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Different Substrates of Non-Sustained Ventricular Tachycardia in Post-infarction Patients With and Without Left Ventricular Dilatation

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ABSTRACT

Background: We investigated the relationship between nonsustained ventricular tachycardia (NSVT) and left ventricular (LV) dilatation, function, remodeling, and scar tissue extent in patients with previous myocardial infarction (MI).

Methods and Results: Eighty-two patients (ages 64 ± 10 years) with first previous MI were referred for 24-hour electrocardiogram recording and cine and delayed enhancement (DE) cardiac magnetic resonance (CMR). LV volumes, ejection fraction, systolic wall thickening, sphericity index, and core and peri-infarctual areas of scar tissue by CMR were evaluated. LV dilatation was observed in 39 patients. Episodes of NSVT were recorded in 32 patients: 23 with LV dilatation and 9 without. In the entire population, NSVT was related to ejection fraction, LV volumes, LV mass, and sphericity index; end-systolic volume ($P = .001$) resulted in the only independent predictor at multivariate analysis. In patients without LV dilatation, the occurrence of NSVT was only positively related with percentage of contracting segments with DE ($P = .008$). Conversely, in patients with LV dilatation, increase in LV mass ($P = .020$) and end-systolic volume ($P = .038$) were independent predictors of NSVT.

Conclusions: Necrotic and viable myocardium coexistence within the same wall segments predicted occurrence of NSVT in patients without LV dilatation, whereas LV mass and end-systolic volume were predictors of NSVT in those with LV dilatation. (*J Cardiac Fail 2010;16:61–68*)

Key Words: Cardiac magnetic resonance, scar tissue, myocardial infarction.

Although the use of an implantable cardioverter defibrillator (ICD) helps to prevent sudden cardiac death in patients at risk of fatal ventricular arrhythmias,[1–6] objective criteria for identifying those patients who would best benefit from ICD implantation are still under investigation. Aside from left ventricular (LV) ejection fraction (EF), the only parameter used in deciding whether to perform ICD implantation, a variety of markers derived from clinical and instrumental data (12-lead electrocardiogram [ECG], 24-h Holter ECG, electrophysiological studies, parameters of cardiac performance obtained with different cardiac imaging techniques, and laboratory assays) has been proposed as predictors of sudden cardiac death.[7,8] Although debated, nonsustained ventricular tachycardia (VT), which is frequent in patients with dilated cardiomyopathy ranging from 30% to 80%,[9] has been identified as an independent predictor of sudden death, as documented in a meta-analysis of 11 peer-reviewed papers enrolling more than 100 patients each.[9–11] In addition, in the Multicenter Unsustained Tachycardia Trial, the presence of nonsustained VT and other variables increased the risk of sudden death in patients with LV ejection fraction >30%, resulting higher than that of patients with ejection fraction <30% but without other risk factors.[12] Other parameters proposed as potential predictors of cardiac arrhythmic

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risk include indices of LV remodeling, such as LV sphericity index, LV mass/LV end-systolic volume, systolic and diastolic LV volumes, parameters of global and regional LV function, and extent of myocardial necrosis.[7,8,13,14] The interactions between nonsustained VT and these variables are not well-defined in patients with postischemic LV dysfunction with and without LV enlargement.

Cardiac magnetic resonance (CMR) accurately quantifies LV volumes and function, and provides unique information on LV remodeling and indices of regional and systolic function, as well as the extent of both myocardial necrosis and peri-infarctual tissue (''gray zone'').[15–17]

Thus the aim of this study was to investigate the interactions between occurrence of episodes of nonsustained VT and CMR parameters of LV remodeling, function and myocardial necrosis extent in patients with previous myocardial infarction (MI).

