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Relationship between Framingham Risk Score and Left Ventricular Remodeling after Successful Primary Percutaneous Coronary Intervention in Patients with First Myocardial Infarction and Single-Vessel Disease

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Abstract

Background: Limited data is available on the potential value of estimated cardiovascular event risk for prediction of left ventricular (LV) remodeling and size of infarcted tissue after ST-elevation myocardial infarction (STEMI).

Methods: Therefore, we assessed in a consecutive series of patients with first STEMI, successful primary percutaneous coronary intervention (PCI), and single-vessel disease the potential relationship between the Framingham Risk Score and parameters of both LV remodeling and infarct tissue characteristics, as determined with contrast-enhanced (CE) cardiovascular magnetic resonance (CMR) 6 months after the index event. Parameters of LV remodeling were end-diastolic and end-systolic volumes, ejection fraction, and wall motion score index; infarct tissue characteristics comprised core, peri, and total infarct size, and transmural extent.

Results: A total of 25 patients (21 men, 56 ± 10 years) were studied, and the mean Framingham Risk Score was 14.1 ± 5.8%. There was a significant relation between Framingham Risk Score and multiple parameters of LV remodeling: LV ejection fraction, end-diastolic volume, end-systolic volume, and wall motion score index after 6 months (r=-0.55-0.76; p=0.000 for all). Framingham Risk Score showed no relation with various infarct tissue characteristics (ns). Male gender was the only component of the Framingham Risk Score that correlated *individually* with a few parameters of LV remodeling: LV end-diastolic volume and end-systolic volume (p=0.000 for both).

Conclusion: In a series of consecutive patients with first STEMI, successful primary PCI, and single-vessel coronary artery disease, we observed a significant relation between the Framingham Risk Score and several CMR-based parameters of LV remodeling.

The results of our small hypothesis-generating study underline the supremacy of multifactorial risk scores as tools for prediction of unfavorable cardiovascular outcome. Additionally, the data support the hypothesis that there might be a future role for a *novel and specific* multifactorial risk score in predicting unfavorable LV remodeling, which finally could trigger risk-adjusted preventive measures.

Keywords: Framingham Risk Score; Myocardial infarction; LV remodeling; Infarct tissue characteristics; Cardiovascular magnetic resonance imaging

Abbreviations: STEMI: ST Segment Elevated Myocardial Infarction; LV: Left Ventricle; CMR: Cardiovascular Magnetic Resonance; CE: Contrast Enhancement; PCI: Percutaneous Coronary Intervention; MI: Myocardial Infarction; HDL: High Density Lipoprotein

Introduction

Major determinants of poor outcome following ST-elevation myocardial infarction (STEMI) are, left ventricular (LV) remodeling, as well as size, location, transmurality, and heterogeneity of the infarcted tissue as assessed by (histo)pathologic analyses [1,2]. The best strategy to limit LV remodeling and infarct size is a successful early revascularization of the culprit artery [3]. In addition, chronic medical therapy has been shown to reduce the extent of LV remodeling during follow-up [4-6]. Both LV remodeling and infarct tissue characteristics can be assessed with cardiovascular magnetic resonance (CMR) imaging in combination with the contrast enhancement (CE) technique [7-10]. There is no such thing as a "single cause" of LV remodeling, which can even be observed following successful revascularization procedures. In fact, there is growing evidence that multiple factors are involved in the process of LV remodeling [2,11-16], and for that reason there is increasing interest in the impact of various cardiovascular risk factors on LV remodeling [17,18] Identification of patients with a particularly high risk of developing LV remodeling is warranted, as it may represent a first step towards further targeted treatment of patients at risk. It has recently been shown that the Framingham Risk Score, an established cardiovascular event risk score for primary prevention [19], predicts the likelihood of certain adaptive changes in LV structure and function during lifetime within a general population [20]. Therefore,

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we hypothesized that the Framingham Risk Score may also be related to LV remodeling and/or tissue characteristics following STEMI. We used CE-CMR in a consecutive series of patients with first STEMI, successful primary percutaneous coronary intervention (PCI), and single-vessel disease to assess the potential relationship between the Framingham Risk Score and parameters of LV remodeling and infarct tissue characteristics at 6-month follow-up.

