Magnetic resonance estimates of the extent and heterogeneity of scar tissue in ICD patients with ischemic cardiomyopathy predict ventricular arrhythmia

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Abstract

BACKGROUND—The majority of patients receiving implantable cardioverter-defibrillator (ICD) implantation under current guidelines never develop sustained ventricular arrhythmia; therefore, better markers of risk for sustained ventricular tachycardia and/or ventricular fibrillation are needed.

OBJECTIVE—The purpose of this study was to identify cardiac magnetic resonance arrhythmic risk predictors of ischemic cardiomyopathy before ICD implantation.

METHODS—Forty-three subjects (mean age, 64.5 ± 11.9 years) with previous myocardial infarction who were referred for ICD implantation were evaluated by cardiac magnetic resonance imaging (MRI). The MRI protocol included left ventricular functional parameter assessment using steady-state free precession and late gadolinium enhancement MRI using inversion recovery fast gradient echo. Left ventricular functional parameters were measured using cardiac magnetic resonance software. Subjects were followed up for 6–46 months, and the events of appropriate ICD treatments (shocks and antitachycardia pacing) were recorded.

RESULTS—Twenty-eight patients experienced 46 spontaneous episodes during a median follow-up duration of 30 months. The total myocardial infarct (MI) size (18.05 ± 11.44 g vs 38.83 ± 19.87 g; $P = .0006$), MI core (11.63 ± 7.14 g vs 24.12 ± 12.73 g; $P = .0002$), and infarct gray zone (6.43 ± 4.64 g vs 14.71 ± 7.65 g; $P = .0004$) were significantly larger in subjects who received appropriate ICD therapy than in those who did not experience an episode of ventricular tachycardia and/or ventricular fibrillation. Multivariate regression analyses for the infarct gray zone and MI core adjusted for New York Heart Association class, diabetes, and etiology (primary or secondary prevention) revealed that the gray zone and MI core were predictors of appropriate ICD therapies ($P = .0018$ and $P = .007$, respectively).

CONCLUSION—The extent of MI scar may predict which patients would benefit most from ICD implantation.

Keywords

Implantable cardioverter-defibrillator; Cardiac magnetic resonance imaging; Ventricular tachycardia; Ventricular fibrillation; Gray zone; Infarct core

Introduction

Myocardial scarring caused by myocardial infarction is a substrate for ventricular arrhythmias and sudden cardiac arrest (SCA) ascribed to these arrhythmias.¹ Implantable cardioverter-defibrillator (ICD) implantation is now an established cost-effective therapy that improves prognosis in patients with a history of life-threatening ventricular arrhythmia.² A wealth of data support the prophylactic use of ICDs for primary prevention in patients, with known myocardial scarring and significant deterioration in left ventricular (LV) function, who had never before experienced a documented clinical episode. However, only a fraction of ICD recipients under current guidelines for both primary and secondary prevention develop sustained ventricular arrhythmia and thus require ICD therapy. In contrast, many patients who are not candidates for ICD therapy under current guidelines are at risk for sustained ventricular arrhythmia.^{3,4}

The established anatomical substrate for sustained ventricular arrhythmia in the post-infarction heart consists of an area of slow electrical conduction within or around areas of dense scar. This viable conducting tissue is frequently located near the border zone of the dense scar or as islets and channels interrupting the scar.⁵ Large areas of homogeneous scar are, in theory, less likely to produce ventricular tachycardia (VT) than an irregular heterogeneous scar that contains islets of isolated scar. This heterogeneity provides the substrate for a reentry circuit and a milieu for ventricular arrhythmias and subsequent SCA.⁶

Contrast-enhanced magnetic resonance (MR) evaluation of myocardial viability is an excellent technique to assess scar size accurately in humans in vivo.^{7,8} It uses an inversion recovery gradient echo (IR-GRE) sequence and has adequate spatial resolution to identify detailed scar characteristics that may predict VT/ventricular fibrillation (VF). These include the percentage of wall thickness involving the scar (transmurality),^{9–11} an irregular border,⁹ the presence of isolated islets of scar, variations in the density of scar tissue, heterogeneous tissue at the periphery of areas of fibrosis,^{6,12} likelihood of sustained monomorphic VT,^{13,14} and the extent of myocardial scar.¹⁵