Methods

Patients

A total of 283 consecutive inpatients with previous MI (longer than 3 months) underwent routine CMR clinical scan to assess scar tissue extent and LV function and volumes. During hospitalization, all patients underwent stress echocardiography or stress scintigraphy. Of these, 201 patients were excluded according to the following exclusion criteria: multiple MIs (n = 90) to avoid the confounding role of multiple scarring in predicting nonsustained VT; documented myocardial ischemia at stress echocardiography or stress scintigraphy (n = 85); amiodarone therapy (n = 22); other concomitant cardiomyopathies (n = 3); and previous cardiac resuscitation, except in the acute phase of the MI (n = 1).

The final population consisted of 82 inpatients (ages 64 ± 10 years, 9 female) with a single previous MI without inducible ischemia at stress test. All patients underwent 24-hour Holter ECG immediately before the CMR exam. The study was approved by the local ethics review committee, and the investigation conformed to the principles outlined in the Declaration of Helsinki. All patients gave their informed consent before the study.

CMR Data Acquisition

The protocol consisted in evaluating by cine CMR the LV volumes, global LV function, quantitative wall motion by measuring systolic wall thickening (SWT), and by delayed enhancement (DE), the core, gray zone, and transmural extent of myocardial necrosis. CMR was performed using a 1.5 T whole-body scanner (GE Medical Systems, Milwaukee, WI). A 4-element cardiac phased-array receiver surface coil was used for signal reception. A breath-hold segmented-gradient echo fast imaging employing steady-state acquisition (FIESTA) ECG-triggered sequence was used to evaluate global LV function according to standard parameters. In each patient, a total of 9 to 12 short-axis views (depending on the LV volume, with a slice thickness of 8 mm and no interslice gap) and 2 long-axis views (1 vertical and 1 horizontal) were acquired, with a minimum of 30 cine frames for each slice. DE images were obtained 8 to 10 minutes after bolus injection of gadobutrol (Gadovist, Schering, Germany; 0.2 mmol/kg); images were acquired in the same short-axis and long-axis slices as used for cine CMR. A fast gradient echo inversion recovery sequence was used with the following parameters: repetition time 4.2 ms, echo time minimum, flip angle 20°, matrix 256 × 192, number of excitations 1.00, field of view 36 to 42 mm, slice thickness 8 mm, and no inter-slice gap. The inversion time was optimized to null signal from the normal myocardium.

CMR Data Analysis

To determine LV function, endocardial borders were manually drawn on all LV short-axis images by means of previously validated software (Mass, MEDIS, The Netherlands).

LV end-systolic volume (LV-ESV) and end-diastolic volumes (LV-EDV) were then calculated and LVEF was derived. According to normal ranges for steady-state free precession sequences,[18] a cutoff of LV-EDV of 112 mL/m^2 was used to identify 2 groups of patients: a group with LV dilatation and a group with no LV dilatation. Diastolic sphericity index was obtained as follows: LV-EDV ([longest LA/2]3 * 4,187) using the longest long axis (LA) obtained from the 2- or 4-chamber views.[19] The ratio EDV/mass and sphericity index at diastole were considered as LV remodeling indexes.[19,20] Three short-axis images corresponding to basal, middle, and distal levels of the LV were used to quantify wall thickness at end-diastole and end-systole for the calculation of SWT in 16 segments of LV (the apex was excluded). A value of SWT greater than 10% for each segment was considered as contracting myocardium.[21]

As previously described,[17] to quantify tissue heterogeneity of DE areas, myocardial segments containing the region of high signal intensity (SI) myocardium were outlined, and the maximum SI within this region was determined. The infarct core extent was then defined as myocardium with SI >50% of the maximal SI. A region of interest (ROI) was then placed by a trained observer in an area free of artifacts and with uniform myocardial suppression of the remote myocardium. The gray zone of infarct periphery was defined as the myocardium with SI > peak remote but <50% of maximal SI of the high SI myocardium (Fig. 1). For each patient, the infarct core and gray zone in each short-axis slice were planimetered, and the global size was expressed as percentage of the entire LV myocardium.

