO, 1 – 1–25%, 2 – 26–50%, 3 – 51–75%, 4 – 76–100% involvement of the wall thickness.

Quantitative analysis was done manually by tracing of MVO area at each slice from the apex to the base of the entire left ventricle (Figure 1). The volume and the mass of MVO for each slice were calculated by the MASS software. The results were given in mL and grams. A density of the myocardium of 1.05 g/mL was assumed.

Delayed MVO (dMVO) was defined as a dark region surrounded by enhanced myocardium on images obtained 15 min. after contrast administration. Delayed MVO was assessed quantitatively on delayed enhanced images with the same procedure as described for eMVO. The results were given in grams.

Infarct area was defined as area of hyperenhancement (DE) on delayed gadolinium enhancement images obtained 15 min. after i.v. contrast injection in a short-axis orientation. Endo- and epicardial contours were transferred from the functional analysis and manually corrected if needed. Hyperenhanced and unenhanced regions were automatically detected. Full width at half-maximum technique was used for infarct area detection [16]. In a presence of dMVO, the dark spots were then manually traced so the DE volume included dMVO volume (Figure 2).

The DE and dMVO transmurality were assessed with the same scoring system as for eMVO. The results were given as a sum of scores.

Statistical analysis

Statistical analysis was performed using SPSS 15 (SPSS, Chicago, USA) software.

Results for continuous variables were given as mean ± standard deviation (SD) or median ± interquartile range (IQR).

For assessment of normal distribution (ND), Kolmogorov-Smirnov test was used. Differences between groups were tested with χ^2 test for categorical variables, Student's t-test or one-way analysis of variance with Bonferroni's multiple post-test comparison for continuous variables with ND and Mann-Whitney U or Wilcoxon Signed-Rank test for continuous variables without ND. Pearson test or Spearman rank test were used for linear correlation if there were ND or not NT, respectively. A value of $P<0.05$ indicated statistical significance. Multivariate regression model was used to find out the independent predictors of LV EF at discharge.

