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Clinical predictors for the manifestation of late gadolinium enhancement after acute myocardial infarction

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Abstract

Despite prompt revascularization, some patients with acute myocardial infarction (AMI) develop myocardial scars, which can be visualized by late gadolinium enhancement (LGE) in cardiovascular magnetic resonance imaging (CMR). Our goal was to identify angiographic findings that were predictive for scar development in patients after reperfused AMI.

We examined 136 patients after first ST-elevated myocardial infarction by CMR after a median of 4 days (range: 2–7). Patients with manifestation of LGE were matched to patients without LGE by means of age and gender. Clinical follow-up with a combined primary endpoint including myocardial reinfarction, congestive heart failure, stroke, death and development of left ventricular thrombus was reported after 24 months.

Patients with manifestation of LGE had a significant longer time of symptom-to-intervention, a higher prevalence of anterior AMI, and more proximal culprit lesions. Furthermore, left ventricular ejection fraction was significantly decreased, and peak values of infarct markers were significantly higher in these patients. Preinterventional thrombolysis in myocardial infarction-0-flow was significantly more frequent in patients with LGE manifestation. The presence of 3-vessel disease (odds ratio 53.99, 95% confidence interval 8.22–354.63, $P<.001$), a proximal culprit lesion, and high creatine kinase myocardial band (CK-MB) values were identified as independent predictors of LGE. Follow-up demonstrated a higher incidence of clinical events in the group with LGE, with the most common cause of heart failure (38.2% vs 7.4%, $P<.001$).

The extent of angiographic findings in AMI plays a major role in the manifestation of LGE. The presence of a multivessel disease, a proximal culprit lesion, and high values of CK-MB are strong independent predictors for LGE manifestation.

Abbreviations: AMI = acute myocardial infarction, CK = creatine kinase, CK-MB = creatine kinase myocardial band, CMR = cardiovascular magnetic resonance imaging, LAD = left anterior descending artery, LGE = late gadolinium enhancement, LV-EF = left ventricular ejection fraction, MACE = major adverse clinical events, NSTEMI = non-ST-elevated myocardial infarction, PCI = percutaneous coronary intervention, SSFP = standard steady-state free precession, STEMI = ST-elevation myocardial infarction, TIMI = thrombolysis in myocardial infarction.

Keywords: cardiac magnetic resonance imaging, culprit lesion, late gadolinium enhancement, multivessel disease, myocardial infarction

1. Introduction

The high diagnostic accuracy of cardiovascular magnetic resonance (CMR) imaging with late gadolinium enhancement (LGE) for the detection of scar tissue after an acute myocardial infarction (AMI) is well recognized.[1–3] Despite epicardial revascularization, there is still a wide spectrum of outcomes in terms of left ventricular morphology. Infarct size after AMI as visualized by LGE (Fig. 1) can range from transmural (Fig. 1A) to absent (Fig. 1B).[4–6] Infarct size has a large impact on outcome after AMI. Recent studies have demonstrated that infarct size is of prognostic value in ischemic heart disease.[7–10] Identification of factors that predict infarct size may allow for early risk stratification, even before later cardiac imaging. Therefore, the aim of this study was to examine the clinical and angiographic records of patients with reperfused ST-elevated myocardial infarction (STEMI) and to determine those factors that impacted LGE.

2. Methods

2.1. Study design and clinical endpoint

We screened patients from January to December 2013 for their first STEMI and who underwent primary percutaneous coronary intervention (PCI) and had imaging by CMR during the index event (Fig. 2). STEMI was defined as ST-segment elevation of at least 0.1 mV in more than 2 extremity leads or at least 0.2 mV in more than 2 precordial leads and pathologically elevated plasma levels for creatine kinase (CK), creatine kinase myocardial band

Editor: Jacek Bil.

NA-K and LK contributed equally.

Funding: This study was supported by HELIOS Kliniken GmbH, [Grant ID 003138].

The authors have no conflicts of interest to disclose.