The transmural extent of hyperenhancement was measured by standard techniques.[17] For each segment, the transmural extent of total hyperenhancement was expressed as percentage of total segment area. For each patient, the percentage of segments with transmural extents of hyperenhancement within each quartile (0%–25%; 26%–50%; 51%–75%; or >75%) was determined. Furthermore, the relation between SWT and DE allowed us to classify each segment as follows: contracting segments (SWT >10%) with DE, contracting segments (SWT >10%) without DE, noncontracting segments (SWT <10%) with DE, and noncontracting segments (SWT <10%) without DE (Fig. 2).

These segments represent the combined information about tissue characterization and segmental systolic function of LV myocardium (Fig. 2). Contracting segments with DE (CT-DE) identify a tissue with previous necrosis and preserved contraction; the coronary flow related to these segments is almost certainly preserved. No contracting segments with DE (noCT-DE) identified a tissue with previous necrosis and without contraction: the likelihood of improvement in regional contractility after possible revascularization decreased progressively with the transmural extent of DE.[22]

No contracting segments without DE (noCT-noDE) represent a tissue without necrosis but with depressed segmental systolic function, probably from reduced coronary flow reserve: this tissue can be

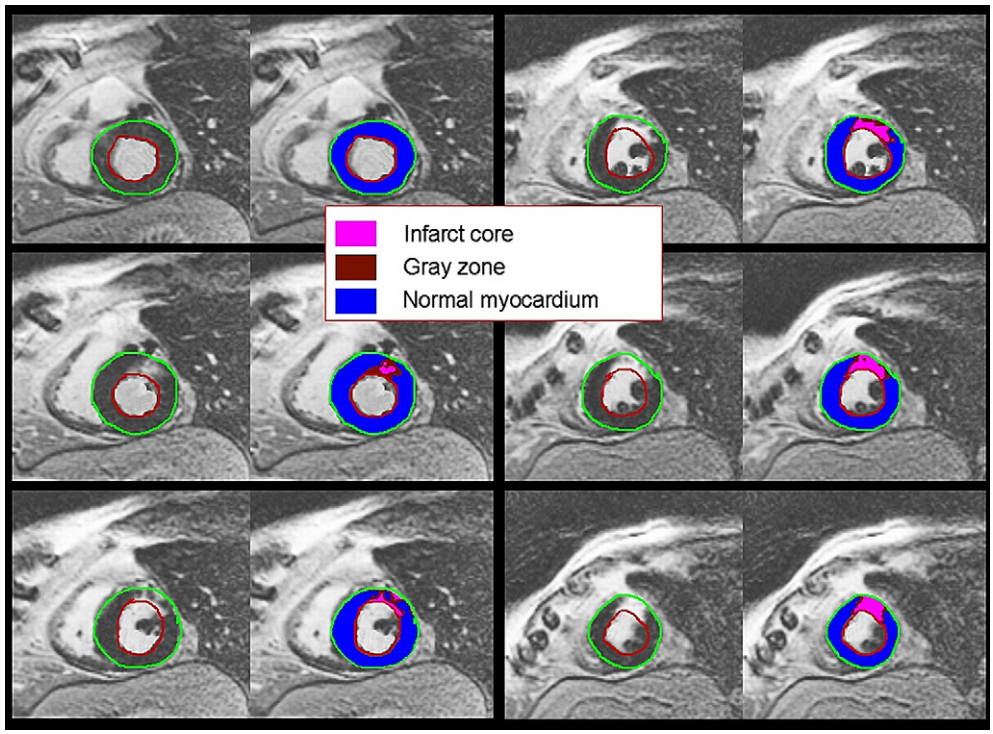

Fig. 1. Gray zone and core measurement. Delayed enhancement short-axis magnetic resonance images from left ventricular (LV) base to LV apex of a patient with an anterior infarct. Computer-assisted, semiautomatic technique for quantifying infarct core and gray zone on delayed-enhancement images. Black area corresponds to infarct core, gray area to peri-scar tissue and white area to normal myocardium.

considered as a hibernating myocardium. Contracting segments without DE (CT-noDE) identify a normal myocardium. All data obtained by CMR exam were blinded to the Holter ECG results.