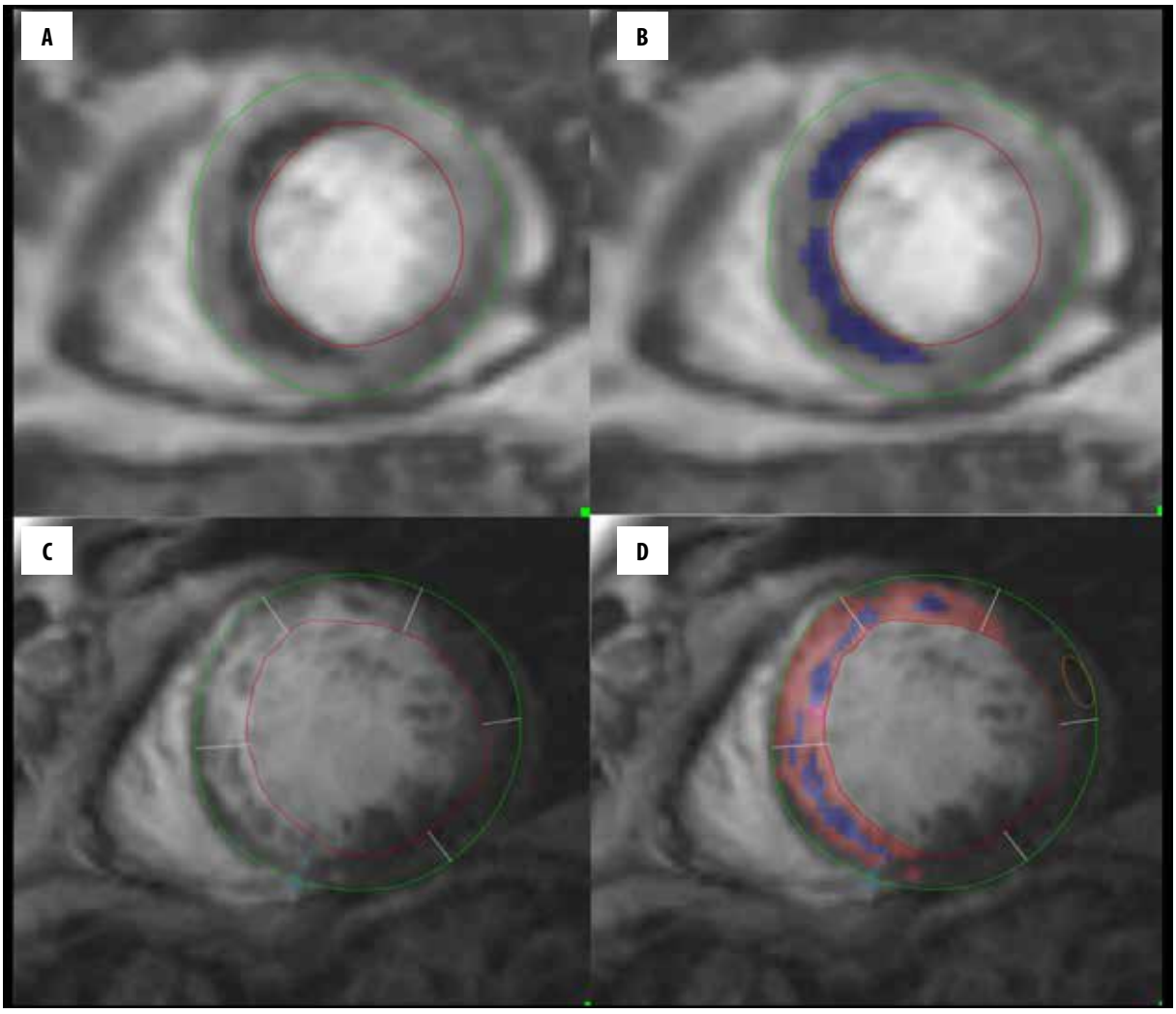


Figure 1. Example of early (eMVO) and delayed microvascular obstruction (dMVO) in a 45-year-old male treated with pPCI within 1.5 hour from chest pain onset. Upper row: early enhancement images without (**A**) and with a manually-traced early MVO area in blue (**B**). Lower row: delayed enhancement images without (**C**) and with manually traced dMVO (**D**). Red line – endocardial border, green line – epicardial border, automatically detected healthy myocardium – orange circle and enhanced myocardium – red circle, infarct – orange area. Microvascular obstruction areas were manually traced (blue).

