Late gadolinium enhancement in cardiovascular magnetic resonance in patients with hypertrophic cardiomyopathy complicated by life-threatening ventricular tachyarrhythmia

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Abstract

Background: Late gadolinium enhancement (LGE) on cardiac magnetic resonance imaging (CMR) has been shown to be associated with ventricular arrhythmias, however, its prognostic role in predicting sudden cardiac death has not yet been established.

Aim: To explore a potential relationship between LGE visualised by CMR and life-threatening ventricular tachyarrhythmia in hypertrophic cardiomyopathy (HCM).

Methods: The LGE in CMR was assessed in 55 HCM patients. We compared the frequency and extent of LGE in HCM patients with sustained ventricular tachycardia (VT) or who survived ventricular fibrillation (VF) or sudden death [group VF (+)] versus HCM patients without these tachyarrhythmias [group VF (–)]. There were 14 patients in the VF (+) group and 41 patients in the VF (–) group, and they were followed for a mean period of 37 months.

Results: In group VF (+), adequate ICD intervention occurred in 9 patients (8 patients with VF and one patient with sustained VT), and VF arrest occurred in 5 patients (4 patients were resuscitated and one patient had a witnessed sudden death). In group VF (+) all patients had LGE whereas in group VF (–) 85% patients presented this abnormality ($p = 0.13$). Moreover, there were no statistical differences between groups in the following parameters: age, total left ventricular (LV) mass, maximal LV wall thickness, mass of hyperenhanced myocardium and percent of hyperenhanced myocardium.

Conclusion: In HCM patients with life-threatening ventricular tachyarrhythmia LGE was both qualitatively and quantitatively comparable with patients without these tachyarrhythmias.

Key words: hypertrophic cardiomyopathy, sudden cardiac death, MRI

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Introduction

Cross-sectional data suggest an association between late hyperenhancement (LGE) and ventricular arrhythmias in hypertrophic cardiomyopathy (HCM) [1-4]. These data, linking LGE to non-sustained ventricular tachycardia (nsVT), and by inference to increased risk of sudden death, provide some preliminary evidence that LGE could ultimately prove to be a new risk marker in HCM. Adabag et al. [3] recently postulated the necessity for longitudinal follow-up studies to clarify whether LGE should be established as an independent risk predictor for sudden death in HCM. In HCM patients, prospective data correlating CMR findings exclusively to sudden death events are not yet available. The current study has focused only on this type of episode.

Methods

Patient population

Diagnosis of HCM was based on echocardiographic documentation of a hypertrophied and non-dilated LV in the absence of another cardiac or systemic disease capable of producing a similar magnitude of hypertrophy.

Our strategy was to have a sufficient number of episodes of life-threatening ventricular tachyarrhythmia available for analysis; therefore the current study was

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planned as prospective as well as retrospective one. It was a combination of prospective follow-up as a continuation of our previous study [2] supplemented by additional patients (retrospective subgroup) who survived ventricular fibrillation (VF). Every HCM survivor after VF and before ICD implantation had a CMR examination. The prospective study [2] was started in March 2004 and CMR was performed at baseline. The inclusion criterion in 2004 was the presence of nsVT. Additionally, a HCM control group without nsVT was recruited (for details see reference [2]). The exclusion criteria were contradictions to a CMR examination (pacemaker, ICD) or 'secondary' instead of 'primary' myocardial scarring by septal alcohol ablation.

In this prospective subgroup during follow-up (maximum 48 months, minimum 12 months, mean 37 months) we examined a potential relationship between LGE visualised by CMR and life-threatening ventricular tachyarrhythmia or its equivalent (sudden death, adequate discharge of ICD). During follow-up patients attended control visits every 6 months. If there was any episode of syncope or ICD discharge, patients underwent immediate control. Additionally, we included in the study new patients who survived VF (retrospective subgroup), in whom a CMR study was performed just before ICD implantation (secondary prevention). In these patients follow-up was unnecessary; CMR was performed exactly at the moment of outcome, survival after VF.