Several studies evaluated myocardial infarct (MI) scar characteristics by magnetic resonance imaging (MRI) as well as their ability to predict the occurrence of ventricular arrhythmias.^{15–18} However, in order to accurately predict VT after MI, cardiac MRI image analysis has to be standardized. To date, there is no such approach for mapping the infarct area and distinguishing between the infarct core and the infarct gray zone. The present study was aimed at identifying cardiac magnetic resonance (CMR) arrhythmic risk predictors of ischemic cardiomyopathy before ICD implantation.

Methods

Patient population

Forty-three patients with previous myocardial infarction referred to our institution for ICD implantation for primary or secondary prevention were enrolled into the study between February 2008 and October 2010. Patients with MR-incompatible implants, such as pacemakers, or intracranial clips and other contraindications for MRI were excluded from the study. The local institutional ethics review board approved the study, and all subjects provided written informed consent. All patients were evaluated by using cardiac MRI including late gadolinium enhancement magnetic resonance imaging (LGE-MRI) before ICD implantation. The patients were followed up in the ICD clinic on a quarterly basis.

MRI protocol

MRI protocols included a precontrast LV functional study using a steady-state free precession (SSFP) sequence and LGE-MRI study using an inversion recovery fast gradient echo (IR-FGRE) pulse sequence postcontrast.¹⁹ All MRI examinations were performed on a Signa HDxt 1.5T system (GE Healthcare, Milwaukee, WI) in the supine position. Electrocardiographic gating and an 8-channel phased-array cardiac coil were used for the study.

Before contrast administration, a short-axis oblique SSFP study covering the whole LV was obtained. The detailed cine SSFP MR parameters were as follows: bandwidth 125 kHz, flip angle 45°, views per segment 16, repetition time/echo time 3.7/1.6 ms, field of view 32 cm, image matrix 256 × 192, and slice thickness 8 mm. Twenty phase-resolved images over the heart cycle were acquired on average through a 12-second breath-hold. IR-FGRE covering the whole LV in short-axis oblique in all subjects with 2- or 4-chamber views in most of subjects were performed 10–20 minutes after a double-dose intravenous bolus injection of gadolinium-diethylenetriaminepentacetate (0.2 mmol/kg of Magnevist, Berlex Inc, Wayne, NJ). For IR-FGRE, the inversion time varied from 200 to 300 ms, depending on the null point of healthy myocardium. The in-plane resolution was around 1.5 × 1.5 mm, and the through-plane resolution was 8 mm for both IR-FGRE and cine SSFP. The detailed MR parameters for IR-FGRE were as follows: repetition time/echo time 6.0/3.0 ms, receiver bandwidth ± 31.5 kHz, flip angle 20°, views per segment 20, and number of excitations 2. The delay time was chosen to yield images at the middle to late diastolic phase. Approximately 20 heartbeats (18-second breath-holds on average) were required to produce a single LGE-MRI image using IR-FGRE.

Image analysis

The measurement of LV functional parameters such as LV ejection fraction, LV end-diastolic volume, LV end-systolic volume, and LV mass at the end-diastolic phase was done using commercial CMR software (CMR42, Circle Imaging, Calgary, Canada).

For the measurement of MI heterogeneity especially the gray zone, the following analysis was performed using customer developed software based on MATLAB (Math-Works, Natick, MA).²⁰ First, on each IR-FGRE image, epicardial and endocardial contours were

