Prognostic value of late gadolinium enhancement in cardiovascular magnetic resonance imaging after acute ST-elevation myocardial infarction in comparison with single-photon emission tomography using Tc99m-Sestamibi

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Background
Infarct size is an important predictor of cardiac risk after acute myocardial infarction. The established modality for its assessment is Tc99m-Sestamibi Single-photon emission computed tomography (SPECT). In recent years, data are emerging demonstrating that scar size as assessed by late gadolinium enhancement in cardiovascular magnetic resonance imaging (CMR) as well as the presence of microvascular obstruction (MO) may also provide prognostic information, however, so far no direct comparisons of both modalities have been reported.

Methods
We retrospectively analysed patients (n = 281) with acute ST-elevation myocardial infarction and primary angioplasty who underwent Tc99m-Sestamibi-SPECT and CMR on a 1.5 T scanner at a median of 4.3 (IQR: 3.7–5.1) and 4.9 (IQR: 4.1–5.9) days after the acute event, respectively. The primary endpoint of the study was a composite of all-cause mortality, recurrent myocardial infarction and congestive heart failure requiring hospitalization.

Results
During a median follow-up of 3.0 (IQR: 2.0–4.5) years, 24 events occurred. The best predictor was MO (P = 0.0001), followed by infarct size by CMR (P = 0.0043) and infarct size by SPECT (P = 0.012) (all P-values corrected for clinical risk). In a multivariate model including clinical and periprocedural parameters, MO remained the only significant predictor in addition to clinical risk.

Conclusions
The extent of MO as determined by CMR has an excellent prognostic value in predicting cardiac events following acute myocardial infarction and may be used as an alternative to infarct size assessment by Tc99m-Sestamibi-SPECT.

Keywords
Cardiovascular magnetic resonance imaging • Tc99m-Sestamibi SPECT • Myocardial infarction • Prognosis

Background
Despite the advances of modern reperfusion strategies, residual necrosis remains a problem in patients suffering from myocardial infarction and leads to significant morbidity and mortality during subsequent years. Non-invasive assessment of infarct size is a useful clinical tool allowing for risk stratification.1 In addition, it can serve as a surrogate marker assessing the success of reperfusion therapy.2 Single-photon emission computed tomography imaging with Tc99m-sestamibi (SPECT) is the established and well-proven tool

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for assessing infarct size with several single and multicentre studies showing a good correlation of infarct size and patient mortality.\(^1\)

In recent years, late gadolinium enhancement in cardiovascular magnetic resonance imaging (CMR) has emerged as an alternative modality for the assessment of myocardial necrosis and fibrosis. The major advantage of CMR compared with SPECT is technical benefits such as higher spatial resolution, which makes this technique suitable for detecting even subendocardial infarcts that have been missed by SPECT in the past.\(^{1,2,4}\) Furthermore, CMR may have logistic advantages due to limitations in the use of SPECT because of restricted availability of technetium-based tracer. In addition, it avoids the considerable radiation exposure to patient and personnel associated with SPECT.

To date, data on the prognostic value of infarct characterization of CMR are limited and currently there are no data available comparing CMR with SPECT. Therefore, the aim of this study was to compare infarct characterization by CMR with infarct size assessed by SPECT for prediction of cardiac events in patients who underwent acute revascularization in acute ST-elevation myocardial infarction.

**Methods**

**Study population and design**

The population of this retrospective, observational study included all patients with acute ST-segment elevation myocardial infarction (STEMI) undergoing both CMR and myocardial perfusion scan after primary angioplasty in the two participating hospitals between 1 April 2002 and 1 October 2010. Patients were diagnosed with STEMI if they presented with typical chest pain lasting at least 20 min in the presence of electrocardiographic (ECG) changes (ST-segment elevation or new onset left bundle branch block). Patients with infarction undergoing primary percutaneous coronary intervention (PCI) were eligible, if the onset of symptoms was <24 h before PCI and if they had ST-segment elevation of at least 0.1 mV in ≥2 limb leads or at least 0.2 mV in ≥2 precardial leads. Patients with prior myocardial infarction were excluded to ensure that CMR findings displayed acute myocardial injury. In addition, patients with contraindications to CMR (pacemaker, internal defibrillator, or other incompatible intracorporal foreign bodies, creatinine clearance <50 mL/min (since May 2007), haemodynamic instability for >7 days after infarction, claustrophobia) were also not included into the study. All the patients gave written informed consent for both investigations.

