Assessment of resting perfusion defect in patients with acute myocardial infarction: comparison of myocardial contrast echocardiography with contrast-enhanced magnetic resonance imaging

Maria Olszowska, Magdalena Kostkiewicz, Piotr Podolec, Paweł Rubiś, Wiesława Tracz

Department of Cardiac and Vascular Diseases, Institute of Cardiology, Jagiellonian University *Collegium Medicum*, Krakow, Poland

Abstract

Background: Contrast-enhanced magnetic resonance imaging (CE-MRI) can identify myocardial scarring following acute myocardial infarction (AMI).

Aim: To compare myocardial contrast echocardiography (MCE) and CE-MRI in detection of resting perfusion defect in patients with acute myocardial infarction.

Methods: Twenty four patients (21 men, 3 women, mean age 58.7 ± 11.4 years) underwent primary percutaneous coronary angioplasty (PCI) for anterior AMI. All patients underwent MCE: segmental perfusion was estimated in real time before and immediately after PCI and on third day after PCI, using low mechanical index (0.3) after 0.3-0.5 ml bolus injections of intravenous OptisonTM. The MCE was scored semiquantitatively as: 1 – homogenous contrast enhancement, 2 – patchy contrast enhancement, 3 – no contrast (non-viable myocardium). All patients underwent CE-MRI on a 1.5 T scanner (SONATA, Siemens) on the third day after PCI. Acquisition of short axis slices was performed before and 20 min after injection of Gd-DPTA (0.15 mmol/kg) with an inversion recovery TurboFLASH sequence (TE 1.1 ms, TR 700 ms, flip angle 30°) in multiple breath-holds. The pattern of hyperenhancement representing MI (which intensity was more than 150% intensity of myocardium) was quantified by planimetry. The CE-MRI was scored according to the severity of myocardial scar as: 1 – without scar, 2 – < 50% of myocardial thickness, 3 – > 50% of myocardial thickness.

Results: Myocardial perfusion was analysed using MCE and contrast-enhanced MRI in 362 segments. Agreement between MCE and CE-MRI for identification of viable versus necrotic myocardium on third day after PCI was 86% ($\kappa = 0.73$). Thirteen (54%) patients showed transmural necrosis at CE-MRI while 11 (46%) showed non-transmural necrosis. Patients from the transmural necrosis group showed a higher creatine kinase peak ($p = 0.0001$), higher CK-MB ($p = 0.00002$) and higher troponine level ($p = 0.008$), and more impaired baseline regional contractile function ($p = 0.045$). All angiographic parameters were less favourable in this group before as well as after PCI than in patients with non-transmural necrosis.

Conclusions: Myocardial contrast echocardiography correlates very well with CE-MRI in the assessment of myocardial perfusion after PCI in AMI. Contrast-enhanced MRI is accurate technique for assessing the infarct zone. Identification by CE-MRI of transmural necrosis was associated with more impaired left ventricular function, non-reperfused MI, and presence of Q waves in ECG.

Keys words: contrast-enhanced magnetic resonance imaging, myocardial contrast echocardiography

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Introduction

Magnetic resonance imaging (MRI) is a new method for assessing myocardial ischemia. It provides information on myocardial perfusion, viability, and potentially the extent of coronary artery disease (CAD) [1-3]. Contrast--enhanced MRI (CE-MRI) can characterise acute myocardial infarction (AMI) with two well-defined CE patterns [4-6]. The first-pass performed immediately after contrast medium – gadolinium (Gd-DPTA) injection, shows regions of decreased perfusion in association with coronary arterial stenosis. Delayed images (10-20 min after contrast injection) demonstrate regional signal hyperenhancement, corresponding to myocardial necrosis.

The aim of this study was to compare myocardial contrast echocardiography (MCE) and CE-MRI in detection of resting perfusion defect in patients with AMI.