manually drawn to isolate pixels within the LV, and then in each slice a region was drawn in healthy myocardium remote from the infarct, for apical slices with full involvement of infarction, the remote region was drawn in a most adjacent slice that contained healthy myocardium. Signal intensity (SI) fluctuation has been minimized through active field shimming during the MRI scan. Careful remote zone selection was performed to avoid the inherent field heterogeneity from adjacent cardiac veins. The mean ($\text{Mean}_{\text{remote}}$), peak ($\text{Peak}_{\text{remote}}$), and standard deviation ($\text{SD}_{\text{remote}}$) of the SIs within the remote region were calculated. To determine the cutoff values for the infarct core and gray zones, a full-width half-maximum (FWHM) approach was used with the following definitions²¹: $\text{SI}_{\text{core}} > 0.5 * \text{Peak}_{\text{infarct}}$ and $\text{Peak}_{\text{remote}} < \text{SI}_{\text{gray_zone}} < 0.5 * \text{Peak}_{\text{infarct}}$, where $\text{Peak}_{\text{infarct}}$ is the peak SI of all pixels in a region manually drawn within the infarct. The gray zone is defined as a region growing around the core infarct with 8 neighbors in the 2-dimensional slice using the FWHM algorithm. Isolated voxels within healthy myocardium with a high signal due to noise might be present and would be classified as the infarct core in the original gray-zone mapping. These isolated voxels were manually removed from these categories and labeled healthy tissue. The total size of the infarct core and gray zone for each patient was expressed in grams of tissue (Figure 1).

ICD follow-up and events

All patients received a single- or dual-chamber ICD or cardiac resynchronization therapy on the basis of the guidelines and the specific need, and it was not influenced by enrollment into the study. Device programming was based on the results of the patient's individual arrhythmia history and defibrillation threshold and was left to the discretion of the implanting physician who was blinded to the study. Most of the programming was standard and included 3 detection rate zones: (1) 150–180 beats/min, (2) 188–220 beats/min, and (3) >220 beats/min.

All patients were followed up in the electrophysiology clinic for 6–46 months with a median follow-up duration of 30 months. Patients were seen at intervals of 3 months, or sooner in cases where device shocks were delivered, and the ICD was interrogated for the relevant ventricular arrhythmic events. Data concerning arrhythmias and device therapy were obtained and stored at the time of device interrogation on each follow-up visit.

The primary outcome measures were appropriate ICD therapy, defined as antitachycardia pacing (ATP) or shock for VT or VF, and any ventricular arrhythmic event, defined as sustained VT and VF.

The incidence and type of arrhythmias that occurred during follow-up (episodes) and the incidence of appropriate defibrillator therapies were determined by reviewing stored intracardiac electrograms. These VT and VF episodes were differentiated on the basis of an electrophysiological review of the intracardiac electrograms with an assessment of atrial-ventricular dissociation, onset characteristics, morphology, stability, and response to therapy. ATP and shocks were considered to be appropriate if the triggering rhythm was determined to be VF or VT. Appropriate ICD therapy in this analysis refers to ICD therapy that was triggered for a single rhythm event, regardless of the total number of actual shocks that were required to satisfy the criteria for the termination of tachycardia.

Statistical analysis

Continuous variables are expressed as mean $\pm$ SD. The Student *t* test was used to compare group differences in continuous variables according to appropriate ICD therapy. For noncontinuous variables, the Fisher exact test was used. Multivariable linear regression using the Cox proportional hazards model was used to identify the MRI indices that were independently associated with the occurrence of VT or VF. Candidate covariates were chosen according to their *P* values $< .15$ in univariate analysis. Then, a model was derived using a backward stepdown selection. All analyses were 2-sided, and $P < .05$ was considered statistically significant. The data were analyzed using the SAS version 9.1 for Windows (SAS Institute Inc, Cary, NC).

Results

Patient characteristics

The demographic and baseline characteristics are presented in Table 1. The mean age of the study population was 64.5 ± 11.9 years (range 40–83 years), and 39 subjects (91%) were men. All subjects had a history of myocardial infarction; of these, 21 subjects (49%) had their ICD as primary prevention. The subjects' demographic and clinical characteristics were comparable between the group of patients who experienced arrhythmic episodes and the group who did not (Table 1). A significantly higher proportion of patients in the group who had 1 or more episodes had diabetes and received antiarrhythmic medication (digoxin or calcium channel blockers) compared with the patient group who did not have any episodes ($P = .042$ and $P = .010$, respectively; Table 1). All patients completed the MRI protocol before ICD implantation.