Immediately after hospital admission, all the patients were treated with PCI of the infarct-related artery; ejection fraction before angioplasty was measured by cineventriculography immediately before revascularization of 0.2 mmol/kg body weight of dimegulin gadopentetat (Magnevist, Bayer HealthCare Pharmaceuticals, Berlin, Germany) after the index event.\(^{10}\) The study protocol was approved by the institutional ethics committee responsible for both participating centres.

**Myocardial perfusion SPECT**

The methods used for the radionuclide studies have been previously described in detail.\(^{11}\) Approximately 1.5 h after iv. injection of 350 MBq technetium-99m-sestamibi myocardial perfusion, SPECT was performed with the patient in the supine position. Dual head camera systems with low-energy, high-resolution collimators were used for the radionuclide studies. Images were acquired in a 64-by-64 matrix with an acquisition time of 40 s per image. The gating technique was used in all the patients with regular heart rhythm; attenuation correction was not available at the time the study was performed. A volumetric sampling tool was applied to create polar maps of the relative distribution of activity throughout the left ventricle.\(^{12}\) Each polar map was adjusted for its own maximal value. The size of the defect was calculated with the use of a threshold of 50%, which was derived from studies that used a phantom, according to previously described methods.\(^{13}\) This method allowed for calculation of the infarct size (as a percentage of the left ventricle) at the time of radionuclide injection.

**CMR imaging**

CMR was performed on 1.5-T systems (Philips Achieva, Philips, Best, the Netherlands and Siemens Sonata and Avanto, both Siemens Healthcare, Erlangen, Germany) equipped with a dedicated cardiac phased-array surface coil. For image acquisition, patients were positioned in a supine position and images were acquired at repeated end-expiratory breath holds with ECG gating. The infarct scar was assessed 15 min after injection of 0.2 mmol/kg body weight of dimegulin gadopentetat (Magnevist, Bayer HealthCare Pharmaceuticals, Berlin, Germany) on the T1-weighted inversion-recovery gradient echo sequence. Pulse sequence parameters were slice thickness 8 mm, excitation every second heart beat for all scanners, repetition time (TR) 3.6 ms, echo time (TE) 1.2 ms, acquisition matrix 160 × 160, field of view (FOV) 320 mm, flip angle 25° for the Achieva scanner, TR 2.3 ms, TE 1.4 ms, matrix 128 × 256, FOV 450 mm flip angle 60° for the Sonata scanner, and TR 40.4 ms, TE 1.5 ms, matrix 176 × 256, FOV 340 mm, flip angle 30° for the Avanto scanner. Contiguous short-axis slices of the left ventricle from the base to the apex as well as two- and four-chamber views of the left ventricle were acquired. The inversion time (TI) was individually adjusted to null normal myocardium (typical range 260–320 ms).

For infarct size, quantification endocardial and epicardial contours were manually traced on each of the short-axis slices by an experienced reader. Infarct size and area of microvascular obstruction (MO) were expressed as a percentage of total LV myocardial volume. Since the definition of the infarcted myocardium based on signal intensity is not standardized at present, we tested both methods published to date—the standard deviation (SD) method as described in the clinical setting by Bondarenko et al.\(^{14}\) and the ‘full-width half-maximum’ method according to Amado et al.\(^{15}\) The SD method calculates mean and SD of remote-healthy myocardium and it takes multiples (usually up to 6) of the SD above the mean remote intensity as threshold for the infarcted area. The full-width half-maximum method uses a threshold of 50% of the maximum signal intensity of the infarct area. In consensus with previous studies, myocardial enhancement required to have at least 10 contiguous myocardial pixels of increased signal intensity.\(^{16}\) MO was defined as an area of hypo-enhancement within the infarcted myocardium and was included in the infarct size calculation. As valid automated detection of MO is not established, the size of MO was quantified by manually delineating areas of decreased signal intensity within the infarcted myocardium, aiming to minimize both an overlap with and a gap to the area of...
automatically detected infarct area. For an image example describing infarct characterization, see Figure 1. The MO analysed in this study is derived from images acquired 15 min after gadolinium application. To distinguish this entity from perfusion defects assessed shortly after gadolinium injection (~1–3 min), it has been described as late\textsuperscript{16} or persistent\textsuperscript{17} MO in other studies. Analysis was performed using software developed in house and validated against a commercially available software package.