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Medicine (2017) 96:21(e7004)

Received: 20 March 2017 / Received in final form: 23 April 2017 / Accepted: 26 April 2017

http://dx.doi.org/10.1097/MD.0000000000007004

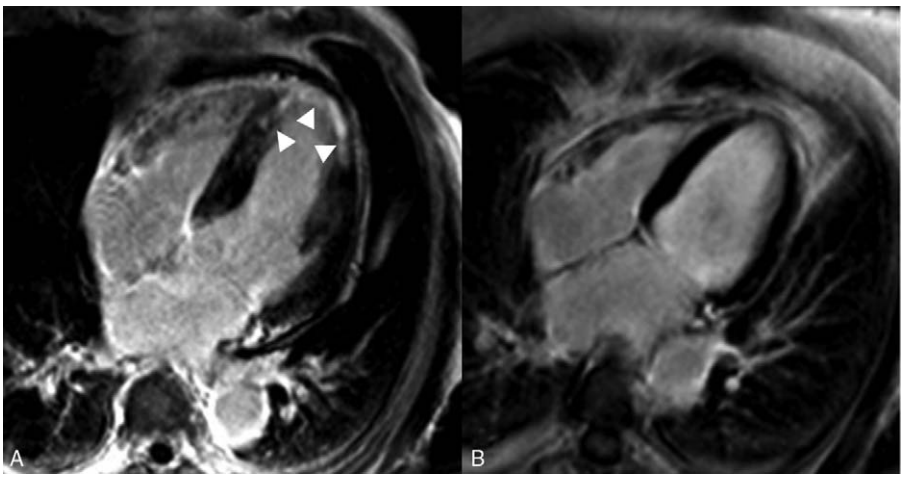

Figure 1. (A) Transmural late gadolinium enhancement (white arrows) of the left ventricular apex in the 4-chamber view after anterior STEMI. (B) Patient without late gadolinium enhancement in the 4-chamber view after anterior STEMI. STEMI = ST-elevation myocardial infarction.

(CK-MB), or high-sensitivity troponin I. Patients with prior myocardial infarction, coronary artery bypass surgery, and patients with contraindications to CMR were excluded. Patients with cardiogenic shock that resulted in a delayed intervention-to-CMR time of >30 days were also excluded. The included 68 patients were matched according to age and gender to a collective of 68 patients with STEMI and absent LGE. This control group included patients of the same or previous years with the same inclusion and exclusion criteria. In total, we included 136 patients into the study.

Peak values of laboratory infarct markers, CMR data, angiographic findings from the time of the index event, and clinical endpoints after a follow-up of 24 months were analyzed. The primary combined endpoint was defined as myocardial reinfarction, congestive heart failure with a left ventricular ejection fraction (LV-EF) <30%, stroke, and death. Independent investigators obtained clinical follow-up data by means of a standardized survey answered by the patients or their physicians and by means of medical records review. In the case of more than 2 clinical events, the more severe one was chosen. All patients gave written informed consent. The local ethics committee approved the study.

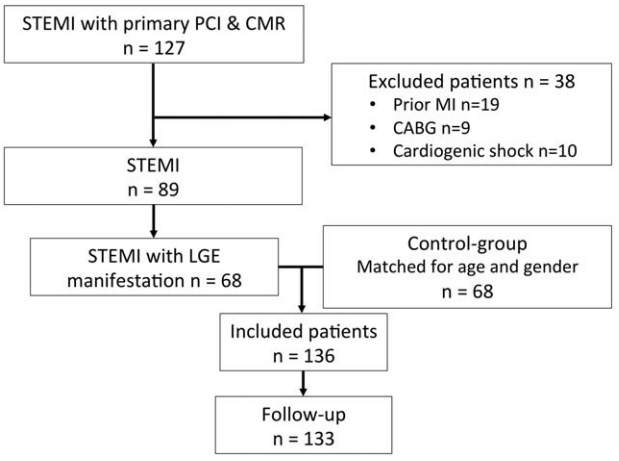

Figure 2. Study flow chart. CABG = coronary artery bypass grafting, CMR = cardiovascular magnetic resonance imaging, MI = myocardial infarction, PCI = percutaneous coronary intervention; STEMI = ST-elevation myocardial infarction.