CMR findings

The image quality was acceptable for analysis in all 43 patients. Scar tissues were detected in all patients, and the transmural scar was greater than 75% of the thickness of infarct segments. There were no differences in the myocardial segmental distribution of the scar between the group who experienced episodes and the group who did not have any episodes. The average MI mass was 31.58 ± 19.94 g, of which the MI core constituted $62.57\% \pm 39.71\%$ (19.76 ± 12.54 g) and the gray zone constituted the remaining $37.43\% \pm 24.70\%$ (11.82 ± 7.80 g). No statistically significant differences in scar characteristics were observed in patients with ICD for primary prevention compared with patients with secondary prevention (data not shown).

Follow-up and incidence of appropriate ICD therapy

Twenty-eight patients (65.12%) experienced at least 1 appropriate ICD therapy (ATP and/or shock) during a median follow-up duration of 30 months (range 6–46 months). Of these, 14 subjects (32.56%) had 1 therapy, 11 subjects (25.58%) had 2 therapies, 2 subjects (4.65%) were treated with 3 therapies, and 1 subject (2.33%) was treated with 4 therapies. No cardiac deaths occurred during the study.

Predictors of appropriate ICD therapy

To correlate with the occurrence of VT or VF and consequently with appropriate ICD therapy, the following MRI indices were analyzed: gray zone, MI core, MI total, ejection fraction, end-systolic volume, end-diastolic volume, LV mass, and stroke volume. Specifically, MR measures were compared between the group of patients who experienced arrhythmic episodes and the group who did not (Table 2). The subjects' LV function parameters—LV ejection fraction, LV end-diastolic volume, LV end-systolic volume, and LV mass—were similar between the group of patients who experienced arrhythmic episodes and the group who did not.

All scar characteristics, namely, the total infarct size (18.05 ± 11.44 g vs 38.83 ± 19.87 g; $P = .0006$), infarct core (11.63 ± 7.14 g vs 24.12 ± 12.73 g; $P = .0002$), and infarct gray zone (6.43 ± 4.64 g vs 14.71 ± 7.65 g; $P = .0004$), were significantly higher in patients who had arrhythmic episodes compared with those who did not have arrhythmic episodes. No statistically significant differences in scar characteristics were observed in patients with ICD for primary prevention compared with patients with secondary prevention.

Two separate multivariate regression analyses for the infarct gray zone and MI core adjusted for New York Heart Association class, diabetes, and etiology revealed that the gray zone and MI core were predictors of appropriate ICD therapies ($P = .0018$ for the gray zone and $P = .0007$ for the MI core; Table 3). A Poisson regression model showed that the gray zone mass positively affected the number of appropriate ICD therapies ($P = .003$), that is, each 1-g increase in gray zone mass increases the odds of the incidence of appropriate ICD therapies by 1.246.

Discussion

Several studies have examined the use of MI scar tissue characteristics on MRI to predict the occurrence of VT or VF in patients being referred for ICD. A study of 91 patients with ischemic cardiomyopathy referred for ICD implantation for primary or secondary prevention showed that the total infarct mass and the gray zone were significantly higher in patients with spontaneous ventricular arrhythmia with subsequent ICD therapy.¹⁸ Scott et al¹⁵ showed that the total percent scar was strongly associated with the occurrence of appropriate ICD therapy in 64 patients with ischemic cardiomyopathy referred for ICD. de Haan et al¹⁶ and Gao et al¹⁷ also showed that total scar predicted arrhythmic events and the incidence of appropriate ICD therapy, SCA, or sudden cardiac death. Moreover, a recent meta-analysis of 11 studies comprising 1105 patients showed that ventricular arrhythmias were more common in patients with a greater extent of LV scar.²² In the present study, we show that the gray zone mass and MI core mass are independent predictors of appropriate ICD therapy as surrogate markers for ventricular arrhythmias.

Customized CMR image analysis tools were used in this study for classifying the infarct core, the gray zone, and the healthy myocardium following what are becoming standardized definitions under the FWHM methodology^{6,21} to relate tissue characteristics to the occurrence of ventricular arrhythmias.