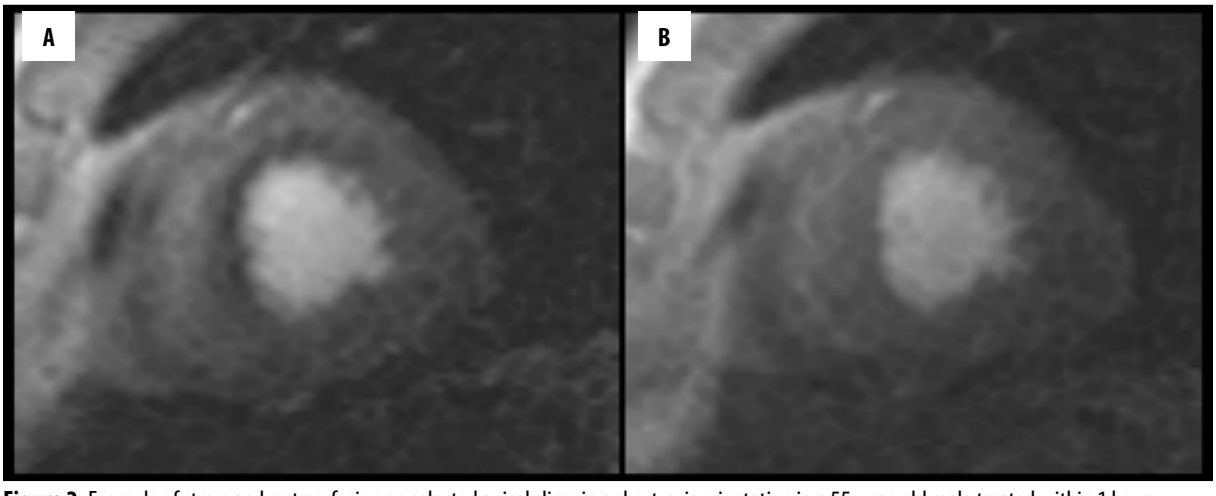


Figure 2. Example of stress and rest perfusion on selected apical slices in a short-axis orientation in a 55-year-old male treated within 1 hour from chest pain onset. (**A**) Stress-induced perfusion defect involving 26–50% of the wall thickness in the septal and anterior segments. (**B**) Defect was not observed at rest.

Results

Feasibility and safety of adenosine stress perfusion

CMR exam was performed 5±2 days after primary PCI. Stress perfusion was successfully preformed in 84 out of 106 included patients. In two patients stress perfusion was stopped by the patient due to severe chest pain after 1 minute of adenosine infusion.

The reason for withdrawal of adenosine infusion in 20 patients were: systolic blood pressure <100 mmHg in 11 pts, bradycardia <50 b/min in 2 pts. or patients' incompliance in 7 pts.

Mild adverse effects occurred in 53% of scanned patients and included feeling of warmth (21%), chest pain (15%), dyspnoe (19%), headache (10%) and temporary atrio-ventricular block 9%.

Significant (more than 20 mmHg) systolic pressure drop or persistent AV block was not observed in any patient. There were no significant adverse events requiring medical treatment during and after adenosine perfusion administration.

Stress, rest, early and delayed MVO

Patients' baseline characteristic is presented in Table 1. Perfusion defects were found in 79 pts. (94%) in stress condition (stress MVO, sMVO) and in 61 pts. (72.6%) at rest (rest MVO).

Rest MVO was never observed in patients without stress-induced MVO. Patients without sMVO had almost normal EF (57%±2.8) and a very small infarct size that covered 7.4±5.1% of the left ventricle. EF was significantly higher and infarct size was significantly smaller compared to the subgroup with stress-induced MVO only (EF=45.7±10.8%; DE%LV 28.6±17.4%, P<0.01), and with both stress-induced and rest MVO (EF=38.4±9.1%; DE% LV 31.8±11%, P<0.01).

The eMVO, dMVO and rest MVO were not found in any patient without stress-induced MVO.

Microvascular obstruction was visible in 59 pts. (70.2%) on early gadolinium enhancement images and in 41 pts (48.8%) on delayed gadolinium enhancement images.

Comparison of rest MVO, stress MVO, early MVO and delayed MVO and its relation to LV function and infarct size

Median sum of scores was significantly different for stress MVO 13 (7;18) versus rest MVO 3 (0.5; 6), P<0.001, for rest MVO 3 (0.5;6) and early MVO 3 (0;8) P=0.047, early MVO and delayed MVO 0 (0;4) P<0.001.

The highest negative correlation with EF was revealed by an infarct size measured semi-quantitatively as a sum of scores (r=–0.687 P=0.01) and stress MVO (r=–0.621 P=0.01).

Differences between LV function, infarct size and time to pPCI in groups of patients with and without MVO assessed separately with stress perfusion, rest perfusion, eMVO and dMVO are presented in Table 2.

Multivariate regression analysis revealed that infarct size, perfusion stress and rest defects are independent predictors of EF measured within one week after p PCI (Table 3).

Segmental analysis revealed the highest negative correlation of segmental kinesis with infarct transmurality scores and stress perfusion transmurality scores: r=–0.820, P<0.001 and r-0.761 P<0.001, respectively.