Address for correspondence:

Maria Olszowska MD, PhD, Klinika Chorób Serca i Naczyń, Instytut Kardiologii, Uniwersytet Jagielloński *Collegium Medicum*, ul. Prądnicka 80, 31-202 Kraków, tel.: +48 12 614 22 87, fax: +48 12 423 43 76, e-mail: molszowska@szpitaljp2.krakow.pl

Methods

Patients

The study population included 24 patients (21 men, 3 women; mean age 58.7 ± 11.4 years) admitted to our cath-lab with the diagnosis of first AMI and single vessel disease (occlusion of the left anterior descending coronary artery). All patients underwent primary coronary angioplasty (PCI), within 12 h from the onset of symptoms. The diagnosis was made on the basis of prolonged chest pain (≥ 30 min), ST-segment elevation ≥ 2 mm in 2 adjacent ECG leads and an increase in serum creatinine kinase or troponin levels. Hemodynamically unstable patients were excluded from the study as well as those with renal failure, claustrophobia, pacemaker, lack of consent and due to technical reasons. The study protocol was reviewed and approved by the local ethics committee.

Contrast echocardiography

All patients underwent MCE before and immediately after PCI and on the third day after PCI. Myocardial perfusion was assessed in real time, using the Optison™, a second generation contrast agent which was injected in a bolus (0.3-0.5 ml), at a low mechanical index (0.3), through harmonic imaging and with a 16-segment model of the left ventricle (LV). A semiquantitative evaluation of segmental myocardial perfusion was conducted by a visual analysis of the gray-scale intensity of individual LV segments, based on a three-point scale: 1 – normal perfusion (homogenous contrast effect), 2 – partial perfusion (patchy myocardial contrast enhancement) and 3 – lack of perfusion (no visible contrast effect).

Contrast – enhanced magnetic resonance imaging

All patients underwent CE-MRI on a 1.5 T scanner (SONATA, Siemens) on the third day after PCI. Acquisition of short axis slices was performed before and 20 min after injection of Gd-DPTA (0.15 mmol/kg) with an inversion recovery TurboFLASH sequence (TE 1.1 ms, TR 700 ms, flip angle 30°) in multiple breath-holds. The pattern of hyperenhancement (LE – late enhancement) representing AMI (which intensity was more than 150% intensity of myocardium) and LV mass was quantified by planimetry. Consequently, the percentage of infarcted tissue (scar mass) was calculated. For CE-MRI, the presence or absence of contrast enhancement as well as the transmural extent of LE within each segment was defined visually as: 1 – no enhancement, 2 – < 50% of myocardial thickness, 3 – > 50% of myocardial thickness. The AMI was labeled as transmural if LE was > 75% in at least one segment.

The results of MCE and CE-MRI were compared for the same segments. There were no contraindications to use contrasts agents (Gd-DPTA and Optison™) in patients with AMI.

The ECG criteria for Q-wave AMI included the presence of Q waves, characterised by length > 0.04 s and/or depth > 1/3 of the height of the following R wave, in at least two leads.

Statistical analysis

Continuous data are expressed as mean ± standard deviation and were compared using the Student's t-test and χ^2. Correlation between MCE in the assessment of myocardial perfusion and LE in MRI was evaluated using the Cohen's Kappa test. The value of $\kappa > 0,4$, $\kappa > 0,6$ and $\kappa > 0,8$ indicate fair, good and excellent agreement, respectively.

Results

Study population characteristics are presented in Table I. Of the 384 segments, interpretation and evaluation of MCE and CE-MRI was feasible in 362. The LE was found in 76 segments by MRI (Figure 1). Perfusion defect was detected in 71 segments by MCE (Table II). The CE-MRI showed LE in 27 segments although MCE did not confirm this. On the other hand, perfusion defect was observed in 22 segments by MCE whereas CE-MRI did not detect this defect. Agreement between the MCE and CE-MRI for identification of viable versus necrotic myocardium on third day after PCI was 86% (κ 0.73).

Thirteen (54%) patients showed transmural necrosis on CE-MRI while 11 (46%) showed non-transmural necrosis. Patients from the transmural necrosis group showed a higher creatine kinase peak (p = 0.0001), higher CK-MB (p = 0.00002), higher troponin level (p = 0.008), and more impaired baseline regional contractile function (p = 0.045). All angiographic parameters were worse in this group before as well as after PCI: more often TIMI 0 or 1 and TMPG 0 or 1 were observed than in patients with non-transmular necrosis. Larger mass of MI (p = 0.001) and no reflow-phenomenon (p = 0.046) were also more often observed. The presence of Q waves showed a significant association with CE-MRI presence of transmural necrosis (0.024) (Table I). Total LV mass ranged from 101 g to 222 g, mean 155.5 ± 34.3; and scar mass was 3-36%, mean 13.2 ± 8.7%.