Finally, the study group consisted of 55 patients (37 males and 18 females). Patients were treated pharmacologically with the following drugs: verapamil (12 patients), beta-blockers including sotalol (39 patients), amiodarone (2 patients), no drug (2 patients). The classical risk factors of sudden death, i.e. nsVT, syncope, left ventricular (LV) wall thickness ≥ 30 mm, positive family history of sudden death, were analysed. Mean number of risk factors was 1.62 per patient. Abnormal response of systemic good pressure to exercise was not analysed because the majority of our patients were above 40 years of age (when this abnormality is no longer considered a risk factor for sudden death). Mean value of LV mass index was 150.3 g/m^2.

We compared the frequency and extent of LGE in 55 HCM patients between two subgroups of patients: the group with VF [VF (+)] and the group without VF during follow-up [VF (–)]. Group VF (+) consisted of 14 patients with any one of the following episodes: sustained ventricular tachycardia/VF during follow-up (with adequate intervention of ICD) or sudden death during follow-up or VF survivors. Group VF (–) consisted of 41 HCM patients without these events.

Methodology of CMR study

Cardiac CMR examinations were performed on a Magnetom Sonata 1.5 T (Siemens) using a body array coil. First, to determine the long and short axes of LV a series of T1 SE scout images were acquired as follows: 1) sagittal orientation, 2) single oblique horizontal plane through LV, 3) double oblique vertical long axis through LV (through centre of LV apex and mitral valve), 4) double oblique horizontal long axis through LV. Then, a series of cine LV long axis projections with retrospective ECG gating (True-FISP; 2-chamber and 4-chamber) were acquired. For function and mass assessment a stack of short-axis pre-contrast cine images (the same parameters as above) through the entire LV was obtained from the mitral valve level to the apex (slice thickness 8 mm, 20% gap, matrix 224 × 256, FoV – minimal possible to avoid wrap-around artefacts, flip angle 45 deg, temporal resolution < 50 ms, TE – 4.3 ms). The LGE images were obtained approximately 10 min after a peripheral bolus injection of 0.2 mmol/kg gadolinium diethylenetriaminepentaacetic acid (Gd-DTPA) using an inversion recovery fast gradient echo sequence, triggered every heartbeat interval with averaging of two acquisitions (IR multislice Turbo-Flash). The parameters of the sequence were as follows: voxel size was 1.7 × 1.4 × 8 mm, the in-plane resolution was 2.4 mm (40 ul/voxel), matrix 192-256 × 192-256, Flip angle 20 deg, TR -, TE -, FoV – minimal possible to avoid wrap-around artefacts, trigger delay set to mid-diastole. Slices were positioned in the same location as the cine images short axis orientation – slice thickness of 8 mm, 20% gap. The optimal inversion time was established using a late hyperenhanced scout sequence (LE-scout) performed before every IR multislice Turbo Flash.

Next, the vertical and horizontal long axis images were obtained using one slice segmented IR Turbo Flash forwarded by LE-scout.

Data analysis

Left ventricular ejection fraction (LVEF) end-diastolic volume and end-systolic volume and LV mass were calculated on the basis of end-diastolic, end-systolic endo- and epicardial manual tracings performed by a trained cardiologist using computer assisted planimetry (Argus Software, Siemens, Leonardo Workstation). The LGE was quantified by thresholding window setting at 2 standard deviations above mean signal intensity of the remote normal myocardium. Fibrosis extent was quantified using computer assisted manual planimetry and reported in grams of fibrotic myocardium. Fibrosis extent was also summarised as percent of LV mass that appeared fibrotic. The pattern of segmental location of hyperenhancement was classified as confluent or diffuse according to the Moon et al. criteria [5].

To determine intra-observer variability for the LGE assessment, the mass of LGE was measured in a random order at least 3 months later by the same observer unaware of prior results. To assess inter-observer variability, LGE was measured by a second observer blinded to the results obtained by the first one.