Table 1. Patients' baseline characteristics and CMR results.

		n= 84	
Age, yrs		57.5	(10.7)
Gender: M		64	(76%)
Hypertension, n		52	(62%)
Diabetes, n		8	(9%)
Hypercholesterolemia, n		20	(24)
Smoking, n		58	(69%)
Segment LAD	seg. 6, n	50	(59.5%)
	seg. 7, n	34	(40.5%)
Time to pPCI, h		3.9	(2; 6)
IIb/IIIa inhibitor, n		67	(79.8%)
Trombectomy, n		4	(4.7%)
TnI max [ng]		52.6	(21; 120)
CMR results			
LV EDV [mL]		174	(42.6)
LV ESV [mL]		103.1	(34.2)
LV EF [%]		41.0	(10.5)
DE mass [g]		45.1	(22.7)
DE% LV [g]		29.4	(19.6; 40.5)
DE sum of scores		20	(15;27)
Stress MVO sum of scores		13	(7; 18)
Rest MVO sum of scores		3	(0.5; 6)
Early MVO sum of scores		3	(0; 8)
Early MVO mass [g]		5.2	(0; 14.2)
Delayed MVO sum of scores		0	(0;4)
Delayed MVO mass [g]		0.31	(0; 3.6)

Gender, hypertension, diabetes, hypercholesterolemia, smoking, segment LAD, treatment with IIb/IIIa inhibitor and trombectomy were given in numbers (%). Age, time to pPCI, TnI max (maximal level of TnI), DE sum of scores (infarct size measured as a sum of scores), stress perfusion sum of scores, rest perfusion sum of scores, early MVO sum of scores, delayed MVO sum of scores, early MVO mass, delayed MVO mass were given as median (IQR). LV EDV, LV ESV, LV EF and DE mass (mass of infarction) were given as mean (SD). yrs – years; n – number; h – hours.

Table 2. Differences between patients with and without MVO assessed by stress perfusion, rest perfusion, early and late gadolinium enhancement.

	Stress MVO (sMVO)			Rest MVO (rMVO)			Early MVO(eMVO)			Delayed MVO (dMVO)		
	Yes 79	No 5	P-value	Yes 61	No 23	P-value	Yes 25	No 59	P-Value	Yes 41	No 43	P-value
LV EDV [mL]	172.8 (42.9)	194 (35.5)	ns	171.7 (40.2)	188.1 (47)	ns	176.6 (39.4)	168 (50)	ns	178.1 (38.1)	169.8 (48.3)	ns
LV ESV [mL]	104.5 (34.6)	83.8 (20.5)	ns	106.1 (33.5)	98.4 (35.3)	ns	110.9 (32.5)	84.8 (31.5)	0.001	112.5 (32.9)	93.5 (34.2)	0.042
LV EF [%]	40 (9.9)	57.2 (2.8)	0.001	38.4 (9,.1)	48.6 (10.7)	0.001	37.3 8.1	49,.8 (10.1)	0.001	36.9 (7.9)	45.4 (11.1)	0.007
DE mass [g]	46.2 (30.8 60.2)	8.3 (4.9;19)	0.001	46.8 (36.2; 62)	29 (8.4; 56.3)	0.014	24.1 9.1; 36.4	49.2 (39.9; 67.5)	0.000	56.6 (44.1; 77.9)	29.4 (18.1; 45.1)	0.000
DE%LV [%]	31.2 (21.2; 41.7)	6.5 (3.3; 11.9)	0.001	33.5 (23; 40.1)	18.5 (6.9; 45)	0.033	15.7 (8.1; 30.7)	34.4 (24.6; 42.7)	0.000	36.8 (28.3; 44.9)	21.0 (13.6; 34.1)	0.001
Time to PCI [hour]	3 (2;5)	1.5 (1;2.5)	0.001	4 (3;5)	3 (1.5;3)	0.017	2 (1.75; 3.5)	4 (3;5)	0.002	4 (3;6)	3 (2;4)	0.023

LV EDV, LV ESV, LV EF were given as mean (SD). Mass of infarct (DE mass), mass of infarct presented as a% of left ventricular mass (DE% LV), time to pPCI were presented as median (IQR).

Table 3. Multivariate regression analysis revealed that only infarct size, stress MVO and rest MVO were independent predictors of LV EF at discharge.