The CE-MRI and MCE were performed safely in this study with no directly attributable adverse events observed.

Discussion

Experimental studies with the use of laboratory animals have confirmed the role of LE by CE-MRI in the determination of the magnitude of the infarction area. The function of the heart during acute ischemia (occlusion of the coronary artery) and reperfusion was studied and a close relationship between LE by CE-MRI and histopatological changes has been reported [7, 8]. However, some investigators postulate that infarct area assessed by CE-MRI is over-estimated in comparison with autopsy

Table I. Characteristics of patients with transmural and non-transmural late enhancement in MRI

	Total n = 24	Transmural LE n = 13	Non-transmural or absent LE n = 11	p
Age [years]	58.8 ± 11.2	62.6 ± 10.5	54.4 ± 10.6	0.066
Women/men	3/21	3/10	0/11	0.141
Reflow in MCE	14	4	10	0.004
No-reflow in MCE	10	9	1	0.046
Scar mass (%)	13.2 ± 8.7	16.6 ± 7.7	4.0 ± 2.4	0.001
CK [U/l]	3409.1 ± 2743.4	5300.8 ± 2300.1	1476.2 ± 1317.9	0.0001
CK/MB [U/l]	402.9 ± 336.4	655.7 ± 284.8	150.1 ± 126.4	0.00002
Troponin [ng/ml]	55 ± 84.1	92.4 ± 96.4	11.6 ± 22.2	0.008
TIMI before PCI				
TIMI 0.1	14	12	2	0.048
TIMI 2.3	10	1	9	0.017
TIMI after PCI				
TIMI 0.1	1	1	0	0.365
TIMI 2.3	23	12	11	0.891
TFC before PCI	63.6 ± 36.2	88.4 ± 41.4	51.9 ± 27.5	0.13
TFC after PCI	42.7 ± 26.2	43 ± 19.4	40.9 ± 31.5	0.487
TMPG before PCI				
TMPG 0.1	13	10	3	0.173
TMPG 2.3	11	3	8	0.138
TMPG after PCI				
TMPG 0.1	8	7	1	0.091
TMPG 2.3	16	6	10	0.301
Q-wave	12	11	1	0.024
WMSI before PCI	1.5 ± 0.09	1.6 ± 0.09	1.5 ± 0.07	0.045
WMSI after PCI	1.4 ± 0.16	1.5 ± 0.14	1.3 ± 0.16	0.032
WMSI 3 day	1.3 ± 0.21	1.4 ± 0.12	1.2 ± 0.19	0.0096

Abbreviations: LE – late enhancement, TIMI – score TIMI (Trombolysis in Myocardial Infarction), cTFC – score cTFC (corrected TIMI Frame Count), TMPG – score TMPG (TIMI Myocardial Perfusion Grade), WMSI – wall motion score index, PCI – coronary angioplasty, MCE – myocardial contrast echocardiography, CK – creatine kinase, p – comparison between transmural vs. non-transmural