Statistical analysis

Data are expressed as means ± standard deviation or number and percentage. Differences between the two groups were tested for statistical significance, using the t-test for continuous variables. The Fisher exact test was used for categorical variables. A p-value < 0.05 was considered to indicate statistical significance.

The intra- and inter-observer variability for LGE mass assessment were determined as mean absolute difference (bias) and 95% CI of the mean difference (limits of agreement) according to the Bland and Altman method.

Results

In the whole HCM group (55 patients) the following risk factors for sudden death were present: syncope (21 patients), nsVT (39 patients), positive family history of sudden death (23 patients) and LV wall thickness ≥ 30 mm (6 patients).

Intra- and inter-observer variability for LGE mass assessment was low with a mean bias of 0.2 g (limits of agreement ± 1.9 g) and –0.4 g (limits of agreement ± 2.2 g), respectively.

Twenty-two patients had LGE at the right ventricular (RV) insertion point with the septum. In group VF (+) all patients (100%) had some LGE whereas in group VF (–) 85% of the patients presented this abnormality (p = 0.13). There were no statistical differences between groups (Table I) in the following parameters: total LV mass, mass of hyperenhanced myocardium and percent of hyperenhanced myocardium. However, the hyperenhanced mass and percentage seemed slightly larger in group VF (+). The subanalysis of the prospective subgroup also revealed nonsignificant differences. Table I contains a summary of comparisons between groups with respect to frequency of syncope, nsVT and family history of sudden death. The differences were insignificant but the size of subgroups was small.

The pattern of hyperenhancement (confluent or diffuse) did not differ between groups. Table II shows quartiles of percent of LGE and event rates.

Table I. Comparison of clinical, echocardiographic and risk factor parameters between the studied groups

Characteristics	Group without VF n = 41	Group with VF n = 14	p
Age	46.8 ± 14.5	47.1 ± 13.3	NS
LV mass [g]	229.1 ± 72.8	245.6 ± 71.4	NS
LGE [g]	15.7 ± 15.9	23.2 ± 20.2	NS
LGE (% of LV mass)	7.2 ± 7.2	9.3 ± 8.1	NS
Maximal myocardial thickness at diastole [mm]	24.9 ± 4.5	24.7 ± 3.2	NS
LV end-diastolic volume [ml]	129.6 ± 32.4	118.9 ± 25.8	NS
LV end-systolic volume [ml]	40.2 ± 13.0	37.1 ± 16.2	NS
Ejection fraction [%]	68.9 ± 8.3	68.8 ± 10.7	NS
Syncope	14/41 (34.1%)	5/14 (35.7%)	NS
nsVT	27/41 (65.8%)	12/14 (85.7%)	NS
Family history of sudden death	19/41 (46.3%)	4/14 (28.6%)	NS
LV wall thickness ≥ 30 mm	4/41 (9.8%)	2/14 (14.3%)	NS

Abbreviations: LV – left ventricular, LGE – late gadolinium enhancement, nsVT – non-sustained ventricular tachycardia

Discussion

In this preliminary study in HCM patients with life-threatening ventricular tachyarrhythmia, the presence and extent of LGE were not significantly different from those in patients without such tachyarrhythmias during follow-up. Perhaps, in a larger group with longer follow-up a significant difference would be found. Another explanation is that the arrhythmogenic substrate may be incompletely imaged (underestimated) by LGE in CMR (e.g. invisible disarray). To test this hypothesis we need direct comparison between CMR findings and pathomorphological examination. In one case report, Moon et al. [6] were able to present such data. They revealed in pathological specimens that LGE represented regions of increased myocardial collagen but not disarray in HCM. Interestingly, it was suggested [7] that any diffuse interstitial expansion would not be visualised since LGE represents only focal regions of increased fibrosis but not all fibrosis. The LGE may correlate with plasma biomarkers of collagen turnover [8], but the contribution from diffuse hypertrophy will be missed.