	Unstandardized coefficients		Standardized coefficients	Sig.	R2 change	F change	Sig. F change
	B	Std Error	Beta				
Infarct size (sum of score)	–0.746	0.136	–0.604	0.001	0.5	79.7	0.0001
Stress MVO (sum of scores)	–0.475	0.145	–.385	0.002	0.035	5.82	0.018
Rest MVO (sum of score)	–0.588	0.247	–0.259	0.020	0.032	5.65	0.02

Discussion

We describe the largest group of patients ever published with STEMI treated with primary PCI that underwent CMR with stress and rest first-pass perfusion within the first week after intervention.

Previous studies on a smaller number of patients reported that there were no significant adverse events during adenosine infusion in patients with an acute coronary syndrome and myocardial infarction [14–19]. However, there was no information in how many cases adenosine infusion was stopped or withdrawn during CMR examination due to patients' incompliance (dyspnoe at rest and difficulty with breath holding), lower systolic blood pressure or bradycardia.

For those reasons, we were unable to perform stress perfusion in 21% of patients. Generally, adenosine is contraindicated if systolic blood pressure is lower than 95–100 mmHg and HR lower than 45–50 BPM. In our study we used stricter criteria with systolic blood pressure border set to 100 mmHg and HR to a border of 50 BPM.

We have not observed any significant adverse event during adenosine infusion but our results are representative for STEMI patients with TIMI 3 flow restoration without bradycardia and hypotonia only.

Despite TIMI flow 3 in the majority of patients we observed a perfusion defect in stress condition within the infarct region.

We propose to use a term "stress MVO" for perfusion defect phenomenon found on stress first-pass perfusion images within the infarct region.

In a recently published study, Wong D. assessed infarct-region myocardial blood flow at rest and during 3 min. of adenosine infusion. He observed that adenosine response of the infracted area was deeply reduced and achieved only an 80% increase in MBF [16].

"No reflow" phenomenon is a very complex pathology with a component of mechanical small vessel obstruction (debris, oedema) but also with endothelial cell dysfunction. The first component could be assessed at rest by: rest MVO, early MVO or delayed MVO. We hypothesized that stress-induced MVO could be related to endothelial cell dysfunction with inappropriate response to vasodilatations rather than to mechanical obstruction [20].

We showed that all 4 methods for MVO imaging gave different results, with delayed MVO (dMVO) showing just the "tip of the iceberg" and stress-induced MVO on the other hand revealing the whole microvascular problem.

The differences in prevalence of delayed MVO and rest MVO were previously reported in STEMI patients [21,22]. Although rest MVO and eMVO were detected in a similar number of patients, there were patients described with eMVO but with a perfusion defect at rest.

The eMVO imaging in our study was always performed after stress perfusion and rest perfusion imaging was always carried out at the end of the study which could theoretically influence the results. That can suggest that the effect of adenosine would last longer and eMVO assessed after stress perfusion could give different results than eMVO assessed after perfusion at rest. This theory needs further research.

We showed that presence of MVO detected with all 4 CMR methods was related to a longer ischemia time measured as time to pPCI and these findings are consistent with previous reports [23].

The prevalence of rMVO and eMVO was similar to the reports previously published [24,25].

The prevalence of dMVO was lower in our report compared to similar previous publications. [24,25]. Probably it is related to the pre-selection of patients with only TIMI 3 flow. However, MVO in patients with TIMI 3 flow was detected previously only in 36% of patients compared to 83% in TIMI 0 and 1 [26].

We showed that independent predictors of EF% at discharge from hospital included not only the infarct size and rest MVO but also stress MVO. Wong et al. showed that microvascular blood flow (MBF) in stress condition was a predictor of EF% at 4 months when assessed in a univariate analysis. That significance was not confirmed in a multivariable analysis [16]. We find these findings very interesting and their impact on a long-term prognosis should be established by further studies.

In our segmental analysis we found a very high correlation between transmurality of infarct as well as the stress perfusion defect and impairment of segmental function. These findings are concordant with previous studies.