The FWHM methodology had been used originally by Schmidt et al⁶ to show that more extensive tissue heterogeneity correlates with increased ventricular irritability by programmed electrical stimulation. Roes et al¹⁸ used the same method to show that infarct tissue heterogeneity on contrast-enhanced MRI is the strongest predictor of spontaneous ventricular arrhythmia with subsequent ICD therapy as surrogate of sudden cardiac death in patients with previous MI. Yan et al¹² showed that the extent of the infarct gray zone characterized by DE-CMR was strongly and independently associated with all-cause mortality and cardiovascular mortality.

In order to accurately predict VT after MI, standardized cardiac MRI image analysis has to be implemented. To date, there is no such approach for mapping the infarct area and distinguishing between the infarct core and the infarct gray zone. Generally, the scar is defined as any region with the SI more than 2 SDs above the mean if a remote normal region or as 50% of the maximum intensity within the infarct (FWHM) as a threshold for a scar. However, suboptimal signal suppression of remote myocardium, image artifacts, and potential interstitial fibrosis may affect the SI of the remote myocardium, and heterogeneous scar tissue may affect the SI of the scar.²³

The infarct gray zone is thought to be composed of areas of fibrosis and preserved myocardium characterized by inherent abnormalities and seemingly identifies myocardium highly susceptible to ventricular arrhythmias.^{6,24} Because of its mixed composition, the infarct gray zone displays an intermediate SI on LGE images that is higher than that of the normal myocardium but lower than that of the infarct core. The separation of the infarct core from the peri-infarct border zone using conventional LGE-MRI images relies on an arbitrary selection of an SI cutoff. This is usually performed manually or semimanually and can lead to interobserver variability.²³ Furthermore, as LGE-MRI images generally have a low signal-to-noise ratio, noise may influence the size of the gray zone. Yan et al¹² defined the gray zone as areas with an SI between 2 and 3 SD above the SI of the remote myocardium, which is normalized as a percentage of total infarct zone. Schmidt et al⁶ defined the core infarct as areas with SI >50% of maximal SI in the hyperenhanced area, and the infarct gray zone as myocardium with SI > peak SI of the remote myocardium but <50% of the maximum SI. Definitions of the core infarct and gray zone given by Roes et al¹⁸ were exclusively based on the maximum SI in the hyperenhanced area (core SI = 50% of maximal SI, gray zone 35% SI, and 50% of maximum SI). A comparison of the ability of these scar assessment methods to predict VT showed that there was no significant difference in the extent of total scar between methods but there was variability in the scar core and gray zone, which indicates that the total infarct size was the best measure for predicting VT after ICD implantation.¹⁶

In the present study, we used the method described by Schmidt et al⁶ to show that the mass of all scar segments was significantly greater in the group of patients who experienced arrhythmias and could be used as independent predictors of appropriate ICD therapies. The method we used may be more stable and/or repeatable compared with the other methods since it bounds the gray zone SIs using both signals in the infarct and remote zones whereas the other 2 approaches use thresholds based only on remote zone or core infarct but not both, leading to a greater instability of the measures.²¹

The substrate for VT/VF is probably anatomical reentry of electrical conduction around isolated areas of scar adjacent to viable tissue with slowed electrical conduction.⁵ Large areas of heterogeneous scar are, in theory, more likely to produce VT than a regular homogeneous scar. This heterogeneity may provide the substrate for reentry circuit and a milieu for ventricular arrhythmias and subsequent SCA.⁶ For that circuit to sustain itself beside the scar, it requires viable tissue and areas of slow conduction, so head can meet tail.²⁵ This concept was shown in a contrast-enhanced MRI 3-dimensional geometrical assessment of scar tissue containing necrotic but still viable tissue, probably with slow conduction that can serve as isthmuses separating islets of necrosis and playing a role in generating and sustaining those reentry circuits.²⁴ Perez-David et al²⁶ went beyond that and compared LGE features in 18 patients with monomorphic VT in which continuous heterogeneous corridors were identified in the scar. Those corridors of diseased but conducting viable myocardium identified by LGE corresponded to the 3-dimensional endocardial voltage mapping data of slow conduction zones, and in half of the patients it corresponded to a critical isthmus for VT.