Regions of LGE do not necessarily represent complete replacement fibrosis [7]. A morphological study assessed the relationship between CMR detected LGE and pathology [6]. When a myocardial segment had more than 20% fibrosis (not 100%), the segment was perceived visually as enhancing. However, the range of fibrosis in enhancing segments was from 20 to 90%, all appearing broadly similar. Importantly, LGE is not a specific finding to HCM, although very common. In aortic stenosis (considered as a phenocopy of HCM [7]) LGE was detected only in 23% and this phenomenon was significantly limited [9].

Table II. Quartiles of percent of LGE and event rates

	1st quartile LGE < 2.9%	2nd quartile LGE 2.9-5.7%	3rd quartile LGE 5.8-10.1%	4th quartile LGE > 10.1%
Number of patients from VF (+) subgroup	2	2	5	5

An additional potential explanation is that life--threatening ventricular tachyarrhythmia may be related to a stronger trigger rather than a larger arrhythmogenic substrate. Alternatively, as postulated by Kwon et al. [1], medical treatment might protect against ventricular arrhythmias. Accordingly, some of our patients (treated also pharmacologically) with high proportion of LGE had no episodes of life-threatening ventricular tachyarrhythmia during follow-up.

Unfortunately, we did not perform periodic CMR during follow-up because in some patients an ICD had been implanted, which is a contraindication for CMR. Also, these preliminary results have to be replicated in larger populations with longer follow-up time.

It has been shown that in ischaemic heart disease [10-12] and dilated cardiomyopathy [13-15] LGE was associated with ventricular arrhythmia or adverse outcomes. The results of our study, in which sudden death and its equivalent were analysed, and those reported recently by Maron et al. [4], in which annual adverse cardiovascular event rate was calculated, contradict this finding. There are several possible explanations for this discrepancy. Disarray occurs only in HCM. The type of fibrosis and its pro-arrhythmic potential is probably different in HCM versus non-HCM diseases.

We hypothesised that the absence of hyperenhancement may be an indicator of more benign prognosis while the presence of LGE may be a marker of higher risk for sudden death. Recently, Maron et al. [16] described a HCM patient, an asymptomatic 21-year-old male student with a prophylactically implanted ICD. The decision for implantation of an ICD was based largely on the presence of apparent extensive myocardial fibrosis on contrast-enhanced CMR, currently not considered a risk factor in this disease. Fifteen months later, VF was interrupted by an appropriate defibrillator shock. Maron et al. [16] concluded that this case suggests a future role for contrast--enhanced cardiovascular CMR in the risk stratification of this complex disease. At this moment we can only conclude that all patients with an event had some LGE on CMR.

Limitations of the study

To test as effectively as possible the hypothesis that VF (+) patients had a larger amount of LGE we tried to maximise the numbers of patients with VF (or equivalent) episodes for analysis and therefore the current study was performed as a combination of prospective/retrospective investigation.

Many of our patients had nsVT since this arrhythmia was an inclusion criteria in our previous study [2]. This appeared to cause a selection bias towards sicker patients. However, HCM patients also without nsVT as a control group were included in the previous study [2] and they have been followed up in the current study.

To maximise the number of patients with the end-point we included in the VF (+) group not only (a) VF survivors and (b) victims of sudden death but also (c) one patient with sustained VT. Sustained VT detected by ICD and treated by anti-tachycardia pacing (as adequate ICD intervention) is considered as an equivalent of VF. Adequate ICD intervention occurred in 9 patients (8 patients with VF and one patient with sustained VT), VF arrest occurred in 5 patients (4 patients were resuscitated and one patient had a witnessed sudden death).

The VF survivors were referred from remote regional centres for ICD implantation (secondary prevention of sudden death). For these patients it was a first visit to our centre and CMR was performed immediately and an ICD was implanted within the same day or the next day. These patients achieved the primary end-point and follow-up was impossible.

The next limitation is the extended recruitment period in our study. However, other investigators have reported a similar situation, e.g. more than 4 years for recruitment [4].