2.2. Cardiovascular magnetic resonance imaging and image analysis

CMR was performed on a 1.5-Tesla scanner (Intera Achieva, Philips, Best, the Netherlands). Left ventricular function was evaluated by a SSFP (standard steady-state free precession) technique with the 2D turbo gradient echo sequence. As previously described, LGE images of the left and right ventricles were acquired 10 minutes after injection of 0.2 mmol/kg of gadoteridol (Prohance (R), Bracco-Imaging, Konstanz, Germany).[11] A 3D inversion-recovery turbo gradient echo sequence was used. Specific software analysis tools (CMR 42, Version 4.0, Circle Cardiovascular Imaging Inc., Calgary, Canada and Extended MR Workspace 2.6.3.4, Philips Medical Systems, Best, the Netherlands) were used to analyze images and infarct area. LV-EF was calculated by biventricular assessment of the volumes of the endocardial contours in diastole and systole of the 4- and 2-chamber slices. Infarct area was defined as the area of LGE according to the analyses suggested by Wagner et al.[12] Independent observers blinded to angiographic results and clinical events evaluated left ventricular parameters and LGE images.

2.3. Statistical analysis

Categorical variables are described by frequencies; continuous data are expressed as median with minimum and maximum. The Wilcoxon signed-rank test for matched pairs was used to test the distribution of continuous data from the 2 groups. To compare the 2 groups with respect to the distribution of categorical variables, McNemar's χ^2 test was performed. In addition, to identify predictors of LGE, conditional logistic regression for matched case–control groups, that is, conditional (fixed-effects) logistic regression, was used. Results are presented as odds ratio a with 95% confidence interval.

P values less than .05 were considered statistically significant. All statistical analyses were performed using STATA/IC 13.1 software (STAT Corp., LP, TX).

Table 1

Baseline characteristics of the study population based on the presence or the absence of LGE.

	LGE present, n = 68	Control group, n = 68	*P*-value
Age, years	68.0 (21.0–86.0)	69.0 (24.0–87.0)	
Male	30 (44.1%)	29 (42.6%)	
Δ Intervention-to-CMR, d	5.0 (0.0–19.0)	3.0 (0.0–21.0)	.002
Δ Symptom-to-intervention, hours	7.3 (0.0–72.0)	4.0 (1.0–48.0)	.017
Angiografic findings			
TIMI-flow pre-PCI			
0	40 (58.8%)	11 (16.2%)	<.001
I	9 (13.2%)	7 (10.3%)	.804
II	8 (11.8%)	13 (19.1%)	.359
III	11 (16.2%)	37 (54.4%)	<.001
TIMI-flow post-PCI			
0	5 (7.4%)	2 (2.9%)	.453
I	2 (2.9%)	2 (2.9%)	1.000
II	5 (7.4%)	0 (0%)	.063
III	56 (82.4%)	64 (94.1%)	.039
Proximal culprit lesion	27 (39.7%)	4 (5.9%)	<.001
Medial culprit lesion	26 (38.2%)	11 (16.2%)	.014
Distal culprit lesion	7 (10.3%)	18 (26.5%)	.035
Thrombus aspiration	22 (32.4%)	4 (5.9%)	<.001
1-vessel disease	21 (30.9%)	15 (22.1%)	.307
2-vessel disease	11 (16.2%)	6 (8.8%)	.267
3-vessel disease	28 (41.2%)	12 (17.6%)	.004
Culprit lesion			
LAD	34 (50.0%)	20 (29.4%)	.024
RCX	12 (17.6%)	7 (10.3%)	.359
RCA	14 (20.6%)	6 (8.8%)	.096
LV-EF, %	50.0 (21.0–71.0)	59.0 (15.0–76.0)	.015
Peak value levels			
High-sensitivity troponin I	1127 (16–21250)	281 (13–7860)	<.001
CK-MB	87 (12–1540)	33 (12–338)	<.00
Glucose	140.0 (89.0–470.0)	125.0 (75.0–358.0)	.247
Minimum GFR	76.0 (41.0–132.0)	80.5 (35.0–128.0	.438
Cardiovascular risk factors			
Arterial hypertension	48 (70.6%)	43 (63.2%)	.458
Diabetes mellitus	15 (22.1%)	9 (13.2%)	.286
Current smoking	32 (47.1%)	24 (35.3%)	.169
Hyperlipidemia	28 (41.2%)	23 (33.8%)	.442
Obesity (BMI >30)	23 (33.8%)	23 (33.8%)	1.000
Family history for myocardial infarction	25 (36.8%)	19 (27.9%)	.405
Platelet therapy			
Clopidogrel	25 (36.8%)	27 (39.7%)	.845
Ticagrelor	16 (23.5%)	19 (27.9%)	.701
Prasugrel	27 (39.7%)	22 (32.4%)	.487
Use of glycoprotein IIb/IIIa inhibitors	15 (22.1%)	2 (2.9%)	.002

Data is presented as number and percentage of patients. Age, Δ intervention-to-CMR, Δ symptom-to-intervention, troponin, CK-MB, glucose, GFR, and LV-EF are presented as median with minimum and maximum ranges.