Study limitation

We used only visual analysis for segmental analysis of function and for stress and rest perfusion assessment. We use this approach in our daily routine practice and theoretical advantages of semi-quantitative assessment are questionable. On the other hand, there is no commercially available software for quantitative perfusion analysis which could be alternative.

Early MVO and delayed MVO were assessed on the entire left ventricle, but stress and rest MVO only on 3 selected slices. The use of high-resolution k-t SENCE first-pass perfusion technique that enables entire left ventricle coverage could be a future [12].

We used visual analysis for regional function assessment instead of wall thickness and thickening measurements. On the other hand, the left ventricular wall thickness and the wall thickening [in mm] in each segment is also very operator-dependent, because it is traced manually. Moreover, due to the ventricle shape, especially at the apical segments, we have to deal with overestimation of segmental thickness where slices are not perpendicular to the LV wall. Also, in basal septal segments the left ventricle outflow track (LVOT) leads to underestimation of the true thickening. With visual assessment we do not experience these problems and the apical segments are additionally assessed in long-axis planes.

We did not compare the CMR data to the detailed angiographic findings. We studied the pre-selected group of patients with the first anterior infarction treated with pPCI of the left anterior descending artery (LAD) with stent implantation and restoration of TIMI 3 flow, without any other significant lesions.

The aim of our study was to compare 4 CMR methods of MVO assessment with each other and not to compare them with angiographic data.

Conclusions

We found significant differences between the prevalence and size of microvascular obstruction detected by stress perfusion, rest perfusion, early and delayed gadolinium-enhanced imaging.

The highest prevalence of stress MVO suggests that this technique assesses the mechanism of MVO not detected by rest techniques. This could be a potential purpose of development of new treatment strategies. Stress-induced MVO, infarct size and rest perfusion were independent predictors of LV EF at discharge.

The results are encouraging for further research on MVO influence on long-term LV function and patient mortality.

References:

