Clinical Significance of Late Gadolinium Enhancement on Cardiac Magnetic Resonance in Patients With Hypertrophic Cardiomyopathy

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Introduction and objectives. In patients with hypertrophic cardiomyopathy, myocardial fibrosis can be detected by late gadolinium enhancement (LGE) on cardiac magnetic resonance imaging. We investigated the relationships between the extent of LGE, left ventricular morphology and function, and clinical characteristics.

Methods. Both cine and gadolinium-enhanced magnetic resonance imaging were performed in 104 patients with hypertrophic cardiomyopathy.

Results. Fifty patients (48%) showed LGE (range: 1–11 segments). The extent of LGE was positively correlated with maximum left ventricular wall thickness (r=0.53; P<0.001), left ventricular mass (r=0.41; P<0.001), and the number of hypokinetic segments (r=0.51; P<0.001), and inversely correlated with ejection fraction (r=–0.32; P<0.001), the magnitude of the subaortic gradient increase during exercise echocardiography (r=–0.26; P=0.03), and age at diagnosis (r=–0.20; P=0.04). Four of the 5 patients with an ischemic response on exercise echocardiography had ≥3 segments showing LGE (P<0.003). Severe hypertrophy (i.e., ≥30 mm) and nonsustained ventricular tachycardia occurred more frequently as the number of LGE segments increased (P<0.001 and P=0.04, respectively).

Conclusions. Extensive LGE reflects greater disease expression. It is associated with more severe myocardial damage (i.e., a lower ejection fraction and a larger number of hypokinetic segments) and with adverse clinical characteristics (e.g., young age at diagnosis, severe hypertrophy, nonsustained ventricular tachycardia, and an ischemic response on exercise), suggesting that it may be closely linked to prognosis.

Key words: Hypertrophic cardiomyopathy. Magnetic resonance imaging. Prognosis.

Significado clínico del realce tardío de gadolínio con resonancia magnética en pacientes con miocardiopatía hipertrófica

Introducción y objetivos. La fibrosis miocárdica puede ser detectada por la resonancia magnética hipertrofía (MCH) mediante resonancia magnética cardíaca (RM) con realce tardío de gadolínio (RT). Analizamos la relación entre la extensión del RT y la morfología y función del ventrículo izquierdo (VI) y los datos clínicos.

Métodos. Estudiamos con RM a 104 pacientes diagnosticados de MCH. Se obtuvieron secuencias de cine-RM y secuencias de realce tardío.

Resultados. Cincuenta pacientes presentaron RT (48%; rango: 1-11 segmentos). La extensión del RT se correlacionó positivamente con el grosor máximo (r = 0.53; p < 0.001), la masa (r = 0.41; p < 0.001) y el número de segmentos hipocinéticos (r = 0.51; p < 0.001) del ventrículo izquierdo, e inversamente con la fracción de eyeción (r = −0.32; p = 0.001), la capacidad de incrementar el gradiente subaórtico durante la ecocardiografía de ejercicio (r = −0.26; p = 0.023) y la edad en el momento del diagnóstico (r = −0.20; p = 0.04). Cuatro de los 5 pacientes con una respuesta isquémica en la ecocardiografía de ejercicio presentaron ≥3 segmentos con RT (p = 0.003). La hiperтроfия severa (≥30 mm) y la taquicardia ventricular no sostenida (TVNS) se asociaron con la extensión del RT (p < 0.001 y p = 0.04, respectivamente).

Conclusiones. La extensión del RT refleja una mayor expresión de esta enfermedad. Se asocia con un mayor riesgo de muerte súbita y progresión, con un mayor número de segmentos hipocinéticos y con parámetros clínicos adversos (edad más joven, en el momento del diagnóstico, hiperтроfия severa, TVNS y respuesta isquémica al ejercicio), lo que indica que podría vincularse al pronóstico.

Palabras clave: Miocardiopatía hipertrófica. Resonancia magnética. Pronóstico.
Dumont CA et al. Late Enhancement on Hypertrophic Cardiomyopathy

function, symptoms, functional capacity, and clinical risk
extent of LGE and left ventricular (LV) morphology and
LGE in HCM, we analyzed the relationship between the
observed.