BMI = body mass index, CK-MB = creatine kinase myocardial band, CMR = cardiac magnetic resonance imaging, GFR = glomerular filtration rate, LAD = left descending coronary artery, LGE = late gadolinium enhancement, LVEF = left ventricle ejection fraction, PCI = percutaneous coronary intervention, RCA = right coronary artery, RCX = circumflex coronary artery, TIMI = thrombolysis in myocardial infarction.

3. Results

3.1. Patient characteristics

Baseline characteristics, cardiovascular risk factors, and pre- and postinterventional results for all patients are found in Table 1. Median period between the index event and CMR examination was 4 days with an interquartile range of 2 to 7. Patients with LGE had a significantly longer period of time elapse between symptom onset and PCI (7.3 vs 4.0 hours, $P = .017$) and between PCI and CMR (5.0 vs 3.0 days, $P = .002$). LV-EF was significantly decreased and laboratory parameters of infarct size (CK-MB and high-sensitivity troponin I) were significantly elevated in patients with LGE (Table 1). There was no significant difference between both groups with respect to the use of antiplatelet therapy and cardiovascular risk factors.

3.2. Angiographic findings

Pre- and postinterventional thrombolysis in myocardial infarction (TIMI) flow, vessel disease, and characteristics of the culprit lesion are presented in Table 1. Anterior infarcts secondary to lesions of the left anterior descending artery (LAD) were more often observed in the group with LGE than in the control group (50% vs 29.4%, $P = .024$). Proximal and medial coronary lesions were also found more often in patients with LGE (39.7% vs 5.9%, $P < .001$ and 38.2% vs 16.2%, $P = .014$). Conversely,

Table 2

Predictors of LGE in conditional and stepwise backward selection conditional logistic regression analysis.

	Logistic regression		Stepwise backward selection	
	Odds ratio (95% CI)	*P*-value	Odds ratio (95% CI)	*P*-value
Δ Intervention-to-CMR, d	0.87 (0.70–1.09)	.236	–	–
Δ Symptom-to-intervention, hours	1.09 (0.98–1.22)	.120	–	–
STEMI vs NSTEMI	2.93 (0.43–20.02)	.274	–	–
LV-EF, %	1.01 (0.95–1.09)	.706	–	–
TIMI-flow pre-PCI = III vs 0–II	0.30 (0.01–9.11)	.487	–	–
TIMI flow post-PCI = 0 vs I–III	0.69 (0.09–5.38)	.724	–	–
Medial culprit lesion	1.70 (0.38–7.62)	.489	–	–
Distal culprit lesion	0 (0.00–0.83)	.044	0.04 (0.01–0.19)	<.001
1&2-vessel disease	14.45 (0.48–437.67)	.125	–	–
3-vessel disease	221.12 (0.17–2.9e+05)	.141	53.99 (8.22–354.63)	<.001
Thrombus aspiration	0.06 (0.00–6.85)	.245	–	–
CK-MB, per 100 U/L	7.30 (3.12–17.08)	<.001	6.18 (2.59–14.73)	<.001
High-sensitivity troponin I	1.00 (1.00–1.000)	.536	–	–
Use of glycoprotein IIb/IIIa inhibitors	460.18 (2.77–76564.08)	.019	–	–

CI = confidence interval, CK-MB = creatine kinase myocardial band; U/L, units per liter, CMR = cardiovascular magnetic resonance imaging, LGE = late gadolinium enhancement, LV-EF = left ventricle ejection fraction, NSTEMI = non-ST-elevation myocardial infarction, PCI = percutaneous coronary intervention, STEMI = ST-elevation myocardial infarction, TIMI = thrombolysis in myocardial infarction.

distal coronary lesions were observed less often in this group (10.3% vs 26.5%, $P = .035$). Furthermore, there was a significantly higher occurrence of preinterventional TIMI-0-flow (58.8% vs 16.2%, $P < .001$) and a lower prevalence of postinterventional TIMI-III-flow (82.4% vs 94.1%, $P = .039$) in the patients with LGE as compared to the control group. The presence of 3-vessel disease (41.2% vs 17.6%, $P = .004$) and the performance of thrombus aspiration during PCI (32.4% vs 5.9%, $P < .001$) were both more common in patients with LGE.