Methods

Study population

Patients were included from March 2009 until December 2011. Patients were selected from a larger database of patients (n=36) with a MI in which remodeling after MI was investigated with a 6-months CMR follow-up. Of these 36 patients, 26 had one-vessel disease; one patient was excluded because of blurred CE-images**.** Patients met the following inclusion criteria: (1) STEMI with successful early revascularization within 12 hours after the start of symptoms and TIMI 3 flow at the end of the procedure, defined as complete perfusion (normal flow which fills the distal coronary bed completely), (2) single-vessel disease at coronary angiogram (only patients with single-vessel disease were assessed in order to investigate a homogeneous patient population without potential confounding ischemia that could otherwise have been caused by coronary lesions in non-culprit vessels), (3) complete CE-CMR data available 6 months after STEMI. After PCI, all patients were treated according to the present guidelines with acetylsalicylic clopidogrel, betablocker, ACE-inhibitor, and statin (unless contra-indicated). This study complied with the Declaration of Helsinki for investigation in human beings and was approved by the institutional ethics committee of Medisch Spectrum Twente and the Dutch Central Committee on Research Involving Human Subjects. All patients provided informed written consent for participation in this study.

For all patients, traditional cardiovascular risk factors and laboratory results were recorded at the time of the index MI: sex, age, systolic and diastolic blood pressure, (hypertension defined as systolic pressure >140 mm Hg and/or diastolic pressure >90 mm Hg), total cholesterol level, serum high density lipoprotein (HDL) cholesterol level, serum low density lipoprotein cholesterol level, triglyceride level, current smoking, family history for coronary artery disease (MI of first-degree family member <60 years of age), diabetes mellitus (known diabetes or repeated fasting glucose levels >120 mg/dl), hypercholesterolemia (medication dependent, total serum cholesterol >200 mg/dl, or low density lipoprotein cholesterol >160 mg/dl). Current cardiovascular medication was also recorded.

The cardiovascular event risk was calculated using the Framingham Risk Score. The Framingham score was calculated by use of an algorithm previously described [19]. The score considers sex, age, total cholesterol, HDL cholesterol, systolic blood pressure, and smoking. It was used to predict the 10-year risk of cardiovascular events (fatal/non-fatal MI or sudden death).

CMR examination was performed on a 1.5-T whole body scanner (Achieva Scan, Philips Medical System, Best, The Netherlands) using commercially available cardiac CMR software. For signal-reception a five-element cardiac synergy coil was used. Electrocardiogram triggering was performed with a vector-electrocardiogram set-up. Subjects were examined in the supine position. Cine (morphologic) images in the cardiac short-axis, four-chamber, three-chamber, and two-chamber long axis, and LV outflow tract views were acquired by using fast field echo cine images (slice thickness 8.0mm, repetition time 3.4ms; echo time 1.7ms; flip angle 60°; matrix 256×256). Myocardial scar was assessed on CE multislice short- axis, two-chamber long-axis, and four-chamber views, obtained approximately 10 minutes after intravenous bolus injection of 0.2 mmol gadolinium/kg-1 body weight (Shering AG, Berlin, Germany). A three-dimensional Turbo Field Echo-inversion recovery T1-weighted sequence was used with the following parameters: repetition time 4.0 ms; echo time 1.3 ms; flip angle 15°; inversion time individually optimized to null myocardial signal (usually between 180-250 ms); matrix 157; and slice thickness 10 mm.

CMR data were analyzed on a workstation, using dedicated software for cardiac analysis (Philips MR workspace, Release 2.5.3.0 2007-12-03; Philips, the Netherlands).