Follow-up and study endpoint
Follow-up information was collected by clinical visits, telephone contacts, or questionnaires sent by mail. All reported events were verified by hospital records or contact with the attending physician and adjudicated by at least two physicians unaware of the imaging results. The endpoint of the study was the occurrence of adverse cardiac events, defined as a composite of all-cause death, recurrent myocardial infarction, and new congestive heart failure requiring hospitalization, whichever occurred first. Recurrent myocardial infarction was defined based on the criteria of typical acute chest pain of >20 min duration and either persistent ST-segment elevation and newly developed Q-waves or elevated cardiac markers, or any combination of these.\textsuperscript{18} Recurrent myocardial infarctions during the hospital stay of the index event were not counted as events due to the immediate proximity to the CMR scan. New congestive heart failure was defined as any congestive heart failure (rales, dyspnoea, New York Heart Association functional class III to IV) occurring after patient discharge from the index event and requiring re-hospitalization.

Statistical analysis
Categorical variables were expressed as frequencies and percentages, continuous variables were expressed as means ± SD. All statistical evaluations are based on the event-free survival for the study endpoint using the Kaplan–Meier method; hazard ratios and multivariable analyses were calculated with the Cox proportional hazard model. To make hazard ratios comparable between different variables in multivariable analysis, they are calculated for an increment equivalent to the difference between the 75th and 25th percentile of the variable of interest. Different predictors were compared using $C^*$-Statistics. Statistical significance was accepted for two-sided $P$-values <0.05. Owing to the limited number of events, correction for clinical risk was performed using the GRACE score as the best clinical risk predictor. The statistical package R version 2.10.1\textsuperscript{19} including the package rms\textsuperscript{20} was used for statistical analysis.

Results

Study population
During the study period, 1294 patients with acute STEMI were treated by primary PCI, of which 154 had a recurrent infarction. Out of the 1140 patients with primary infarction, 912 underwent SPECT imaging, and 309 patients underwent CMR. A total of 292 patients underwent both examinations, of which 8 patients were excluded because the image quality of the CMR study was insufficient for automated analysis. From the remaining 284 patients, 281 could be contacted for follow-up after a median interval of 3.0 (IQR: 2.0–4.5) years, resulting in a follow-up rate of 98.9% (Figure 2).

Of the 912 patients undergoing SPECT after STEMI, 23 patients (2.5%) had creatinine levels >1.5 mg/dl at the time of discharge (clearance values were available only in few of these patients), 3 patients (0.3%) had a permanent pacemaker or an internal
defibrillator, so in total 2.8% of the patients undergoing SPECT were ineligible for CMR. Reasons for not performing CMR in the remaining patients were the absence of patient consent because of claustrophobia or other reasons or prolonged haemodynamic impairment (each extrapolated to 2% occurrence from available data), but mainly logistic problems in scheduling the two examinations before discharge caused by low human resources for study coordination due to limited funding.

Clinical and infarct characteristics

Patient and infarct characteristics are summarized in Table 1. The mean age of the patients was 60 ± 12 years, and 225 patients (80%) were male. Most of the patients (222 or 79%) presented in stable condition with Killip class I while 58 patients (21%) were in higher Killip classes. The mean GRACE score was 156 ± 38, equivalent to a probability of in-hospital death of 5%. PCI was performed 5.2 (IQR: 3.4–9.2) h after the onset of symptoms. The infarct was located anteriorly in 128 patients (46%), inferiorly in 125 patients (45%), and laterally in 28 patients (10%). Revascularization was successful in all patients and resulted in an increase in number of patients with a TIMI flow \( \geq 1 \) from 122 (43%) to 276 (98%).