In the present study, a higher rate of arrhythmic episodes compared with the rate described for similar cohorts in the literature. That might be a result of different factors: first, our cohort consisted of sicker patients compared with similar data in the previous literature, with more than 60% of patients with secondary etiology. Selection bias might have played a role since we targeted admitted patients to give an informed consent; obviously, those patients would have had higher risk factors for future arrhythmic episodes, and this, in our opinion, would have affected the number of episodes but not their distribution between the groups. Second, follow-up in our cohort was much longer than that in previous similar cohorts (up to 4 years in some patients). Third, inclusion of ATP and sustained VT as episodes (not only shocks) has probably contributed to the higher rate of identified episodes as well.

Study limitations

This study lacks pathophysiological and histological correlation with MRI indices, in particular gray zone measurements. Also, the selection of the remote zone might still contribute to some uncertainty in the calculation of the gray zone, although SI fluctuation has been minimized through active field shimming during the MRI scan and careful remote zone selection was performed to avoid the inherent field heterogeneity from adjacent cardiac veins. Reproducibility of the CMR parameters, especially the infarct size and gray zone, was not assessed. In addition, cutoff values based on the optimal sensitivity and specificity of the gray zone as a predictor of future VT/VF were not determined; consequently, the interpretation of the results and their clinical applicability may have been affected.

Conclusion

Absolute and relative amount of MI scar mass might be a useful noninvasive predictor of SCA risk stratification after MI in patients who are candidates for ICD therapy under current guidelines. The findings might provide a rationale for using MRI indices for noninvasive assessment of active arrhythmia prevention. Future studies are warranted to evaluate the use of findings especially in patients with borderline left ventricular ejection fraction.

Acknowledgments

We thank Dr Sharon Furman-Assaf for assisting us in the preparation of the manuscript.

ABBREVIATIONS

ATP	antitachycardia pacing
CMR	cardiac magnetic resonance
ICD	implantable cardioverter-defibrillator
IR-FGRE	inversion recovery fast gradient echo
FWHM	full-width half-maximum
LGE	late gadolinium enhancement
LV	left ventricle/ventricular
MI	myocardial infarct
MRI	magnetic resonance imaging
SCA	sudden cardiac arrest
SI	signal intensity
SSFP	steady-state free precession
VF	ventricular fibrillation
VT	ventricular tachycardia

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CLINICAL PERSPECTIVES

Only a fraction of implantable cardioverter-defibrillator (ICD) recipients under current guidelines for both primary and secondary prevention develop sustained ventricular arrhythmia and thus require ICD therapy; in contrast, many patients who are not candidates for ICD therapy under current guidelines are at risk for sustained ventricular arrhythmia. Therefore, better markers of risk for sustained ventricular tachycardia and/or ventricular fibrillation are needed. The use of late gadolinium enhancement cardiac magnetic resonance imaging (MRI) is an emerging concept for predicting arrhythmic risk. The present study shows that the extent of myocardial infarction (MI) scar and its heterogeneity may be useful noninvasive predictors of risk stratification of sudden cardiac arrest after MI in patients who are candidates for ICD therapy. As a result, it would be easier to identify appropriate patients for ICD, improving clinical decisions as well as cost-effectiveness. The findings of the study might provide a rationale for using MRI indices for noninvasive assessment for active arrhythmia prevention. For accurate prediction of arrhythmia using these MRI indices, a standardized protocol for cardiac MRI image analysis would have to be developed including cutoff values based on the optimal sensitivity and specificity of the gray zone as a predictor of future ventricular tachycardia and/or ventricular fibrillation.

Figure 1.

Short-axis late gadolinium enhancement magnetic resonance images using inversion recovery fast gradient echo for gray zone mapping. Yellow, gray zone; green, myocardial infarct core. **A:** A patient with ischemic heart disease with a small inferior wall myocardial infarct. A gray zone mass of 2.86 g was observed. No ICD therapy for ventricular arrhythmia occurred during the study. **B:** A patient with ischemic heart disease with multiple anteroseptal and lateral wall myocardial infarct. A gray zone mass of 22.09 g. ICD events occurred during the study.