LV geometry and function: LV end-diastolic and end-systolic volumes (mL), LV ejection fraction (%), and end-diastolic wall mass (g) were calculated from contiguous short-axis loops by segmentation of endocardial and epicardial borders on each frame. Papillary muscles were regarded as part of the ventricular cavity. The LV wall regions were further divided into 17 segments according to a standardized myocardial segmentation model. Wall motion was assigned the following scores: normal wall motion was 0, mild hypokinesia 1, severe hypokinesia 2, akinesia 3, and dyskinesia 4. The wall motion score index was calculated by dividing the sum of scores in each segment by the total number of observed segments [9].

Infarct tissue characteristics: The infarcted myocardium was defined as the zone of hyper-enhancement on the CE images, in contrast with the dark-gray signal of the normal myocardium. We used a semi-automatic thresholding technique, in which infarct size was approximated by use of the full width at half maximum criteria [21]. After outlining the myocardial segment containing the region with high signal intensity, the maximum signal intensity region was determined. Scar was divided into an infarct core zone and a heterogeneous zone (i.e. peri-infarct zone). Infarct core was then defined as myocardium with a signal intensity > 50% of the maximal signal intensity. The heterogeneous zone was defined as myocardium with a signal intensity ≥ 35% of the maximal signal intensity and < 50% of maximal signal intensity [21]. Total scar was defined as the sum of infarct core plus heterogeneous zone. By use of planimetry, the extent of CE was first determined on contiguous short-axis images, then summed up to a volume, and finally expressed as a percentage of the total myocardial volume.

Scar tissue characteristics were further quantified according to localization by use of a 17 segmental model. Each segment was scored as follows: a scar score of 0 (0% of segmental myocardial volume is scarred) was considered as normal, 1 as 1-25% scar, 2 as 25-50% scar, 3 as 50-75% scar, and 4 as 75-100% scar [22]. The *transmural extent* of myocardial scar was defined as the number of segments with a scar score 3 or 4. See also Figure 1, which represents one of the study patients who fulfilled a complete CE-CMR examination.

Statistical analyses were performed with SPSS 15.0 (SPSS INC., Chicago IL, USA). Dichotomous variables are presented as frequencies and percentages. Quantitative data are presented as mean ± SD. Correlations between the Framingham risk score (or individual parameters of the risk score) and CE-CMR imaging parameters were calculated using Spearman's rho or Pearson correlations. A two-sided p-value <0.05 was considered significant.

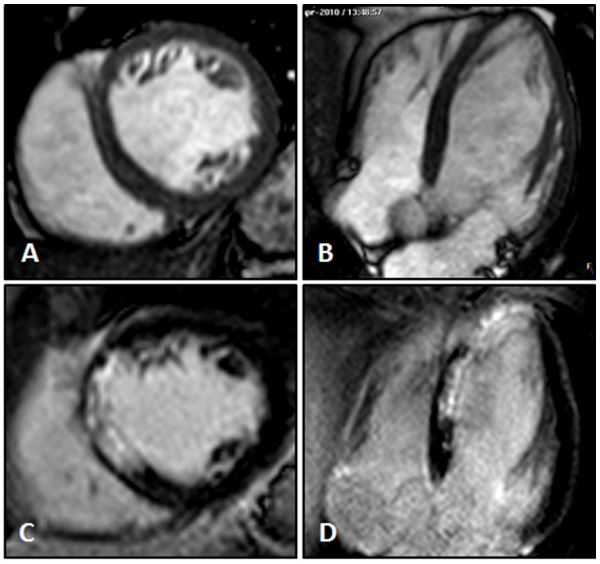

Figure 1: Example of cine- and CE-CMR images of a STEMI patient at 6-month follow-up. The Framingham Risk Score was 17.57%, LVEF 55%, and the total infarct size 12%. A and B: Cine short-axis and four-chamber image; on this images parameters of remodeling are assessed. C and D: Short-axis and four-chamber image with contrast enhancement in the anteroseptal, inferoseptal, and apical region; on this images extent of infarction is assessed.