In an attempt to ascertain the clinical significance of
LGE in HCM, we analyzed the relationship between the
extent of LGE and left ventricular (LV) morphology and
function, symptoms, functional capacity, and clinical risk
factors for sudden death in a large cohort of patients with
HCM.

METHODS

Patient Population

Over a 26-month period, cardiac magnetic resonance
(CMR) studies were performed in 104 consecutive patients
from our cohort of 360. We systematically ask for CMR
studies in our patients with HCM, excluding patients
with implantable cardioverter-defibrillators (ICDs),
pacemakers, and atrial fibrillation because of difficulties
with implantable cardioverter-defibrillators (ICDs),

Cardiac Magnetic Resonance

Image Acquisition

All CMR images were obtained with a 1.5-T system
(Gyroscan NT; Philips Medical Systems, Best, The
Netherlands) in conjunction with a phased-array body
coil and electrocardiogram gating. Scout images were obtained in 3 orthogonal planes to
determine the exact position and axis of the left ventricle. Cine-MR images of the left ventricle were obtained using a
turbo gradient recalled echo sequence (repetition time
ms/echo time ms, 11/4; field of view (FOV), 400 mm; matrix, 147 × 256; section thickness,
10 mm; 1-mm gap between slices). The cine-MR
sequences were obtained during expiration in the
following planes: a short-axis view of the left ventricle
from base to apex with eight to ten sections, one horizontal
long axis view of the left ventricle, and one vertical long
axis view in two left atrium-left ventricle chambers.

Myocardial tissue tagging gradient-echo echo-planar
sequence (repetition time/echo time/echo-planar factor,
750/16/13 ms; flip angle, 13°; FOV, 400 mm; matrix,
102x256; slice thickness, 10 mm; orthogonal grid,
10 mm) was run during expiratory breath-hold and three
short-axis planes (basal, midventricular, and apical) were
obtained.

Delayed contrast-enhanced images were acquired 10
minutes after the injection of the contrast material
according to a previous report, with an inversion-
recovery T1-weighted sequence (repetition time/echo
time ms, 8/4.5, flip angle 15°. FOV, 400 mm; matrix,
144x256; and section thickness, 10 mm) in 3 short-axis
views taken at the base, midpapillary muscles, and the
 apex; 1 horizontal long-axis view; and 1 vertical long-
axis view. The time of inversion was adjusted for each
patient between 200 and 400 ms to achieve optimal
suppression of normal myocardium.10,11 The CMR studies
were completed on all patients without complications.

Image Analysis
All CMR images were analyzed on a satellite
workstation console with commercial image post-
processing software (EasyVision, version 4.0, Philips
Medical Systems) by 2 radiologists with experience in
cardiac MR imaging (RS and ER), whose joint opinion
was reached by consensus. The CMR studies were read
blinded to the clinical information. The American Heart Association 17-segment model
for the left ventricle12 was used to analyze wall thickness,
contractile function, and delayed enhancement per
segment. Three representative short-axis slices obtained
at the base, mid-ventricle, and apex were divided into 6,
6, and 4 segments, respectively. The true apex (segment
17) was analyzed on the horizontal or vertical long axis
of the left ventricle.

Endocardial and epicardial contours were traced
manually at end-diastole and end-systole on the short-
axis cine sets in order to measure left ventricular volumes,
and calculate the LV mass, stroke volume, the ejection
fraction, and the cardiac output.

Wall motion at rest was visually assessed as a change
in myocardial tagging grip shape with respect to the
original diastolic pattern. Circumferential segment
shortening was judged as normal, hypokinetic, or
dyskinetic in each myocardial segment.

Late gadolinium enhancement was considered present
when the signal intensity of any area within the
myocardium was highly hyperintense and persists in the
same slice after swapping the phase encoding in order
to exclude artifact images. The patterns of LV hypertrophy by CMR were defined,
as asymmetric when a ratio ≥1.3 of septum to free wall
was present and apical when both an apical wall thickness
≥15 mm and a ratio ≥1.3 of maximum left ventricular
short axis thickness at the apical level to the basal level
were present.14

Exercise Echocardiography
Two-dimensional echocardiography using harmonic
imaging was performed in standard parasternal and apical
views, at baseline, peak exercise, and immediately after
exercise.15,16 Peak exercise was defined when signs of
exhaustion, ST depression >2 mm in the absence of chest
pain, significant arrhythmia, severe hypertension (systolic
blood pressure >240 mm Hg or diastolic blood pressure
>110 mm Hg), severe hypotensive response (decrease
>20 mm Hg from baseline), or limiting symptoms were
present. The development of a new regional dysfunction
or worsening from a previous hypokinetic region to
akinesia was considered an ischemic response, as well
as the decrease of LV ejection fraction >5% at the end of exercise.17,18 Differences of ejection fraction and
subaortic gradient between basal, and peak exercise
conditions were correlated with the number of segments
with LGE. Exercise echocardiography assessment was
performed by one investigator (J.P.), who was blinded
to the clinical data.