Some patients took part in randomized trials investigating different therapeutic strategies in acute myocardial infarction: 8 patients (3%) in ISAR-BRAVE-1, \(^{21} 5 \) patients (2%) in ISAR-BRAVE-2, \(^{22} 60 \) patients (21%) in ISAR-REVIVAL-2, \(^{23} 96 \) patients (34%) in ISAR-REVIVAL-3, \(^{24} \) whereas 112 patients (40%) were not included in any randomized trial.

There was no significant difference in age (\( P = 0.97 \)), Killip class (\( P = 0.39 \)), and GRACE score (0.22) between these groups, but due to the specific inclusion criteria, there were significant differences in cardiac markers (\( P < 0.001 \) for initial CK and maximal CK) and time to balloon (\( P < 0.0001 \)) between the different trial groups and the patients not included into trials. From the patients included into the ISAR-BRAVE-2 trial, only those with invasive therapy within 24 h from start of symptoms were included into this analysis.

Neither was there a significant difference in outcome between these groups (\( P = 0.43 \)), nor was a significant difference in outcome...
between all patients taking part in randomized trials (16 events or 9%) and those who did not (8 events or 7%, $P = 0.66$).

**Results of SPECT and CMR**

SPECT was performed 4.3 (IQR: 3.7–5.1) days after revascularization and CMR was performed 4.9 (IQR: 4.1–5.9) days after revascularization, so CMR was performed 0.4 (IQR: 0.1–0.8) days later than SPECT ($P < 0.001$).

The mean infarct size in SPECT was 15.1 ± 17.2%, whereas the infarct size in CMR ranged from 8.9 ± 9.1% to 17.2 ± 12.5 for the 6 and 3 SD thresholds, respectively. In 99 patients (35%), MO was observed, and the size of MO was 1.0 ± 2.7%. A summary of SPECT and CMR results is provided in Table 2. There was no significant difference between infarct size in SPECT between the study population and those patients undergoing SPECT alone (15.1 ± 17.2% vs. 13.9 ± 17.3%, $P = 0.20$).

Infarct size in CMR was significantly larger than infarct size in SPECT at a threshold of 2 SD, while measurements using thresholds of 5 and 6 SD and the full-width half maximum method resulted in significantly smaller infarct sizes in comparison with SPECT. The difference between both modalities was the smallest at a threshold of 4 SD above remote myocardium. (Table 3). For the threshold of 4 SD, Pearson’s correlation coefficient $r$ was 0.75.

Using the 4 SD threshold for CMR, the size of anterior and lateral wall infarctions was measured by SPECT as 18.7 ± 20.0% and by CMR as 15.5 ± 13.0; Pearson’s $r$ was 0.79. The size of posterior wall infarctions was measured by SPECT as 10.6 ± 15.0% and by CMR as 12.1 ± 9.1; Pearson’s $r$ was 0.60 (Figure 3).

There was no correlation between time from start of symptoms to coronary intervention and infarct size ($P = 0.44$ for 4 SD threshold) or MO ($P = 0.49$).

**Cardiac events**

During follow-up, 10 patients (3.6%) died, 2 suddenly, 1 due to fatal myocardial infarction, 2 due to cancer, and 5 of unknown cause.
Significant clinical and periprocedural risk predictors were age ($P = 0.03$), ejection fraction before angioplasty ($P = 0.006$), and peak troponin level ($P = 0.008$) as well as the clinical risk scores. Both the GRACE risk score ($P = 0.001$) and the TIMI risk score ($P = 0.002$) showed a significant association with outcome (Table 4).

