Delayed Enhancement on Cardiac Magnetic Resonance and Clinical, Morphological, and Electrocardiographical Features in Hypertrophic Cardiomyopathy

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ABSTRACT

Background: The clinical, morphological, and electrocardiographical relevance of delayed enhancement (DE) in cardiac magnetic resonance (CMR) was studied in patients with hypertrophic cardiomyopathy (HCM).

Methods and Results: A total of 56 patients underwent both gadolinium-enhanced CMR and 12-lead electrocardiogram. The CMR demonstrated DE at the left ventricular (LV) wall in 39 patients. The patients with DE included more cases with dilated phase of HCM, higher New York Heart Association (NYHA) classes and incidence of ventricular tachyarrhythmias (VT), lower LV ejection fraction (LVEF) and mean LV wall thickness (WT), and a larger ratio of maximum to minimum LVWT. The QRS duration was prolonged and the QRS axis deviated toward left with increases in the DE volume ($r = 0.58$ and $r = 0.41$, $P < .01$). Abnormal Q waves were present in 5 patients and the location coincided with the DE segments in 4 patients, but the concordance was not significant. The amplitude of T waves correlated with the ratio of the apex to basal LVWT ($r = 0.38$, $P < .01$) and was more negative in cases with DE at the apex.

Conclusions: In HCM, the DE was associated with higher NYHA classes and prevalence of VT, impaired global LV function and asymmetrical hypertrophy, and conduction disturbance, abnormal Q waves, and giant negative T waves. (*J Cardiac Fail 2009;15:419–427*)

Key Words: Cardiac magnetic resonance, delayed enhancement, hypertrophic cardiomyopathy, electrocardiogram.

Hypertrophic cardiomyopathy (HCM) is a relatively common primary cardiac disease with various morphologic, functional, and clinical features. In HCM patients, a 12-lead electrocardiogram (ECG) demonstrates a variety of abnormalities, including left ventricular (LV) hypertrophy, abnormal Q waves, bundle branch blocks, and giant negative T waves.[1,2] The mechanisms of such ECG abnormalities have been studied using echocardiography and cardiac catheterization, but remain still controversial.

Cardiac magnetic resonance (CMR) is noninvasive, uses no ionizing radiation and has a high spatial resolution. Therefore, the value of CMR is becoming established in the assessment of the cardiac function.[3,4] Recently, delayed gadolinium (Gd) enhancement (DE)-CMR has been shown to be able to detect a small and focal myocardial abnormalities, and it may be useful to diagnose various cardiac diseases.[5,6] The myocardial DE visualized by DE-CMR is a common feature of HCM and spreads in various areas of the left ventricle.[7–13] The DE in HCM may indicate interstitial fibrosis, myocardial disarray, necrosis, and scarring.[14,15] We previously clarified the relationship between the extent of DE and global and local LV dysfunction in HCM patients and also showed the diffuse and extensive spread of DE in the dilated phase of HCM (D-HCM).[16]

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The patients with D-HCM have been reported to have severe heart failure symptoms, a high risk for fatal arrhythmias, and a high mortality rate.[17]

The clinical importance of DE is now being established in various heart diseases, but it is still unclear whether DE is related to morphological and electrical abnormalities, disease progression, and the risk for sudden death in HCM patients. This study analyzed both the 12-lead ECG and CMR in Japanese HCM patients and examined the relevance of DE in the clinical and morphological features and ECG abnormalities.

Methods

Patients

A total of 56 consecutive patients of HCM who underwent CMR between May 2003 and November 2007 were studied. HCM was diagnosed according to the World Health Organization/International Society and Federation of Cardiology definition of cardiomyopathies.[18] The diagnosis of HCM was based on echocardiographic documentation of a hypertrophied nondilated LV in the absence of other cardiac systemic diseases that could produce the magnitude of hypertrophy evident at some time during the natural course of the disease. Therefore, the patient group consisted of 18 patients with asymmetrical septal hypertrophy (ASH), 17 with apical hypertrophy (APH), 10 with concentric hypertrophy (CH), and 11 with D-HCM. Among them, 9 cases were diagnosed as an obstructive type of HCM (HOCM). The D-HCM was defined as an LV ejection fraction below 45% by the cine-mode of CMR, reflecting global systolic dysfunction, at the study entry.[16] These patients were proven to have a period of hypertrophy during the natural time course of the disease. Eleven age- and sex-matched normal controls were also examined to obtain the normal values for LV function. The normal controls consisted of patients with ventricular premature complexes who had no findings of organic heart disease in other examinations. This study protocol was in accordance with the Declaration of Helsinki and approved by the institutional review board (Hamamatsu University School of Medicine, Hamamatsu, Japan) and all patients gave their informed consent.