Statistical Analysis
Data were analyzed using the SPSS software (version
12.0). Patients were classified in four groups on the basis
of number of segments with late-enhancement: 0, 1, 2,
or ≥3. The χ2 for trend was used to test for an association
between these groups and each dichotomous baseline
variable. Continuous variables were expressed as mean
(SD) and associations were tested by linear regression.
A P value less than .05 was considered significant.

RESULTS

Population Characteristics
There were 67 (65%) males and 37 (35%) women. Mean
age at diagnosis was 43 years (12 - 76) and at the
time of CMR imaging was 51 years (16 - 78). Most
patients (98%) were in NYHA functional class I (50%) or
II (48%); 19 (18%) reported exertional chest pain; and
8 (8%) previous syncpe. Maximal left ventricular wall
thickness on echo was 21 (6) mm (≥30 mm in 13 patients)
and there was a subaortic gradient ≥30 mm Hg in 36
patients (35%). Family history of sudden death was
present in 12 patients (11%), non-sustained ventricular
arrhythmia in 12 patients (11%), and abnormal blood pressure
response on exercise test in 31 (30%). Forty-four patients
had no risk factor for sudden death (42%), 42 (41%) had
one, 14 (13%) had two, 3 (3%) had three, and 1 patient
(1%) had four risk factors. Medical treatment used during
follow-up included beta-blockers (65% of the patients),
calcium antagonists (28%), disopyramide (3%),
amiodarone (19%), acenocoumarol (12%), ACE inhibitors
(13%), and small doses of diuretics (20%). During the
follow-up, 4 patients received an ICD (clinical data in
Table 1), 4 alcohol septal ablation, and 1 patient underwent
septal myectomy.

Cardiac Magnetic Resonance
The mean LV wall thickness was 14 (3) mm and the
mean maximal wall thickness was 23 mm (range: 14 to
42 mm). The number of segments hypertrophied per
Asymmetrical hypertrophy was present in 67 patients (65%), symmetrical in 25 (24%), and apical in 12 (11%). Hypertrophy was observed most frequently in anterior and posterior portions of the upper and middle septum.

Late-enhancement was observed in 50 patients (48%), or in 131 segments (8%) out of 1768 segments, ranging from 1 to 11 segments per patient. Late-enhancement occurred most frequently within hypertrophied regions of the interventricular septum. The delayed enhancement patterns observed were: diffuse or confluent patchy mural foci (70%), subendocardial (10%), small foci (14%) and subepicardial (6%) (Figure 1).

Although the majority...
of the hypokinetic segments presented LGE, of the 111 segments only 75 had LGE.

**Extent of Late Enhancement Related to Clinical Data**

Table 2 summarizes the relation of LGE with age, symptoms, exercise test parameters, and risk factors for sudden death. There was no relationship between the extent of LGE and patients age at the time of CMR imaging \((P=0.3)\), but the number of LGE segments was inversely correlated with age at diagnosis \((r=-0.20; \ P=0.04)\). With regard to sudden death risk factors profile, there was a higher proportion of wall thickness \(\geq 30\) mm and non-sustained ventricular tachycardia (NSVT) with increasing the number of LGE segments \((P<0.01\) and \(P=0.04\), respectively). There was no significant differences in therapy and risk factors for coronary artery disease among LGE groups.

Two patients presented an ejection fraction <50% with a non-dilated LV, one with 2, and the other with 3 segments with LGE. The increase in subaortic gradient during exercise echocardiography correlated inversely with the number of LGE segments \((r=-0.26; \ P=0.023)\). An ischemic response on exercise echocardiography was reported in 5 patients, and four of them had \(\geq 3\) segments with LGE \((P=0.003)\). Of the 5 patients with an ischemic response, 3 had angiographically normal coronary arteries and the other two patients did not have angina, or other symptoms indicative of coronary artery disease, and were reluctant to undergo a coronary angiography (one with a septal diffuse patchy LGE pattern and the other with a subepicardial pattern).