Table 1

Baseline demographic and clinical characteristics of the study population

Characteristic	Total population (N = 43)	No episodes (n = 15)	One or more episodes (n = 28)	P*
Age (y)	64.49 ± 11.95	64.53 ± 14.03	64.46 ± 10.95	.986
Sex				.655
Female	4 (9)	1 (7)	3 (10)	
Male	39 (91)	14 (93)	25 (89)	
ICD indication				.085
Primary prevention	21 (49)	10 (67)	11 (39)	
Secondary prevention	22 (51)	5 (33)	17 (60)	
NYHA functional class				.070
0	7 (17)	5 (36)	2 (7)	
1	13 (31)	5 (36)	8 (29)	
2	13 (31)	2 (14)	11 (39)	
3	9 (21)	2 (14)	7 (25)	
Antiarrhythmic medication	7 (16)	0 (0)	7 (25)	.010
Smoking	22 (51)	9 (60)	13 (46)	.395
Hypertension	28 (65)	9 (60)	19 (68)	.608
Diabetes	10 (23)	1 (7)	9 (32)	.042
Hyperlipidemia	34 (79)	10 (67)	24 (85)	.151
QRS duration	116.81 ± 28.65	120.67 ± 28.08	114.75 ± 28.78	.525
Left bundle branch block	11 (26)	5 (33)	6 (21)	.399

Continuous data are expressed as mean ± SD and categorical data as n (%).

ICD = implantable cardioverter-defibrillator; NYHA = New York Heart Association.

* Pvalue by χ^2 test for categorical values and Student *t* test for continuous values.

Table 2

Comparison of MRI indices between subjects who did not have any episodes and subjects who had 1 or more episodes

MRI index	Total population (N = 43)	No episodes (n = 15)	One or more episodes (n = 28)	P*
Gray zone (g)	11.82 ± 7.80	6.43 ± 4.64	14.71 ± 7.65	.0004
MI core (g)	19.76 ± 12.54	11.63 ± 7.14	24.12 ± 12.73	.0002
MI total (g)	31.58 ± 19.94	18.05 ± 11.44	38.83 ± 19.87	<.0001
Left ventricular mass (g)	119.18 ± 48.55	127.69 ± 58.22	114.62 ± 42.98	.407
Gray zone/left ventricular mass (%)	10.33 ± 6.01	5.84 ± 4.54	12.73 ± 5.33	<.0001
MI core/left ventricular mass (%)	17.38 ± 10.70	10.55 ± 7.03	21.04 ± 10.61	.0014
MI total/left ventricular mass (%)	27.70 ± 16.41	16.38 ± 11.20	33.77 ± 15.64	.0005
Ejection fraction (%)	27.21 ± 10.68	30.89 ± 10.47	25.23 ± 10.44	.098
End-systolic volume (mL)	243.38 ± 94.42	225.10 ± 82.09	253.17 ± 100.44	.359
End-diastolic volume (mL)	183.54 ± 90.44	158.89 ± 75.11	196.74 ± 96.33	.194
Stroke volume (mL)	59.36 ± 17.45	65.52 ± 18.15	56.05 ± 16.45	.090

MI = myocardial infarct; MRI = magnetic resonance imaging.

* Pvalue by Student *t* test.

Table 3

Multivariate analysis of predictors of the incidence of ventricular tachycardia or ventricular fibrillation

MRI Index	Unadjusted			Adjusted		
	Odds ratio	95% Wald confidence limit	P	Odds ratio*	95% Wald confidence limit	P
Gray zone	1.25	1.08, 1.44	.003	2.09	1.14, 3.85	.018
MI core	1.12	1.03, -1.21	.005	1.21	1.05, 1.38	.007

MI = myocardial infarct; MRI = magnetic resonance imaging.

* Adjusted for New York Heart Association class, diabetes, and primary vs secondary prevention.