3.3. Predictive determinants of LGE

After conditional regression analysis with stepwise backward selection, the presence of 3-vessel disease (OR 53.99; 95% CI 8.22–354.63, $P < .001$), higher CK-MB values (OR 6.18; 95% CI 2.59–14.73, $P < .001$), and proximal or medial coronary lesions (distal lesion OR 0.04; 95% CI 0.01–0.19, $P < .001$) remained independent predictors of LGE (Table 2).

3.4. Clinical follow-up analysis

Clinical follow-up data were obtained and evaluated for 133 (98%) patients. The combined endpoint occurred in 31 patients. There was a higher rate of events in patients with LGE (38.2% vs 7.4%, $P < .001$). Those patients with LGE had a higher incidence of severe heart failure (LV-EF < 30%) compared to those without LGE (23.5% vs 1.5%, $P < .001$). No significant difference in the incidence of intraventricular thrombus, myocardial reinfarction, stroke, or death was observed between the groups (Table 3).

4. Discussion

Several studies have investigated the clinical impact of LGE on patient outcome after AMI.[7,8] However, few studies have evaluated angiographic factors or laboratory parameters that may be predictive for the development of LGE. Identification of such predictors for the development of LGE post-AMI may facilitate early risk stratification, even before cardiac imaging. Our multivariate analyses revealed that proximal coronary artery lesions, 3-vessel coronary artery disease, and high CK-MB values were independent predictors of LGE. Furthermore, patients with LGE had reduced LV-EF and a higher rate of adverse clinical events, especially severe heart failure, during the 24 months after their AMI. In summary, all of these factors underscore the assumption that LGE is only detected in patients with severe myocardial infarctions with extensive myocardial necrosis.

4.1. Angiographic findings

We identified 3-vessel disease and proximal coronary lesions to be predictors of LGE after AMI. The presence of multivessel disease in AMI has been demonstrated to correlate with impaired survival[13] and is associated with older age, longer ischemia time, and more cardiogenic shock in a study with scintigraphic assessment.[14] These authors observed a significantly higher rate of previous myocardial infarction in patients presenting with multivessel disease. Of note, they also found that multivessel disease had no impact on the measured infarct size. However, a major limitation of the study was the timing of the imaging, which was performed 30 days after the index event. It is well known that remodeling of an infarcted area happens within the first 6 weeks.[15] In our study, which excluded patients with prolonged intervention-to-CMR times, the occurrence of multivessel disease was an independent predictor for LGE after first STEMI. It has previously been shown that infarct size has an impact on outcome after AMI. Our data emphasize the fact that multivessel disease is disadvantageous in AMI. Only a few studies have investigated the relationship between multivessel disease

Table 3

Clinical events of the study population after 24 months.

	LGE present, n = 68	Control group, n = 68	*P*-value
Without clinical event	42 (61.8%)	63 (92.6%)	<.001
Myocardial reinfarction	6 (8.8%)	2 (2.9%)	.289
Heart failure	16 (23.5%)	1 (1.5%)	<.001
Stroke	1 (1.5%)	1 (1.5%)	1.000
Death	1 (1.5%)	1 (1.5%)	1.000

Data is presented as number and percentage of patients.
LGE = late gadolinium enhancement.

and its impact on infarct size. Tarantini et al[16] compared STEMI patients with single vessel disease to patients with multivessel disease. The authors confirmed the data of De Luca et al[14] with respect to a higher prevalence of comorbidities. They also found that infarct size, as assessed by CMR, was not different in patients with multivessel disease. However, multivessel disease was an independent predictor of myocardial reinfarction and ventricular remodeling. Thus, these prior studies focused on infarct size, but in our study we evaluated the presence or the absence of LGE and determined predictors of it.