Table 3 shows the relation between LGE and other CMR parameters. The extent of LGE correlated positively with the LV maximum wall thickness \((r=0.53; \ P<0.001)\), the LV mass \((r=0.41; \ P<0.001)\), and the number of hypokinetic segments \((r=0.51; \ P<0.001)\), and inversely with the ejection fraction \((r=-0.32; \ P=0.001)\) (Figure 2).

**DISCUSSION**

Myocardial fibrosis plays an important part in both sudden cardiac death and end stage disease as shown by Dumont CA et al. Late Enhancement on Hypertrophic Cardiomyopathy

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**TABLE 2. Relation of Late Gadolinium Enhancement With Symptoms, Exercise Parameters, and Risk Factors for Sudden Death**

<table>
<thead>
<tr>
<th>Number of Segments With Late Enhancement</th>
<th>0 (n=54)</th>
<th>1 (n=19)</th>
<th>2 (n=12)</th>
<th>(\geq 3) (n=19)</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demography</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>51 (16-78)</td>
<td>54 (21-72)</td>
<td>52 (21-76)</td>
<td>47 (22-74)</td>
<td>.3†</td>
</tr>
<tr>
<td>Age at diagnosis, y</td>
<td>46 (13)</td>
<td>41 (14)</td>
<td>44 (17)</td>
<td>37 (15)</td>
<td>.04±</td>
</tr>
<tr>
<td>Male sex</td>
<td>39 (72%)</td>
<td>11 (58%)</td>
<td>8 (67%)</td>
<td>9 (47%)</td>
<td>.07†</td>
</tr>
<tr>
<td>Clinical</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NYHA II</td>
<td>26 (48%)</td>
<td>7 (37%)</td>
<td>6 (50%)</td>
<td>11 (58%)</td>
<td></td>
</tr>
<tr>
<td>NYHA III/IV</td>
<td>1 (2%)</td>
<td>0</td>
<td>0</td>
<td>1 (5%)</td>
<td>.3†</td>
</tr>
<tr>
<td>NYHA III/IV episodes</td>
<td>8 (15%)</td>
<td>1 (5%)</td>
<td>1 (8%)</td>
<td>4 (21%)</td>
<td>.7†</td>
</tr>
<tr>
<td>Atrial fibrillation episodes</td>
<td>7 (13%)</td>
<td>4 (21%)</td>
<td>2 (16%)</td>
<td>0</td>
<td>.2†</td>
</tr>
<tr>
<td>Family history sudden death</td>
<td>5 (9%)</td>
<td>1 (5%)</td>
<td>3 (25%)</td>
<td>3 (16%)</td>
<td>.2†</td>
</tr>
<tr>
<td>Non-sustained VT</td>
<td>7 (14%)</td>
<td>3 (17%)</td>
<td>2 (20%)</td>
<td>7 (37%)</td>
<td>.04†</td>
</tr>
<tr>
<td>Abnormal BP response</td>
<td>19 (40%)</td>
<td>4 (25%)</td>
<td>4 (40%)</td>
<td>4 (25%)</td>
<td>.2†</td>
</tr>
<tr>
<td>Syncope</td>
<td>6 (11%)</td>
<td>0</td>
<td>1 (8%)</td>
<td>1 (5%)</td>
<td>.4†</td>
</tr>
<tr>
<td>Subaortic gradient (\geq 30) mm Hg</td>
<td>19 (35%)</td>
<td>6 (31%)</td>
<td>5 (42%)</td>
<td>6 (32%)</td>
<td>.4†</td>
</tr>
<tr>
<td>Wall thickness (\geq 30) mm</td>
<td>1 (2%)</td>
<td>2 (10%)</td>
<td>3 (25%)</td>
<td>7 (37%)</td>
<td>&lt;0.01†</td>
</tr>
<tr>
<td>Exercise echocardiography</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>METS</td>
<td>10 (3)</td>
<td>11 (2)</td>
<td>9 (3)</td>
<td>10 (3)</td>
<td>.3†</td>
</tr>
<tr>
<td>Ischemic response</td>
<td>0</td>
<td>1 (8%)</td>
<td>0</td>
<td>4 (25%)</td>
<td>.003†</td>
</tr>
<tr>
<td>Gradient difference (\geq 30) mm Hg</td>
<td>41 (18-250)</td>
<td>18 (10-100)</td>
<td>1 (2-20)</td>
<td>12 (6-68)</td>
<td>.023†</td>
</tr>
<tr>
<td>Risk factors for sudden death</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Risk factors (\geq 2)</td>
<td>8 (15%)</td>
<td>1 (5%)</td>
<td>3 (25%)</td>
<td>6 (32%)</td>
<td>.09†</td>
</tr>
</tbody>
</table>