All imaging parameters were significantly correlated with the outcome. Figure 4 depicts Kaplan–Meier plots showing the risk of cardiac events, stratified by infarct size, and MO by CMR, and infarct size by SPECT. After correction for clinical risk assessed by the GRACE score, the best predictor of the outcome was the size of MO ($P < 0.0001$), followed by infarct size by CMR using the 6 SD threshold ($P = 0.004$) and infarct size by SPECT ($P = 0.012$); other quantification methods like ‘full-width half-maximum’ ($P = 0.008$) or measurements including infarct areas of lower intensity had no better predictive value. Infarct size at 6 SD threshold had a significantly better predictive value than infarct size at 2 SD threshold, compared with the threshold of 3 SD, there was a strong trend ($P = 0.009$ and $P = 0.059$, respectively). MO had a significantly better predictive value than infarct size at all thresholds, with $P$-values ranging from 0.012 for 6 SD to 0.0032 for 2 SD. Detailed results are presented in Table 2.

For the single endpoint of total mortality ($n = 10$), infarct size by SPECT ($P = 0.07$), infarct size by CMR ($P = 0.06$ at 6 SD threshold), and the presence of MO ($P = 0.07$) showed a strong trend with the outcome, but no significant correlation. For the single endpoint of recurrent myocardial infarction ($n = 13$), there was a significant correlation with outcome for infarct size by SPECT ($P = 0.02$) and infarct size by CMR ($P = 0.04$ at 6 SD threshold), while the size of MO correlated highly significant with outcome ($P < 0.001$).

Stepwise backward multivariate Cox regression analysis was performed including statistically significant clinical, periprocedural, and imaging parameters of the univariate model. Besides the GRACE score ($P = 0.0014$), only the size of MO in CMR ($P < 0.0001$) remained a significant predictor of the study endpoint (Table 5).

The presence of MO allowed for detection of a high-risk group of 99 patients (35%) with an event rate of 14.1%, whereas patients without MO had an event rate of 5.5%. With a risk ratio of 2.6, the result of the power calculation of 2.9 was not fully reached.
The main findings of the present report can be summarized as follows: (i) the characterization of acute ST-elevation myocardial infarction by late gadolinium enhancement in CMR provides significant incremental prognostic information beyond clinical risk assessment; (ii) the size of MO has the best prognostic value of all CMR parameters; (iii) the prognostic value of CMR seems to be equivalent to the infarct size assessed by SPECT myocardial perfusion imaging.

Late gadolinium enhancement is a proven method for the detection and quantification of myocardial scars and multiple studies found a good correlation of scar extent by CMR with SPECT\(^1\),\(^2\),\(^5\) in acute myocardial infarction. In addition, Wagner et al.\(^26\) as well as Ibrahim et al.\(^4\) showed that CMR systematically detects subendocardial infarcts that are missed by SPECT.

Although several studies demonstrated that infarct characterization by CMR in acute myocardial infarction is predictive for composite endpoints including stable angina or revascularization,\(^17\),\(^27\) data on the prediction of hard cardiac events are only beginning to emerge. Eitel et al.\(^28\) analysed the ability of CMR to predict cardiac events defined as a composite of death, myocardial infarction, and heart failure requiring rehospitalization, during a 6-month follow-up in a group of 208 patients undergoing acute reperfusion of ST-elevation myocardial infarction and found a significant predictive value both for infarct size ($P < 0.001$) and for late MO ($P = 0.004$). In addition, de Waha et al.\(^16\) demonstrated the incremental predictive value of late MO over and above clinical risk factors and left ventricular ejection fraction ($P = 0.002$) in 408 patients during 19 months of follow-up.

The findings of our study on a population of 281 patients from two tertiary referral centres are consistent with these earlier reports. Both infarct size and MO were significant predictors of the adverse outcome after correction for clinical risk. Of the CMR parameters analysed, MO had the best clinical risk prediction. In a multivariate analysis of clinical and interventional parameters, it was the only remaining significant variable besides the GRACE score.

This finding corroborates the evidence that CMR is a robust modality for risk assessment after acute reperfusion of STEMI. Indeed,
this modality may replace Sestamibi-SPECT for this indication, which would allow for a significant reduction of radiation exposure in these patients.