Holter Monitoring

A 24-hour Holter ECG recording was performed in all patients the day before the CMR examination using a commercial system (Elamedical, Synescope, Cedex, France).

The parameters analyzed were the number of premature ventricular complexes (PVCs) and the presence of episodes of ventricular tachycardia (VT) defined as >3 consecutive ventricular contractions with a ventricular rate >100 beats/min.

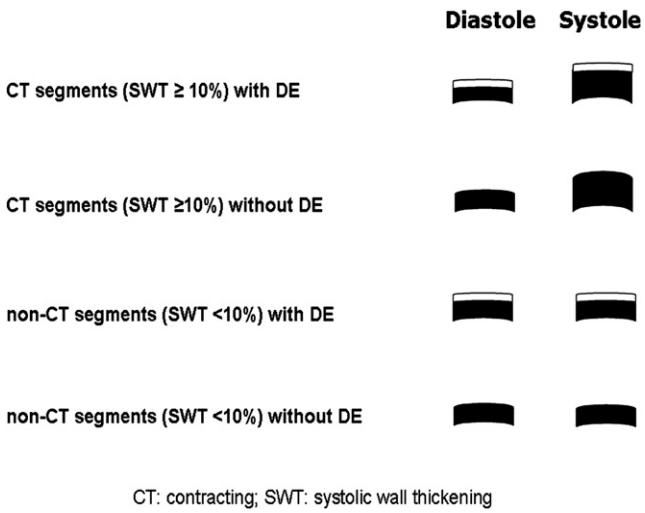

Fig. 2. Illustration of pattern of segments in the relation between systolic wall thickening (SWT) and delayed enhancement (DE).

Statistical Analysis

Continuous variables are expressed as mean ± SD and categorical variables as percentages. Continuous variables with a skewed distribution were logarithmically transformed for further statistical analysis. Unpaired *t*-test was used to assess the differences between the groups with and without LV dilatation or with and without nonsustained VT. The strength of correlation between variables was assessed by Pearson coefficient (R). Continuous variables (age, EDV, ESV, EF, mass/EDV, sphericity index, SWT, infarct core and gray zone, total DE [core + gray zone], contracting segments without DE (CT-noDE), non-contracting segments without DE (noCT-noDE), non-contracting segments with DE (noCT-DE), contracting segments with DE (CT-DE), DE transmural extent 1%-25%, 26%−50%, 51%−75%, 76%−100%), and categorical variables (gender, hypertension, obesity, diabetes, dyslipidemia, smoking, medical treatment, New York Heart Association Class), were entered into a univariate, logistic regression model to identify predictors of nonsustained VT in the entire population and in the subsets with and without LV dilation. Univariate predictors were then entered in a multivariate logistic regression model. A *P* value less than .05 was considered to be statistically significant. All analyses were performed using SPSS for Windows (version 11.00; SPSS Inc, Chicago, IL).

Results

Of the entire population of 82 patients, 43 (52%) had LV dilatation, whereas 39 patients (48%) had preserved LV

Table 1. Baseline Characteristics of the Entire Population and of Patients with and without LV Dilatation