Results

A total of 25 patients with single-vessel disease (age 56 ± 10 years; 21 men) were examined in this study. All patients demonstrated ST-elevation on the electrocardiogram; none of the patients had electrocardiographic signs of LV hypertrophy. All patients had undergone successful early revascularization by means of primary PCI in 9 left anterior descending, 3 left circumflex, and 13 right coronary arteries as the culprit vessel. Baseline patient characteristics are shown in Table 1. *Data on LV geometry and function after 6 months*: LV ejection fraction was 56 ± 9%, wall motion score index was 0.34 ± 0.26, end-diastolic volume was 195 ± 44mL, and end-systolic volume was 89 ± 39mL. The *infarct tissue characteristics* core, peri, total infarct size, and transmural extent were 5.7 ± 3.8%, 7.2 ± 4.4%, 12.9 ± 7.7%, and 2.1 ± 2.0, respectively.

The Framingham Risk Score was 14.1 ± 5.8%. There was a significant relation between the Framingham risk score versus left ventricular ejection fraction (r=-0.76; p=0.000), end-diastolic volume, (r=0.70; p=0.000), end-systolic volume (r=0.76; p=0.000), and WMSI (r=0.54; p=0.000; Figure 2). Even after excluding a single outlier, there were still significant relationships between the Framingham risk score versus parameters of remodeling (r=0.55-0.66; p=0.000). No significant correlations were observed between the Framingham risk score versus various CE-CMR tissue characteristics. There were significant relations between the Framingham risk score and various parameters of LV remodeling (left ventricular ejection fraction, wall motion score index, end-diastolic volume, and end-systolic volume). When assessing the potential relation between individual risk factors as components of the Framingham risk versus LV remodeling, only male gender was related with a greater extent of LV remodeling (i.e. larger end-diastolic and end-systolic volumes; p=0.000 for both, Table 2). In addition, current smoking showed a significant relation with transmural extent of scar (p=0.04, Table 2).

Discussion

In this relatively small but homogeneous series of consecutive patients with first STEMI, successful primary PCI, and single-vessel coronary artery disease, we observed a significant relation between the Framingham Risk Score and several parameters of LV remodeling as assessed with CE-CMR six months after the event. Of the individual risk factors that form the Framingham Risk Score, only male gender was individually related to some CE-CMR parameters that indicate LV remodeling. This may be explained by the fact that the process of LV remodeling is multifactorial, which is better reflected by a comprehensive risk score than by a single, individual risk factor. Nevertheless, the individual risk factor male gender has contributed to the calculated risk score and to the significant relations observed between the Framingham Risk Score and CE-CMR parameters of LV remodeling following acute MI.

In the present study, many patients suffered from arterial hypertension. Nevertheless, only a minority of them was on antihypertensive drugs as many of these patients had been symptom-free prior to the STEMI and their arterial hypertension had been undetected. During follow-up, all patients were treated according to current guidelines, which included the prescription of a beta-blocker and an ACE inhibitor.

In our study, women had a more favorable course of LV remodeling following STEMI, which could be related to gender differences in levels of sex hormones [23]. Cavasin et al. have demonstrated that estrogen and testosterone play different and opposing roles in the development of