In HCM, prospective data correlating CMR findings exclusively to episodes of sudden death are not yet available. In the recent study of Maron et al. [4] a combined end-point was used. The end-point defined as adverse events occurred in 11 patients, including 7 patients with LGE (2 with sudden death, 2 with appropriate ICD discharge, and 3 with progressive heart failure symptoms) and 4 patients without LGE (3 with sudden death and 1 with progressive heart failure). In the study of Maron et al. [4] only 7 patients with sudden death (or its equivalent) were analysed while in our study 14 such patients were available. Summing up, both in the current study and in the study by Maron et al. [4] the outcome analysis was clearly underpowered considering the low event rate characteristic of HCM. A substantially longer follow-up period will be required in a particularly large patient population to achieve adequately powered positive or negative data in this respect.

Conclusions

In HCM patients with life-threatening ventricular tachyarrhythmia LGE was both qualitatively and quantitatively comparable with that in patients without these tachyarrhythmias. Hypothetically, the arrhythmogenic substrate may be incompletely imaged (underestimated) by LGE in CMR or life-threatening ventricular tachyarrhythmia may be related to a stronger trigger rather than a larger substrate. However, larger populations are needed to confirm this finding.

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Późne kontrastowanie mięśnia sercowego w rezonansie magnetycznym u chorych z kardiomiopatią przerostową powikłaną częstoskurczem komorowym lub migotaniem komór

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Streszczenie

Cel: Weryfikacja hipotezy o istnieniu ewentualnej zależności pomiędzy zjawiskiem późnego kontrastowania mięśnia sercowego (LGE) uwidocznionym w kardiologicznym rezonansie magnetycznym (CMR) a występowaniem groźnych komorowych zaburzeń rytmu.

Metody: Kontrastowanie gadolinem (w tym patologiczny objaw późnego kontrastowania – LGE) zostało ocenione u 55 chorych z kardiomiopatią przerostową. Porównano częstość występowania LGE oraz jego rozległość pomiędzy grupą chorych z utrwalonym częstoskurczem komorowym (VT) lub zreanimowanym migotaniem komór (VF), lub nagłym zatrzymaniem krążenia – grupa VF (+), i grupą pacjentów bez powyższych tachyarytmii i bez epizodu nagłego zatrzymania krążenia – grupa VF (–). W grupie VF (+) było 14 chorych, a w grupie VF (–) 41, średni okres obserwacji wynosił 37 miesięcy.

Wyniki: W grupie VF (+) stwierdzono adekwatne interwencje ICD u 9 chorych (8 osób ze zdefibrylowanym epizodem VF i jedna osoba z epizodem utrwalonego VT przerwanego stymulacją *anty-tachycardia pacing*, natomiast VF przed implantacją ICD wystąpiło u 5 chorych (4 chorych skutecznie zreanimowano, u jednego pacjenta doszło do nagłego zatrzymania krążenia w obecności świadka). W grupie VF (+) LGE stwierdzono u wszystkich osób, natomiast w grupie VF (–) u 85% badanych (p = 0,13). Pomiędzy grupami VF (+) i VF (–) nie stwierdzono istotnych statystycznie różnic pod względem: wieku (46,8 ± 14,5 *vs* 47,1 ± 13,3 roku), całkowitej masy lewej komory (229,1 ± 72,8 *vs* 245,6 ± 71,4 g), maksymalnej grubości ściany lewej komory (24,9 ± 4,5 vs 24,7 ± 3,2 mm), masy obszaru późnego kontrastowania (15,7 ± 15,9 *vs* 23,2 ± 20,2 g), procentowej wielkości mięśnia sercowego ulegającemu zakontrastowaniu (7,2 ± 7,2 *vs* 9,3 ± 8,1%).

Wnioski: U chorych z kardiomiopatią przerostową wielkość obszaru późnego kontrastowania mięśnia sercowego była porównywalna pomiędzy grupami chorych z groźnymi komorowymi zaburzeniami rytmu (utrwalony VT lub VF, lub nagłe zatrzymanie krążenia jako ekwiwalent) i bez tych tachyarytmii oraz bez epizodu nagłego zatrzymania krążenia.

Słowa kluczowe: kardiomiopatia przerostowa, nagłe zatrzymanie krążenia, rezonans magnetyczny

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