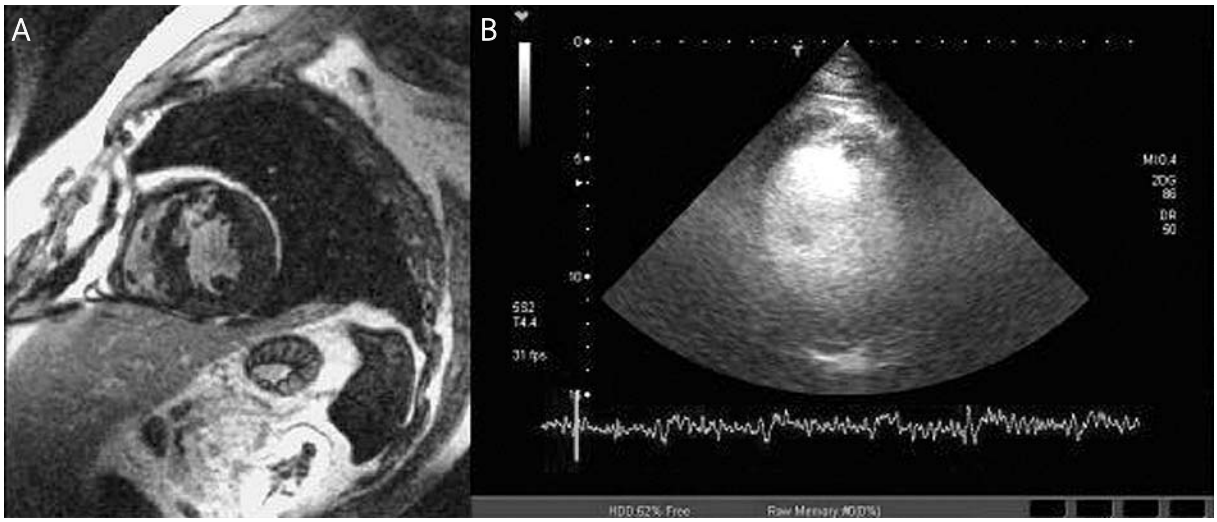

Figure 1. Short axis view in patient on third day after PCI. **A** – CE-MRI: late enhancement in interventricular septum, **B** – MCE perfusion defect in the same region

Table II. Correlation between contrast echocardiography in the assessment of myocardial perfusion and the late enhancement in magnetic resonance: analysis by segments related to acute infarct territory

		MCE	
		perfusion	defect
LE	absent	264	22
	present	27	49
	agreement		86%
	κ		0.73

Abbreviations: MCE – myocardial contrast echocardiography, LE – late enhancement

studies. This discrepancy is best explained by the amount of time between the injection of the contrast media and the onset of analysis. The best correlation of the infarct area assessed by CE-MRI and morphological studies was observed when the time between contrast injection and analysis was 20-30 min. Additionally, time from the onset of infarction until the CE-MRI examination is also of great importance. Studies performed after resolution of the edema of paranecrotic tissue were more accurate in the determination of the infraction area [9]. In our study, all CE-MRI examinations were performed in patients on the third day of infraction and the time between contrast injection and analysis was 20 min.

In the current literature, only limited number of studies analysed myocardial perfusion with both MCE and CE-MRI. In these reports a close correlation between MCE and CMR in the assessment of perfusion deficit in patients with intramural MI has been observed [10]. Our results confirm these findings.

Some authors compared the mass of necrotic tissue assessed by LE with other clinical parameters. Schuijf et al. performed qualitative (visual) and quantitative analysis of the infarcted area and compared it with the contraction of LV. They reported a close correlation of visual and quantitative analysis for the assessment of infarcted area [11]. Likewise, Ingkanisorn et al. stressed the utility of CE-MRI in the assessment of the amount of necrotic tissue [12].

The relationship between LE and the presence of a Q-wave in ECG in patients with MI is still controversial. Petersen et al. did not observe any relation between Q-wave and the presence of LE [13]. According to Moon et al., the presence of a Q-wave is by far determined by the magnitude of the infarction area [14]. However, Cacciavillani et al. reported a close correlation between Q-wave MI and LE. In our study, we observed a Q-wave in ECG in the great majority of patients (85%) with transmural late enhancement [15].

The results of our study show that CE-MRI and MCE have high sensitivity for detection of perfusion defects and may be applied in the clinical setting. The MCE has the relevant advantage of being easily performed at the bedside, providing immediate information on myocardial perfusion that might affect therapeutic strategy. Both these methods performed on the third day after AMI can be used to distinguish between myocardial necrosis and normal myocardium. These techniques do not require exercise or pharmacological stress testing, which may be of practical utility in patients with AMI. The CE-MRI and MCE may be suitable for staging and follow-up of patients with coronary artery disease as well as for evaluating new cardioprotective agents designed to preserve ischemicaly injured myocardium.

Conclusions

The MCE correlates very well with CE-MRI in the assessment of myocardial perfusion after PCI in AMI. The CE-MRI is accurate technique for assessing of infarct zone. Identification by CE-MRI of transmural necrosis is associated with more impaired left ventricular function, non-reperfused MI, and presence of Q-waves.