*Data are presented as the mean value (SD) or range, or number (%) of subjects. NYHA indicates New York Association functional class; VT, ventricular tachycardia; BP, blood pressure; gradient difference, post-exercise-basal gradient.

χ² for trend.

Linear regression (the number of segments with LGE was considered as a continuous variable).
necropsy studies, but its role in developing disease has not been established because of the lack of an in vivo quantification technique.11-14,30,31

Late gadolinium enhancement CMR provides a means of quantifying fibrosis in vivo in HCM and gives a new tool in order to better characterize the phenotype of this disease. This study, with the largest sample size of HCM studied with CMR to date, supports the clinical perspective that myocardial fibrosis, detected as LGE, may play an important role in disease expression.

LGE Related to Risk Factors for Sudden Death

Identifying patients at higher risk is an important aspect of the clinical management of HCM, particularly considering that effective preventive therapy is available (ICDs). The need for accurate risk stratification is challenging, taking into account that HCM patients who undergo ICD implantation are younger than patients with coronary artery disease and it is likely that their lifetime risk of serious ICD related complications will be high. LGE may potentially identify a substrate for increased risk for sudden death. However, it is important to highlight that although the extent of LGE may relate to malignant ventricular arrhythmias, the presence of LGE in itself should not be considered as indicative of an adverse prognosis, since LGE is a common finding in HCM (50% in our study) and the overall risk of sudden death in our patient population is low (<1%). Moreover, the absence of LGE probably will not have a high negative predictive value. For example, one patient who received an ICD for primary prevention, presented multiple prolonged runs of NSVT and did not show LGE (Table 1). This may be explained by the fact that image contrast of LGE is created by suppressing normal myocardium and diffuse myocardial involvement can potentially be missed.21,22

On the other hand, the expression of this abnormal substrate is, in turn, influenced by factors such as autonomic tone and myocardial ischemia.9,33 In our study, an ischemic response during exercise echocardiography was not commonly observed, contrary to a previous dobutamine exercise echocardiography study,23 but its presence was linked to greater extent of LGE, which reflects another adverse clinical marker in this group of patients.

TABLE 3. Late Enhancement and Other CMR Parameters

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=54)</td>
<td>(n=19)</td>
<td>(n=12)</td>
<td>(n=19)</td>
</tr>
<tr>
<td>Mean wall thickness</td>
<td>13 (3)</td>
<td>14 (3)</td>
<td>16 (4)</td>
<td>15 (3)</td>
</tr>
<tr>
<td>Maximum wall thickness</td>
<td>20 (14-32)</td>
<td>23 (16-36)</td>
<td>29 (16-42)</td>
<td>28 (18-41)</td>
</tr>
<tr>
<td>Number of hypertrophied segments</td>
<td>4 (1-11)</td>
<td>6 (1-14)</td>
<td>7 (2-15)</td>
<td>8 (3-15)</td>
</tr>
<tr>
<td>LV mass, g</td>
<td>171 (67)</td>
<td>200 (75)</td>
<td>237 (74)</td>
<td>252 (91)</td>
</tr>
<tr>
<td>LA H, mm</td>
<td>45 (9)</td>
<td>44 (9)</td>
<td>44 (9)</td>
<td>46 (6)</td>
</tr>
<tr>
<td>LVED volume, mL</td>
<td>92 (26)</td>
<td>96 (26)</td>
<td>95 (28)</td>
<td>96 (26)</td>
</tr>
<tr>
<td>LVES volume, mL</td>
<td>21 (8)</td>
<td>22 (7)</td>
<td>28 (12)</td>
<td>33 (19)</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>76 (7)</td>
<td>74 (8)</td>
<td>70 (9)</td>
<td>69 (10)</td>
</tr>
<tr>
<td>Pattern of hypertrophy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asymmetric septal hypertrophy</td>
<td>32 (59%)</td>
<td>12 (63%)</td>
<td>8 (67%)</td>
<td>15 (79%)</td>
</tr>
<tr>
<td>Symmetric</td>
<td>20 (37%)</td>
<td>1 (3%)</td>
<td>1 (8%)</td>
<td>3 (16%)</td>
</tr>
<tr>
<td>Apical</td>
<td>2 (4%)</td>
<td>6 (39%)</td>
<td>3 (20%)</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>Presence of hypokinesia</td>
<td>5 (9%)</td>
<td>5 (26%)</td>
<td>5 (41%)</td>
<td>12 (63%)</td>
</tr>
<tr>
<td>No. segments with hypokinesia</td>
<td>0.25 (0-5)</td>
<td>1 (0-5)</td>
<td>1.75 (0-6)</td>
<td>3 (0-8)</td>
</tr>
</tbody>
</table>