	Entire Population (n = 82)	No LV Dilatation (n = 39)	LV Dilatation (n = 43)	*P* Value
Age (y)	64 ± 10	66 ± 8	62 ± 12	NS
Male (%)	89	82	95	NS
Family history of CAD (%)	48	49	48	NS
Hypertension (%)	58	60	56	NS
Diabetes (%)	30	31	42	NS
Hypercholesterolemia (%)	50	46	56	NS
Smoker (%)	45	44	45	NS
BMI >30 (%)	19	22	17	NS
Q-wave MI (%)	71	80	61	NS
Anterior MI (%)	62	63	60	NS
NYHA >II (%)	16	0	34	<0.0001
Number of stenosed vessels	1.9 ± 0.8	1.8 ± 0.6	2.1 ± 0.8	NS
Syncope (%)	6	3	10	NS
ICD implantation (%)	23	10	33	0.008
β-blocker (%)	76	76	76	NS
ACE-I/ARB (%)	83	81	84	NS
Spironolactone (%)	29	16	42	0.02
Digoxin (%)	13	3	24	0.01
CMR-MI interval (months)	78 ± 77	68 ± 81	85 ± 73	NS

BMI, body mass index; MI, myocardial infarction; NYHA, New York Heart Association; CAD, coronary artery disease; ICD, implantable cardioverter defibrillator, ACE-I, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; CMR, cardiac magnetic resonance.

volumes. The characteristics of the whole population and of the 2 subsets with and without LV dilatation are shown in Table 1. During hospitalization, 19 (23%) patients (5 without and 14 with LV dilatation) received an implantable cardioverter device after the execution of CMR examination, according to Second Multicenter Automated Defibrillator Implantation Trial criteria.

CMR Variables and Arrhythmic Profile at Holter Monitoring

A high percentage (85%) of contracting segments with DE had a transmural extent ≤50%, whereas 72% of noncontracting segments with DE had a >75% transmural extent of DE. During Holter monitoring the number of PVCs (expressed by log10 of PVCs) was significantly more frequent in patients with LV dilatation than in those without LV dilatation (2.91 ± 0.83 vs. 1.82 ± 1.1 $P < .001$). Considering the whole population, a positive correlation was found between the number of PVCs and EDV (R = 0.63, $P < .001$), ESV (R = 0.66, $P < .001$,), and sphericity index (R = 0.50, $P < .001$). A correlation was also found with total extent of DE (core + gray) (R = 0.39, $P = .01$), noncontracting segments with DE (R = 0.44, $P = .001$). Conversely, a negative correlation was observed among number of PVCs and EF (R = −0.58, $P < .001$), SWT (R = −0.54, $P < .001$), mass/EDV (R = −0.55, $P < .001$), and contracting segments without DE (R = −0.27, $P = .02$). In patients with LV dilatation, the number of PVCs was related to EDV (R = 0.51, $P = .001$), ESV (R = 0.60, $P \leq .001$), EF (R = −0.40, $P = .01$), mass/EDV (R = −0.38, $P = .02$), and sphericity index (R = 0.49, $P = .004$). In patients without LV dilatation, EDV (R = 0.33, $P = .04$), and ESV (R = 0.40, $P = .01$) correlated with the number of PVCs.

Holter-ECG monitoring showed no episodes of sustained VT in any patient. Episodes of nonsustained VT occurred in 32 patients: 23 with and 9 without LV dilatation. The incidence of nonsustained VT was significantly different in the 2 subsets. The CMR variables of the patients with and without nonsustained VT are shown in Table 2.

CMR Variables in Dilated and Nondilated LV

Compared with patients with no LV dilatation, those with LV dilatation had a significantly lower EF and mass/EDV ratio and SWT, higher LV mass, sphericity index, and a greater gray zone and total extent of DE than those with normal LV volumes (Table 3). Moreover, patients with LV dilatation had lower percentage of contracting segments without DE and higher percentage of noncontracting segments with DE (Table 3).

Predictors of Nonsustained VT

In the entire population at univariate analysis, EDV, ESV, EF, LV Mass, LV mass/EDV, and sphericity index were predictors of occurrence of nonsustained VT (Table 4). Multivariate analysis selected ESV as the only independent predictor of nonsustained VT (Table 4).