Quantification of infarct size by CMR is not yet standardized. Both the full-width half maximum and the SD method are used, and while traditionally every signal > 2 SD above remote-healthy myocardium is considered as late enhancement, currently 5 SD are recommended as starting point for analysis in myocardial infarction. A threshold best correlating with visual assessment was found by some authors at 6 SD, which could not be avoided due to logistical reasons. Furthermore, the mean infarct size is quite small and likely accounts for the small size of MO and the relatively small number of clinical events during the follow-up. Owing to the low event rate during the follow-up and the small absolute number of events, the study was slightly underpowered and a full correction for pre-test risk was therefore not feasible.

Confounding effects may derive from the fact that SPECT was performed as a clinical routine examination, while CMR was performed by a team that paid highest attention to optimal image quality. In addition, attenuation correction was not available at the time the study was performed. In addition, infarct size by CMR might be slightly underestimated in comparison with SPECT, because CMR was performed some hours later, which could not be avoided due to logistical reasons.

Although inclusion into this study did not depend on participation in one of the four randomized trial performed during the study period, the conduction of these trials may increase heterogeneity of the study population and may influence outcome.
Finally, the duration between start of symptoms and revascularization of 5.1 h is longer than reported from previous studies including only patients revascularized within the first 12 h from the first symptoms. A reason for this is the policy of the participating hospitals to perform primary revascularizations in STEMI later than 12 h after the onset of symptoms, too, which inevitably increases the median time to PCI.22

In conclusion, late MO on pre-discharge CMR is an incremental predictor of adverse cardiac events and may be useful in the risk stratification of survivors of acute myocardial infarction. The predictive value of MO assessed by CMR is at least comparable with that of infarct size assessment by Tc99m-sestamibi-SPECT. Taken together CMR assessment of infarct size in association with MO detection might represent a superior approach to risk assessment of patients after reperfusion in acute myocardial infarction.

Authors’ contributions
M.H. conceived of the study, participated in study design, data acquisition, statistical analysis, and drafted the manuscript. B.L., A.K., and T.I. participated in data acquisition and analysis and were involved in drafting the manuscript. J.H., C.S., R.B., and J.M. were involved in data acquisition and revised the manuscript critically for important intellectual content. S.M. and A.S. participated in study design and revised the manuscript critically for important intellectual content. All authors read and approved the final manuscript.

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References
Left ventricular partitioning device (‘Parachute’): a multi-modality imaging perspective

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Coronary artery disease is the most important cause of chronic heart failure. After an acute myocardial infarction left ventricular (LV) remodelling (with increased volumes) may occur and is associated with a poor clinical outcome. (i) Although major breakthroughs have been made in the medical treatment of these patients, LV remodelling may progress despite optimal medical therapy. (ii) Symptomatic patients (NYHA class III–IV) with apical aneurisms may be candidates for aneurismectomy, aiming to reduce both systolic and diastolic volumes, decrease myocardial wall stress, and thereby improving LV haemodynamics.

Recently, a percutaneous ventricular partitioning device (Parachute, CardioKinetix, Inc.) has been developed as alternative to the surgical approach. This ‘umbrella-like’ or ‘parachute-like’ device is placed in the apex of the left ventricle and aims to improve LV haemodynamics by isolating the dysfunctional apical region.

Despite this approach is still under research, patients involved in clinical trials are already being treated using this technique. Physicians should be aware of the device usual location and normal characteristics in different imaging modalities in order to identify the device and diagnose potential complications.

The authors present different views of an implanted Parachute device using different imaging modalities. (Panels A–D).

Left ventricular partitioning device (Parachute) seen in different imaging modalities. (A) Four-chambers view transthoracic echocardiography; (B) maximal intensity projection in double-oblique projection of a non-contrasted CT acquisition; (C) cardiac magnetic resonance balanced steady-state-free-precession in the four-chamber view; (D) Coronary angiography revealing the ‘parachute-like’ device in the left ventricle apex. Occasionally, on a regular chest X-ray the device may not be easy to identify, given its low opacity.

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