Similar to our results, previous studies observed that time-to-treatment and pre- and postinterventional TIMI flow correlated with infarct size or microvascular obstruction.[17–20] However, the multivariate analysis of our 136 patients did not confirm time-to-treatment as an independent predictor for LGE. In the study of Thiele et al,[21] where time-to-treatment delay was one of the predictors in STEMI patients, infarct size was categorized by means of extent and transmurality. One explanation for our results might be that we have focused solely on LGE.

4.2. Infarct markers

In our multivariate analysis, another independent predictor of LGE was the peak value of CK-MB. Similarly, Choi et al[8] observed a strong correlation between infarct size and CK-MB values in a study of 24 consecutive patients with AMI.

In our univariate analysis, peak values of high sensitivity troponin I were significantly elevated in the presence of LGE; however, in the multivariate analysis the statistical significance was lost.

Similar results were observed by Nguyen et al.[22] They demonstrated that peak levels of high-sensitivity troponin at 48 and 72 hours post STEMI independently predict large infarct scar size, poor myocardial salvage, and reduced LV-EF. In addition, Klug et al[23] observed a correlation between cardiac troponin at 3 to 4 days, CK value, and infarct size in reperfused STEMI. Another study with 80 patients demonstrated a significant correlation between cardiac troponin and CK and infarct size during the index event and 4 months post-event in successfully reperfused AMI.[24] Our data confirm this correlation between laboratory infarct parameters and infarct size, and we specifically found that peak CK-MB values were strong prognosticators of the occurrence of LGE.

4.3. Clinical events

We demonstrated that patients with LGE had a higher rate of adverse clinical events. Severe heart failure with a LV-EF $<30\%$ was the most common clinical complication. These findings are consistent with previous studies that demonstrated infarct size had an impact on later clinical events. Wu et al[25] also found that the extent of infarct size in STEMI and non-ST-elevated myocardial infarction patients correlated with the occurrence of major clinical events in a relatively small study population. Interestingly, unstable angina with rehospitalization was the most frequent event in their study. Bello et al[7] have observed in a clinical 5-year follow-up that the presence of infarct sizes greater than or equal to 24% of left ventricular mass and reduced LV-EF were predictors of death. Wu et al[26] showed that acute infarct size directly relates to LV remodeling. In a study with 50 patients, right ventricular involvement of a left ventricular AMI as assessed by LGE was associated with a higher rate of major adverse clinical events compared to patients without right ventricular involvement.[27] This highlights the fact that not only the existence, but also the extent of LGE plays a major role in the prognosis of patients with AMI. Nevertheless, there probably is more than 1 factor that negatively impacts patient outcome after AMI. It increasingly seems as though the pathological mechanism is multifactorial, with different key factors affecting the outcome in STEMI patients. As mentioned previously, multivessel disease, with its recurrent ischemic events, is known to influence outcomes.[14,16,28] Also, biomarkers for myocardial infarction and hyperglycemia on admission have been associated with a poor prognosis in AMI.[29] Our data, taken in context with these studies, further expand the concept of multiple pathological factors in AMI and the importance of identifying these factors for early risk stratification.

4.4. Study limitations

A limitation of our analyses is the absence of precise infarct size quantification. However, studies on the interdependency of infarct size and clinical endpoints already exist. We instead wanted to analyze if the sole presence of LGE was determined by different clinical factors. If these factors have an impact on infarct size still needs to be evaluated in future investigations. Including additional standard 2D echocardiography for wall motion abnormalities would have enhanced the study and is a task for future studies. Despite age- and gender matching, year-related bias could not be completely ruled out. Furthermore, we have analyzed determinants of LGE occurrence retrospectively. Prospective studies will need to be conducted to confirm our findings.

5. Conclusion

In conclusion, the presence of myocardium infarction, as visualized by LGE, depends on multiple independent predictors. The presence of a 3-vessel disease, a proximal coronary lesion and high CK-MB values in AMI are strongly associated with the development of LGE. Early risk stratification in AMI depends on the identification of such predictors, which require confirmation and supplemental analysis. Our data contribute to the development of a profile that could be used for early risk stratification in AMI, even before cardiac imaging is performed.

Acknowledgments

The authors thank Lisa C. Costello-Boerrigter, MD, PhD, HELIOS Klinikum Erfurt for support and critical revision of the manuscript. They also thank the staff of the CMR department of the HELIOS Medical Centre Wuppertal.

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