In the subset of patients with LV dilatation, variables able to predict occurrence of nonsustained VT were: EDV (HR 1.024, CI 1.003−1.046, $P = .023$), ESV (HR 1.024, C.I. 1.004−1.044, $P = .016$), and LV mass (HR 1.047, C.I. 1.007−1.088, $P = .012$). Multivariate analysis selected LV mass (HR 1.047, C.I. 1.007−1.088, $P = .020$) and ESV (HR 1.027, C.I. 1.002−1.053, $P = .038$) as independent predictors of nonsustained VT. Conversely, in patients with preserved LV volumes, the percentage of contracting segments with DE (HR 1.116, C.I. 1.029−1.210, $P = .008$) was the only predictor of nonsustained VT at univariate analysis.

Table 2. CMR Variables in Patients with and without Nonsustained VT

	Nonsustained VT Absence (n = 50)	Nonsustained VT Presence (n = 32)	*P* Value
LV-EDV (mL/m^2)	116 ± 39	163 ± 54	<.0001
LV-ESV(mL/m^2)	78 ± 40	129 ± 45	<.0001
LV-EF (%)	36 ± 13	26 ± 12	.001
LV mass (gr/m^2)	82 ± 21	97 ± 27	.02
LV mass/EDV (gr/ml)	0.78 ± 0.3	0,62 ± 0,2	.02
Sphericity index	0.54 ± 0.1	0,66 ± 0,2	.003
SWT	30 ± 19	23 ± 17	NS
Extent of DE			
Infarct core	16 ± 8	15 ± 10	NS
Gray zone (%)	14 ± 8	14 ± 10	NS
Total (core + gray) (%)	28 ± 13	30 ± 16	NS
Transmural infarct extent: % of segments grouped by quartiles of transmurality			
No infarct	48 ± 20	48 ± 18	NS
1% to 25% infarct transmurality	6.4 ± 13	7.7 ± 9	NS
26% to 50% infarct transmurality	5.2 ± 6	6 ± 6	NS
51% to 75% infarct transmurality	19 ± 13	20 ± 13	NS
76% to 100% infarct transmurality	21 ± 18	188 ± 18	NS
CT-noDE (%)	39 ± 24	33 ± 20	NS
noCT-noDE (%)	10 ± 13	17 ± 17	NS
noCT-DE (%)	27 ± 17	29 ± 17	NS
CT-DE (%)	19 ± 17	21 ± 16	NS

LV, left ventricular; EDV, end-diastolic volume; ESV, end-systolic volume; EF, ejection fraction; SWT, systolic wall thickening; DE, delayed contrast enhancement; CT-noDE, contracting segments without DE; noCT-noDE, no-contracting segments without DE; noCT-DE, noncontracting segments with DE; CT-DE, contracting segments with DE; VT, ventricular tachycardia.

Discussion

This study shows that the occurrence of nonsustained VT is influenced by different factors in post-MI patients according to the presence or absence of LV dilatation. In particular, an index of LV remodeling as the ESV is the only independent predictor of nonsustained VT in the entire population; ESV and LV mass are predictor of NSVT in patients with LV dilatation, whereas an index of mixed myocardial necrosis/viability—derived from the combined analysis of DE with SWT—is a predictor of nonsustained VT in post-MI patients without LV dilatation. These results suggest that the substrate for LV arrhythmias may differ in post-MI patients: on 1 hand, LV geometry, more than mere contractility (as expressed by the EF), is the main determinant of ventricular arrhythmias in patients with LV dilatation; on the other hand, the coexistence of necrotic and viable contracting tissue within the same segment proves to be the major trigger of ventricular irritability in nondilated LV patients.