	All patients (n=25)
Male sex	21 (84%)
Age (years)	56 ± 10
Hypertension	10 (40%)
– Systolic blood pressure (mmHg)	137 ± 23
– Diastolic blood pressure (mmHg)	78 ± 13
Diabetes	2 (8%)
Hypercholesterolemia (mmol/l)	4 (16%)
– Total cholesterol	5.6 ± 0.9
– HDL cholesterol	1.4 ± 1.3
– LDL cholesterol	3.6 ± 1.0
– Triglycerides	2.0 ± 1.5
Current smoking	10 (40%)
Positive family history of CAD	16 (64%)
Cardiovascular medication at admission	
– Betablocker	1 (4%)
– Calcium antagonist	1 (4%)
– Ace-/Angiontension inhibitor	0 (0%)
– Statin	2 (8%)
– Diuretic	1 (4%)
Culprit lesion	
– LAD	9 (36%)
– LCX	3 (12%)
– RCA	13 (52%)
Time to revascularization (hours)	3.7 ± 1.5
CK max (U/l)	1026 (176-8074)
LV hypertrophy on ECG	0 (0%)
Infarct age (months)	5.6 ± 0.6
Framingham Risk Score	14.1 ± 5.8

Table 1: Patient baseline characteristics. Continuous data are expressed as mean ± standard deviation or median with range if appropriate; and categorical data as frequencies and percentage. HDL = high density lipoprotein, LDL=low density lipoprotein, CAD = coronary artery disease, LAD = left anterior descending, LCX = left circumflex, RCA = right coronary artery, CK = creatine kinase, LV = left ventricle, ECG = electrocardiogram.