*Data are presented as the mean value (SD) or range, or number (%) of subjects. LVED indicates left-ventricular end-diastolic; LVES, left-ventricular end-systolic; LA, left atrium diameter in horizontal long axis view.
†χ² for trend.
‡Linear regression (the number of segments with LGE was considered as a contiguous variable).
§Mean value (95% confidence interval).
Although we did not find any association between the extent of LGE and patient age at the time of CMR scan, LGE extent was associated with an earlier diagnosis of the disease. This may imply that in some patients, extensive myocardial fibrosis does not need time to develop and large amount of fibrosis could be present at a young age. Moreover, the rate of LGE development may be important and extensive LGE at a young age may carry more significance than a similar degree of LGE in an older patient.

LGE Related to Systolic Dysfunction

Previous studies showed that LGE extent is associated with lower ejection fraction and with progressive ventricular dilation. Additional evidence that suggests a relation between the extent of LGE and systolic impairment is our finding of a positive correlation with the number of segments with hypokinesia and inverse correlations with the LV ejection fraction, and the capacity to increase subaortic gradient during exercise. The clinical course of end-stage phase in HCM proved to be variable, unpredictable, and generally unfavourable. Clinical markers that reliably anticipate evolution to systolic dysfunction are difficult to define in an heterogeneous disease like this. However, the extent of LGE with the other clinical features (young age at diagnosis, greater wall thickness, etc) may help us identify a subgroup of patients in which a close follow up may be warranted.

CONCLUSIONS

Late gadolinium enhancement has a great potential to provide new insights in the assessment of patients with HCM. The extent of LGE reflects a greater expression of this disease. It is associated with a more severe myocardial damage (lower ejection fraction and increased number of hypokinetic segments) and adverse clinical parameters (younger age at diagnosis, non-sustained ventricular tachycardia, severe hypertrophy, and ischemic response on exercise echocardiogram), suggesting it may be linked to prognosis. A follow up of this population will help us to evaluate the predictive accuracy of this technique for both sudden cardiac death and the development of systolic dysfunction.

Study Limitations

First, we estimated the extent of late enhancement in a semiquantitative way compared with the measurement of the volume of hyperenhancing lesions evaluated in previous studies. This methodological approach could...
explain the difference between our study and previous with regard to the strength of relationships observed between the extent of LGE and markers of clinical risk. However, this difference and the lower incidence of LGE in our study compared with previous may also be explained by different enrolling criteria and sample size. Choudhury et al studied only 21 HCM patients and Moon et al chose patients with high or low clinical risk factors for sudden death rather than taking a population “consecutively enrolled” as here. Our study confirms that the quantification of the extent of late gadolinium enhancement using the standardized myocardial segmentation model for tomographic imaging of the heart is clinically relevant and easily available for clinical application. Second, although all patients with documented coronary artery disease were excluded from this study, only patients with typical chest pain or symptoms indicative of coronary disease underwent coronary angiography, being possible that some patients with coronary artery disease were included. However, none of the patients with an ischemic response on exercise echocardiography had a LGE pattern indicative of coronary artery disease.

REFERENCES