Postischemic LV Remodeling and Arrhythmogenic Risk

Our findings are consistent with the evidence that postischemic LV dilatation and remodeling are critical risk determinants of ventricular arrhythmias.[8,23] Sutton et al showed a relation between LV dimensions and LV mass with ventricular arrhythmias, both early and late (up to 2 years) after MI.[8] Moreover, LV dilatation, but not dysfunction, was found to be related to signs of electrical instability, suggesting that LV remodeling might act as a common determinant of electrical instability and sudden death.[24] Furthermore, cardiac remodeling proved to be linked with other factors predisposing to arrhythmias, including increased sympathetic activity and myocardial adaptive phenomena, such as hypertrophy, apoptosis, myosin isoform change, and alterations in the cellular matrix.[25,26]

Myocardial Necrosis and Arrhythmogenic Risk

Previous studies have shown that scar tissue is an important predictor of ventricular arrhythmias in both ischemic and nonischemic cardiomyopathies.[27–33] The relation between occurrence of nonsustained VT and the percentage of contracting segments with DE—an index of coexistence of necrosis and viability within the same myocardial segment—suggests that this particular tissue may be a substrate for arrhythmogenesis, due to the coexistence of low-voltage dense fibrosis areas, eliciting conduction block, and viable myocardium, producing slow-conduction path circuits and thus promoting intramural re-entry.[34–36]

More recently, the extent of peri-infarct or gray zone at DE-CMR has acquired an important pathophysiological significance. The gray zone is strongly associated with ventricular irritability by programmed electrical stimulation during electrophysiological study.[17] In addition, the gray zone may have a prognostic role in predicting cardiovascular mortality in patients with previous MI.[16] In our study, gray zone and total extent of DE were significantly higher in patients with dilated LV volumes. The gray zone did not correlate to PVCs, nor to nonsustained VT. This discrepancy could be attributed to the different ''end points'' considered (ie, spontaneous nonsustained VT vs. induced monomorphic ventricular tachycardia during electrophysiological study), but also to

Table 3. CMR Variables and Incidence of Nonsustained VT in Entire Population and in Patients with and without LV Dilatation

	All Patients (n = 82)	No LV Dilatation (n = 39)	LV Dilatation (n = 43)	*P* Value*
LV-EDV (mL/m^2)	134 ± 50	92 ± 17	173 ± 38	<.001
LV-ESV(mL/m^2)	97 ± 52	54 ± 16	136 ± 42	<.001
LVEF (%)	32 ± 14	41 ± 11	23 ± 10	<.001
LV mass (gr/m^2)	84 ± 24	78 ± 22	98 ± 22	<.001
LV mass/EDV	0.7 ± 0.3	0.9 ± 0.3	0.6 ± 0.1	<.001
Sphericity index	0.59 ± 0.16	0.49 ± 0.14	0.67 ± 0.14	<.001
SWT	28 ± 19	38 ± 17	16 ± 13	<0.0001
Extent of DE				
Infarct core (%)	15 ± 9	14 ± 7	17 ± 9	NS
Gray zone (%)	14 ± 9	12 ± 7	16 ± 11	.03
Total (core + gray) (%)	29 ± 14	25 ± 111	34 ± 17	.01
Transmural infarct extent: % of segments grouped by quartiles of transmurality				
No infarct	48 ± 19	50 ± 22	46 ± 16	NS
1% to 25% infarct transmurality	6.9 ± 12	6.4 ± 13	7.8 ± 10	NS
26% to 50% infarct transmurality	5.4 ± 6	5.2 ± 6	6 ± 6	NS
51% to 75% infarct transmurality	19.5 ± 13	19.3 ± 12.7	19.9 ± 13	NS
76% to 100% infarct transmurality	20.6 ± 18	21.8 ± 17	25.8 ± 21	NS
CT-noDE (%)	37 ± 23	46 ± 23	30 ± 20	.002
noCT-noDE (%)	13 ± 15	9 ± 11	16 ± 17	NS
noCT-DE (%)	28 ± 17	19 ± 12	35 ± 17	<.001
CT-DE (%)	20 ± 16	21 ± 16	19 ± 17	NS
Nonsustained VT (%)	39	23	53	.004

LV, left ventricular; EDV, end-diastolic volume; ESV, end-systolic volume; EF, ejection fraction; SWT, systolic wall thickening; DE, delayed contrast enhancement; CT-noDE, contracting segments without DE; noCT-noDE, no-contracting segments without DE; noCT-DE, no-contracting segments with DE; CT-DE, contracting segments with DE; VT, ventricular tachycardia.