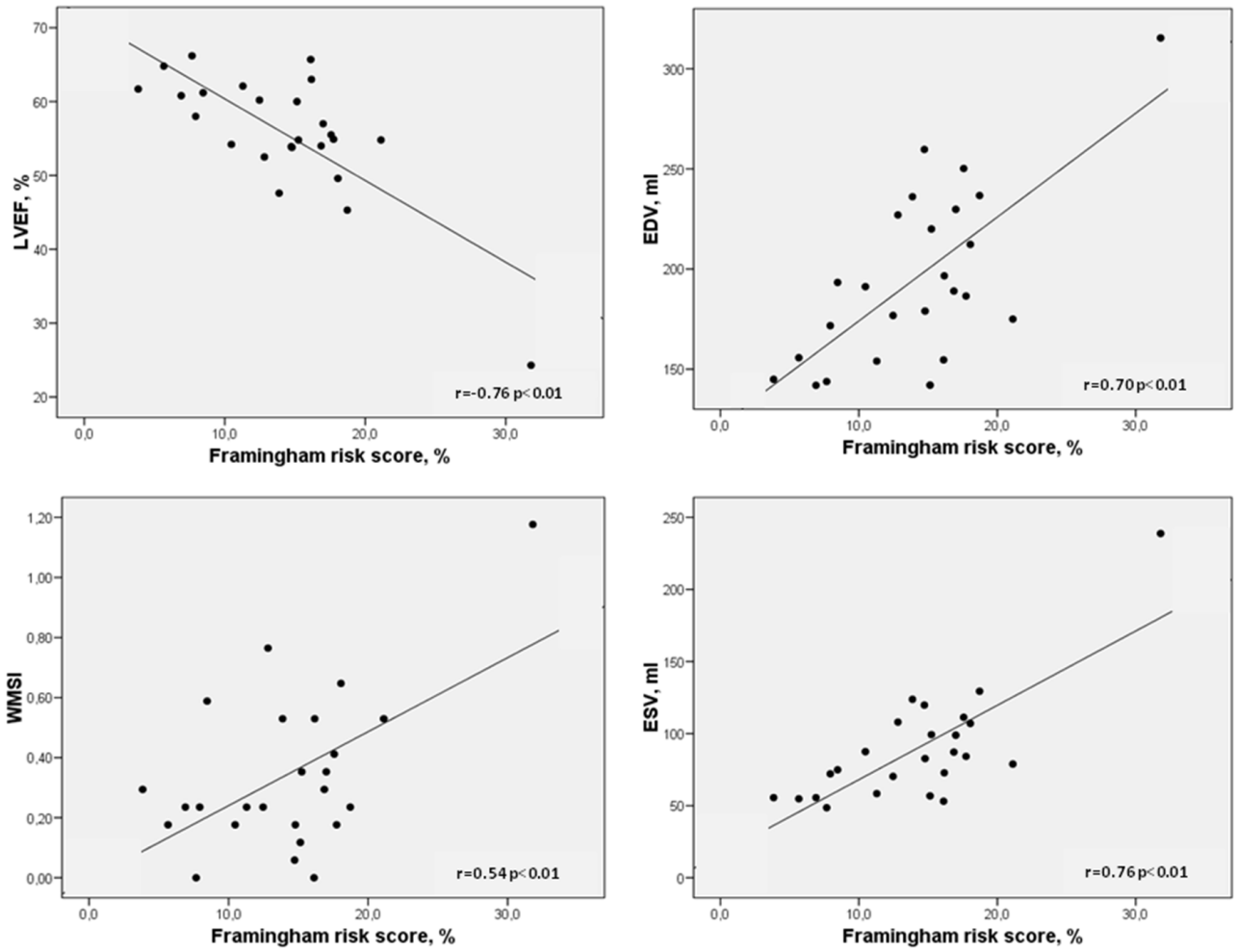

Figure 2: Univariate correlations between parameters of LV remodeling 6 months after STEMI versus the Framingham Risk Score.

Framingham Risk Score parameters	LVEF%	WMSI	EDV,mL	ESV,mL	Infarct size core, %	Infarct size peri, %	Infarct size total, %	Trans-mural extent
Male sex	p=0.13	p=0.27	p=0.000	p=0.000	p=0.28	p=0.48	p=0.34	p=0.26
Age	r=-0.25; p=0.23	r=0.10; p=0.63	r=0.16; p=0.45	r=0.22; p=0.29	r=-0.13; p=0.55	r=-0.11; p=0.60	r=-0.13; p=0.55	r=-0.19; p=0.36
Total cholesterol	r=-0.29; p=0.16	r=0.17; p=0.41	r=0.14; p=0.50	r=0.20; p=0.34	r=-0.13; p=0.54	r=0.16; p=0.45	r=0.16; p=0.45	r=0.26; p=0.20
HDL-cholesterol, mmol/l	r=-0.07; p=0.73	r=0.18; p=0.38	r=-0.11; p=0.60	r=-0.04; p=0.84	r=-0.10; p=0.96	r=-0.02; p=0.92	r=-0.02; p=0.94	r=-0.06; p=0.77
Systolic blood pressure, mmHg Diabetes Mellitus	r=-0.19; p=0.36 p=0.73	r=0.20; p=0.33 p=0.59	r=0.20; p=0.35 p=0.91	r=0.20; p=0.35 p=0.99	r=-0.33; p=0.53 p=0.41	r=-0.22; p=0.30 p=0.37	r=-0.19; p=0.36 p=0.36	r=-0.13; p=0.55 p=0.96
Current smoking	p=0.58	p=0.76	p=0.77	p=0.56	p=0.10	p=0.09	p=0.09	p=0.04
LV hypertrophy on ECG	NA	NA	NA	NA	NA	NA	NA	NA

Table 2: Correlations between individual risk factors versus parameters of LV remodeling and infarct tissue characteristics. HDL=high density lipoprotein, ECG=electrocardiogram, LVEF=left ventricular ejection fraction, WMSI=wall motion score index, EDV=end-diastolic volume, ESV=end-systolic volume., NA=not applicable.

heart failure and long-term LV remodeling following MI. In particular, high testosterone levels enhance acute myocardial inflammation, and adversely affect myocardial healing and early remodeling, which may result in worsening LV function following MI [24,25]. In addition, we cannot completely exclude that gender differences in normal LV end-diastolic and end-systolic volumes (with generally higher LV volumes in men, as was earlier reported by others) [18] may to some extent have exaggerated the outcome of a favorable post-STEMI course of LV dimensions in women.

