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