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Ocena spoczynkowego ukrwienia mięśnia sercowego u chorych z ostrym zawałem serca metodą echokardiografii kontrastowej i rezonansu magnetycznego z zastosowaniem kontrastu paramagnetycznego

Maria Olszowska, Magdalena Kostkiewicz, Piotr Podolec, Paweł Rubiś, Wiesława Tracz

Klinika Chorób Serca i Naczyń, Instytut Kardiologii, Uniwersytet Jagielloński *Collegium Medicum*, Kraków

Streszczenie

Wstęp: Rezonans magnetyczny z zastosowaniem kontrastu paramagnetycznego (ang. *contrast-enhanced magnetic resonance imaging*, CE-MRI) umożliwia ocenę obszaru uszkodzenia mięśnia lewej komory u chorych z ostrym zawałem serca (ang. *acute myocardial infarction*, AMI) oraz precyzyjne określenie jego pełnościennego i niepełnościennego charakteru.

Cel: Porównanie spoczynkowego defektu perfuzji mięśnia sercowego ocenianego metodą echokardiografii kontrastowej (ang. *myocardial contrast echocardiography*, MCE) i CE-MRI u chorych z AMI.

Metody: Badaniami objęto 24 chorych (21 mężczyzn, 3 kobiety, średni wiek 58,7 ± 11,4 roku) z ostrym zawałem ściany przedniej, u których wykonano skuteczną pierwotną przezskórną angioplastykę wieńcową (PCI) gałęzi międzykomorowej przedniej. U wszystkich chorych wykonano badanie MCE z zastosowaniem Optisonu. Perfuzję mięśnia sercowego analizowano bezpośrednio przed i po PCI, a także w 3. dniu po PCI. Zakontrastowanie mięśnia serca oceniano półilościowo w skali trójstopniowej: 1 – prawidłowa perfuzja, 2 – częściowa perfuzja, 3 – brak perfuzji. Badanie CE-MRI przeprowadzono w 3. dobie obserwacji przy użyciu tomografu Siemens Magnetom SONATA 1,5 T, przed i 20 min po dożylnym podaniu 0,15 mmol/kg paramagnetycznego środka kontrastowego – gadolinium Gd-DTPA, z zastosowaniem sekwencji TurboFLASH (TE 1,1 ms, TR 700 ms, kąt magnetyzacji 30°). Obszar późnego kontrastowania (LE) charakteryzował się co najmniej 150-procentową intensywnością sygnału otaczającego miokardium. Obszar LE uznawano za pełnościenny, jeśli obejmował > 50% grubości mięśnia lewej komory.

Wyniki: W 362 segmentach analizowano perfuzję mięśnia sercowego przy użyciu MCE i CE-MRI. Zgodność oceny obecności nieodwracalnego obszaru martwicy przy użyciu CE-MRI z segmentami pozbawionymi perfuzji w MCE wynosiła 86% (κ = 0,73). W CE-MRI u 13 (54%) chorych stwierdzono pełnościenne, a u 11 (46%) niepełnościenne LE. U chorych z pełnościennym LE stwierdzono istotnie statystycznie wyższe stężenia: kinazy kreatynowej (p = 0,0001), frakcji CK-MB (p = 0,00002) i troponiny (p = 0,008), a także gorszą wyjściową kurczliwość lewej komory (p = 0,045). Parametry angiograficzne były również gorsze w tej grupie chorych zarówno przed, jak i po PCI. W grupie z pełnościennym LE częściej występowało zjawisko *no-reflow* (p = 0,046), załamek Q w EKG (p = 0,024), stwierdzono większą masę uszkodzonego mięśnia serca (p = 0,001).

Wnioski: Echokardiografia kontrastowa dobrze koreluje z CE-MRI w ocenie defektu perfuzji mięśnia sercowego u chorych z zawałem serca po PCI. Rezonans magnetyczny z zastosowaniem kontrastu paramagnetycznego (gadolinium) umożliwia ocenę lokalizacji i rozległości zawału serca. Rozpoznanie pełnościennego zawału serca w CE-MRI korelowało z gorszą kurczliwością lewej komory, częściej występującym zespołem *no-reflow* i obecnością załamka Q w EKG.

Słowa kluczowe: rezonans magnetyczny, echokardiografia kontrastowa

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Adres do korespondencji:

dr hab. n. med. Maria Olszowska, Klinika Chorób Serca i Naczyń, Instytut Kardiologii, Uniwersytet Jagielloński *Collegium Medicum,* ul. Prądnicka 80, 31-202 Kraków, tel.: +48 12 614 22 87, faks: +48 12 423 43 76, e-mail: molszowska@szpitaljp2.krakow.pl