Other studies also reported relations between cardiovascular risk factors and LV remodeling [17,26-28]. Kenchaiah et al. found hypertension to be associated with subsequent LV dilatation following

acute MI.(26) However, that study investigated only MI patients with LV dysfunction (i.e. ejection fraction ≤40%), whereas in our study of consecutive first MI patients with single-vessel disease the LV ejection fraction was on average preserved, which may explain the difference in findings of both studies. In our study, no relation was found between age and parameters of remodeling. This is in contrast with an echocardiography-based study by Carabba et al., who found a relation with age, as acute MI patient's ≥70 years had a higher prevalence of LV remodeling than younger patients [17].

In the absence of a validated risk score for secondary prevention, established primary-event risk scores have already been used to assess the relations between predicted cardiovascular risk and (1) the extent of plaque progression as assessed with serial intravascular ultrasound [29], and (2) the extent of coronary calcifications in patients with a first MI [30]. Notably, in the latter study by Pohle et al., which also investigated patients with a first MI, the estimated 10-year event risk as calculated by the Framingham Risk Score (14.3 ± 4.4%) was similar to that of our study (14.1 ± 5.8% ((i.e. <10%; low risk, 10-20%; intermediate, and ≥20%; high risk)) [30].

Our present study also assessed potential relationships between the Framingham Risk Score and various infarct tissue characteristics as determined by CE-CMR 6 months after a first STEMI, but we found no such relation. Only smoking as an individual risk factor correlated with a single infarct tissue parameter – the transmural extent of infarction. Similar to our finding, Todt et al. also previously reported a relation between smoking and infarct size [28].

Conflicting results were reported regarding the question of whether individual risk factors other than smoking might affect the size of infarct tissue characteristics. As in the present study, other groups previously demonstrated the absence of a relation between both total cholesterol and arterial hypertension versus infarct size [26,27]. In our study population, there were only two diabetic patients, which actually prevents meaningful analyses of this risk factor, but Donnino et al. recently found no difference in the extent of mycardial scar between diabetics and non-diabetic patients [31]. On the other hand, Mather et al. recently observed with CE-CMR that MI patients with hyperglycemia or diabetes mellitus had larger infarct sizes than normoglycemic patients [18].

In clinical practice, the prediction of unfavorable LV remodeling remains difficult, while there is much interest in this field [2,11-16,32]. The results of our small hypothesis-generating study underline the supremacy of multifactorial risk scores as tools for the prediction of unfavorable cardiovascular outcome. In addition, the data support the hypothesis that there might be a future role for a *novel and specific* multifactorial risk score in predicting unfavorable LV remodeling, which finally could trigger risk-adjusted preventive measures. Nevertheless, such risk sore could only be derived from data of prospective studies with longer-term follow-up of a much larger patient population than assessed in our present study.

Limitations

The study population is relatively small and the findings should be considered as hypothesis generating. Nevertheless, the population is very homogeneous as it consists only of patients with first STEMI, single-vessel disease, and a complete CMR examination including the assessment of CE. In the absence of a generally accepted, validated risk score for secondary prevention, we used the Framingham-algorithm that was initially developed for risk estimation in the context of primary prevention [19] and predicts both fatal and non-fatal adverse cardiovascular events, which we felt to be most appropriate for the assessment of potential relations with LV remodeling. The high number of the relationships examined may have increased the likelihood of finding a statistically significant relation due to chance. On the other hand, significant relationships were already found with a relatively small-sized but homogeneous study population.

Conclusions

In a series of consecutive patients with first STEMI, successful primary PCI, and single-vessel coronary artery disease, we observed a significant relation between the Framingham Risk Score and several parameters of LV remodeling, as assessed by CMR.

Disclosures

CvB is a consultant to and/or has received lecture fees or travel expenses from Abbott Vascular, Boston Scientific, and Medtronic; he has received travel expenses from Biotronik and a speaker's honorarium from MSD. There are not other potential conflicts of interest.

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