1. Ndrepepa G, Keta D, Schulz S et al: Prognostic value of minima blood flow restoration In patients with acute myocardial infarction after reperfusion theraphy. Clin Res Cardiol, 2010; 99: 13–19
2. Kloner RA, Ganote CE, Jennings RB: The "no-reflow" phenomenon after temporary coronary occlusion in the dog. J Clin Invest, 1974; 54: 1496–508
3. Judd RM, Lugo-Olivieri CH, Arai M et al: Physiological basis of myocardial contrast enhancement in fast magnetic resonance images of 2-day-old reperfused canine infarcts. Circulation,1995; 92: 1902–10
4. Beek AM, Nijveldt R, van Rossum AC: Intramyocardial hemorrhage and microvascular obstruction after primary percutaneous coronary intervention Int J Cardiovasc Imaging, 2010; 26: 49–55
5. Wu KC, Zerhouni EA, Judd RM et al: Prognostic significance of microvascular obstruction by magnetic resonance imaging in patients with acute myocardial infarction. Circulation, 1998; 97: 765–72
6. Gerber B, Rochitte C, Melin J et al: Microvascular obstruction and left ventricular remodeling early after acute myocardial infarction. Circulation, 2000; 101: 2734–41
7. Rochitte CE, Lima JA, Bluemke DA et al: Magnitude and time course of microvascular obstruction and tissue injury after acute myocardial infarction. Circulation, 1998; 98: 1006–14
8. Hombach V, Merkle N, Bernhard P et al: Prognostic significance of cardiac magnetic resonance imaging: Update 2010. Cardiol J, 2010; 17: 549–57
9. Kramer CM, Barkhausen J, Flamm SD et al: Standardized cardiovascular magnetic resonance imaging (CMR) protocols, society for cardiovascular magnetic resonance: board of trustees task force on standardized protocols. J Cardiovasc Magn Reson, 2008; 10: 35
10. Trieb T, Mayr A, Klug G et al: Patterns of myocardial perfusion in the acute and chronic stage after myocardial infarction: A cardiac magnetic resonance study. Eur J Radiol, 2012; 81: 767–72
11. Weir RA, Murphy CA, Petrie CJ et al: Microvascular obstruction remains a portent of adverse remodeling in optimally treated patients with left ventricular systolic dysfunction after acute myocardial infarction. Circ Cardiovasc Imaging, 2010; 3(4): 360–67
12. Mather AN, Lockie T, Nagel E et al: Appearance of microvascular obstruction on high resolution first-pass perfusion, early and late gadolinium enhancement CMR in patients with acute myocardial infarction. J Cardiovasc Magn Reson, 2009; 11(1): 33
13. Nijveldt R, Beek AM, Hofman MB et al: Late gadolinium-enhanced cardiovascular magnetic resonance evaluation of infarct size and microvascular obstruction in optimally treated patients after acute myocardial infarction. J Cardiovasc Magn Reson, 2007; 9: 765–70
14. Hombach V, Grebe O, Merkle N 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(6): 549–57
15. Plein S, Younger JF, Sparrow P et al: Cardiovascular magnetic resonance of scar and ischemia burden early after acute ST elevation and non-ST elevation myocardial infarction J Cardiovasc Magn Reson, 2008; 10: 47
16. Wong DT, Leung MC, Richardson JD: Cardiac magnetic resonance derived late microvascular obstruction assessment post ST-segment elevation myocardial infarction is the best predictor of left ventricular function: a comparison of angiographic and cardiac magnetic resonance derived measurements Int J Cardiovasc Imaging, 2012; 28(8): 1971–81
17. Greenwood JP, Younger JF, Ridgway JP et al: Safety and diagnostic accuracy of stress cardiac magnetic resonance imaging *vs.* exercise tolerance testing early after acute ST elevation myocardial infarction Heart, 2007; 93: 1363–68
18. Wong DT, Leung MC, Das R et al: Diagnostic accuracy of adenosine stress cardiovascular magnetic resonance following acute ST-segment elevation myocardial infarction post primary angioplasty. J Cardiovasc Magn Reson, 2011; 13: 62
19. Ingkanisorn WP, Kwong RY, Bohme NS et al: Prognosis of negative adenosine stress magnetic resonance in patients presenting to an emergency department with chest pain. J Am Coll Cardiol, 2006; 47: 1427–32
20. Vanhoutte PM: Endothelial dysfunction and atherosclerosis. Eur Heart J, 1997; 18(Suppl.E): E19–29
21. Lund GK, Stork A, Saeed M et al: Acute myocardial infarction: evaluation with first pass enhancement and delayed enhancement MR imaging compared with 201Tl SPECT imaging. Radiology, 2004; 232(1): 49–57
22. Yan AT, Gibson CM, Larose E et al: Characterization of microvascular dysfunction after acute myocardial infarction by cardiovascular magnetic resonance first-pass perfusion and late gadolinium enhancement imaging. J Cardiovasc Magn Reson, 2006; 8(6): 831–37
23. Tarantini G, Cacciavillani L, Corbetti F et al: Duration of ischemia is a major determinant of transmurality and severe microvascular obstruction after primary angioplasty: a study performed with contrast-enhanced magnetic resonance. J Am Coll Cardiol, 2005; 46: 1229–35
24. Bekkers SC, Backes WH, Kim RJ: Detection and characteristics of microvascular obstruction in reperfused acute myocardial infarction using an optimized protocol for contrast-enhanced cardiovascular magnetic resonance imaging. Eur Radiol, 2009; 19: 2904–12
25. Nijveldt R, Hofman MBM, Hirsch A et al: Assessment of microvascular obstruction and prediction of short-term remodeling after acute myocardial infarction: Cardiac MR imaging study. Radiology, 2009; 250: 363–70
26. Lima JA, Judd RM, Bazille A et al: Regional heterogeneity of human myocardial infarcts demonstrated by contrast-enhanced MRI: Potential mechanisms. Circulation, 1995; 92: 1117–25