ECG Analyses

A 12-lead ECG was performed before CMR in all subjects in the supine position. The LV hypertrophy was determined by 3 different ECG criteria (Romhilt-Estes criteria, Sokolow-Lyon voltage criteria, and Cornell voltage-duration criteria). The criteria of abnormal Q wave were 25% of ensuring R wave in depth and 0.04 seconds in duration in at least 2 leads, except for aV_R. The P-R interval, QRS duration, QRS axis, and the maximum amplitude of positive or negative T waves at lateral precordial leads were also analyzed. In addition, all the patients underwent a 24-hour ambulatory Holter ECG. The intervals between the analysis by CMR and Holter ECG were within 3 months. Nonsustained ventricular tachycardia (VT) and supraventricular tachycardia (SVT) were defined as 3 or more consecutive premature complexes with a heart rate of >100 beats/min.

Magnetic Resonance Imaging Protocol

All 56 patients and 11 controls underwent ECG-gated CMR. Imaging was performed on a 1.5 tesla (T) MR system (Signa Infinity Twinspeed, GE Medical Systems, Waukesha, WI) with a gradient system performance of maximum amplitude of 40 mT/m and slew time of 150 T/m/s. An 8-element phased array cardiac coil was used in all studies. Typically, 3 planes such as short axis, sagittal long axis, and 4-chamber view were obtained for 2-dimensional FIESTA cine images and DE images. The 6 to 9 slices were used to cover whole heart and the slice thickness/gap was typically 10 mm/0 mm.

Breath-hold cine magnetic resonance images were obtained in contiguous short-axis planes from the apex to the base of the heart with the patient in a resting state. The 2-dimensional FIESTA cine images were based on the steady state free precession sequence. The imaging parameters were as follows; matrix of 192×192, field of view 34 cm, flip angle 45°, and readout bandwidth 125 kHz. Sixteen data lines were acquired per each segment. The shortest repetition time and echo time were selected; however, the values were not exactly the same for each study, because they were related to the orientation of the scanning plane and slice thickness.

After the 2-dimensional FIESTA cine images were acquired, 0.2 mmol/kg of contrast material (Gd-DTPA-BMA, Fuji Pharma., Tokyo, Japan) was injected and, after a 15-minute delay, DE images were acquired. DE imaging was based on the inversion recovery prepared fast gradient echo (IR-FGRE) sequence. The imaging parameters were as follows: matrix of 256×160, field of view 34 cm, flip angle 20°, and readout bandwidth 31.25 kHz. The IR-FGRE technique repeated during every R-to-R interval and the trigger delay for IR-FGRE was 300 ms; however, for the patients with tachycardia, the system-dependent trigger delay time was selected, which was shorter than 300 ms. The readout data line was 160 each, where 24 data lines were acquired per segment. Before the DE images, pilot DE images were acquired with 5 different inversion times. Therefore, an optimum inversion time was measured right before the DE imaging, which was between 200 ms and 240 ms. The process to identify optimum contrast was concluded within 3 minutes.

Analyses of Magnetic Resonance Images

Two experienced cardiovascular radiologists interpreted all the CMR images without any knowledge of the clinical and ECG findings. LV end-diastolic volume (LVEDV), end-systolic volume (LVESV), ejection fraction (LVEF), and LV mass were acquired from the 2-dimensional FIESTA cine images in the short axis view. The values for LV volume and mass were indexed by dividing them with the body surface area (LV end-diastolic volume indexes [LVEDVI], LV end-systolic volume indexes [LVESVI], and LVMI). Regional analyses of DE-CMR were performed using the 17-segments model. To assess DE quantitatively, all the short-axis slices from base to apex were inspected visually to identify areas of normal (nulled) myocardium. In each image, the boundaries of contrast enhanced areas were manually traced and were accepted as DE areas if the mean signal intensity was at least 3 SD above that of the nulled myocardium. The summed DE area was rendered to DE volume and the percentage against total muscle volume (%DE volume) was calculated.[16]

Statistical Analyses

All the data were expressed as the means ± SD of the indicated numbers (n). Categorical variables were compared between the patient groups by chi-square analyses. Differences between groups were examined by the Mann-Whitney U test. Correlations