*Referring to comparison between the two subsets of patients with or without LV dilatation.

differences in patient characteristics (all patients enrolled in the study by Schmidt et al had clinical indications of ICD vs only 36% of our population).[17]

Clinical Implications

Patients with postischemic LV dysfunction and nonsustained VT represent a worrisome group at higher risk of sudden death.[37–39] The observation that a combination of subendocardial necrosis and preserved contractility is a predictor of arrhythmic risk only in patients without LV enlargement—but not in dilated LV patients with LV dysfunction—suggests that the weight of each variable in predicting arrhythmic risk may be related to morphological and functional LV status. Moreover, in the subset of patients with LV dilatation and dysfunction, indexes of geometrical remodeling (namely the ESV) seem to be more effective in identifying patients at higher arrhythmogenic risk when compared to indexes of contractility commonly employed in clinical guidelines (ie, EF). This hypothesis implies that any arrhythmic risk stratification algorithm may be effective in selected patients with LV dysfunction having similar characteristics of LV morphology and function. Therefore, in a large heterogeneous group of patients with postischemic LV dysfunction and nonsustained VT a multimarker strategy might be necessary to better stratify patients at high arrhythmic risk.

Limitations of the Study

The small number of patients and events (nonsustained VT) are this study's main limitations. However, LV function and morphology were assessed by CMR, a noninvasive and nonionizing technique considered the gold standard approach to assessing volumes and regional and global function of LV. The high quality of imaging and the 3-dimensional approach of CMR allows assessment of LV postischemic remodeling accurately and with high

Table 4. Predictor of Nonsustained Ventricular Tachycardia in the Entire Population (n = 82)

	Univariate Analysis		Multivariate Analysis	
	HR (95% CI)	*P* Value	HR (95% CI)	*P* Value
EDV(mL/m^2)	1.021 (1.010–1.033)	.001	—	
ESV(mL/m^2)	1.022 (1.011–1.033)	.008	1.028 (1.012–1.044)	.001
EF (%)	0.937 (0.901–0.975)	.025		
LV Mass (gr/m^2)	1.026 (1.004–1.048)	.021		
LV mass/EDV	0.083 (0.009–0.784)	.030		
Sphericity index	1.053 (1.014–1.093)	.007		

EDV, end-diastolic volume; ESV, end-systolic volume; EF, ejection fraction.

reproducibility, enabling the smaller sample size to reach statistical significance.[40,41]

It is uncertain whether nonsustained VT can be considered a true or surrogate marker of sudden death in patients with postischemic LV dysfunction because there is no clear evidence of its prognostic potential.[39–44] Although it assumes increasing importance when combined with evidence of myocardial dysfunction, becoming a better prognostic predictor of cardiac death than electrophysiological testing,[45] the status of nonsustained VT is often not known, and determining its presence or absence is not straightforward, because its detection depends on the frequency of nonsustained VT episodes and the duration of monitoring. Moreover, detection during prolonged in-hospital monitoring is more likely but may be associated with a different prognosis.[46]

Conclusion

The results of this study show that morphological and functional variables have different weight in predicting nonsustained VT in patients with previous MI; LV remodeling indices are important in the general population and in patients with dilated LV, whereas regional systolic function with mixed necrotic and viable tissue in patients without LV dilatation. In this context, CMR may be an appropriate noninvasive and nonionizing imaging technique for accurately assessing, in a ''one-shop stop'' modality, morphology, function and necrosis extent in post-MI patients. Further studies are needed to define this hypothesis.

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