Late Gadolinium Enhancement Magnetic Resonance Imaging in the Diagnosis and Prognosis of Endomyocardial Fibrosis Patients

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Background—Endocardial fibrous tissue (FT) deposition is a hallmark of endomyocardial fibrosis (EMF). Echocardiography is a first-line and the standard technique for the diagnosis of this disease. Although late gadolinium enhancement (LGE) cardiovascular magnetic resonance (CMR) allows FT characterization, its role in the diagnosis and prognosis of EMF has not been investigated.

Methods and Results—Thirty-six patients (29 women; age, 54 ± 12 years) with EMF diagnosis after clinical evaluation and comprehensive 2-dimensional Doppler echocardiography underwent cine-CMR for assessing ventricular volumes, ejection fraction and mass, and LGE-CMR for FT characterization and quantification. Indexed FT volume (FT/body surface area) was calculated after planimetry of the 8 to 12 slices obtained in the short-axis view at end-diastole (mL/m²). Surgical resection of FT was performed in 16 patients. In all patients, areas of LGE were confined to the endocardium, frequently as a continuous streak from the inflow tract extending to the apex, where it was usually most prominent. There was a relation between increased FT/body surface area and worse New York Heart Association functional class and with increased probability of surgery (P < 0.05). The histopathologic examination of resected FT showed typical features of EMF with extensive endocardial fibrous thickening, proliferation of small vessels, and scarce inflammatory infiltrate. In multivariate analysis, the patients with FT/body surface area > 19 mL/m² had an increased mortality rate, with a relative risk of 10.8.

Conclusions—Our study provides evidence that LGE-CMR is useful in the diagnosis and prognosis of EMF through quantification of the typical pattern of FT deposition. (Circ Cardiovasc Imaging. 2011;4:304-311.)

Key Words: myocardial cardiomyopathy disease • endomyocardial fibrosis • restrictive cardiomyopathy • heart failure • magnetic resonance imaging • gadolinium • prognosis

Endomyocardial fibrosis (EMF) is the most frequent restrictive cardiomyopathy, affecting about 12 million people in the world.1 It is characterized by fibrotic tissue (FT) deposition in the endocardium of the inflow tract and apex of one or both ventricles. Ventricular morphology is usually distorted with normal or reduced volumes, whereas atrial volumes are increased. The cause of EMF is unknown; however, early hypereosinophilia may play a role in its pathogenesis.1 Fibrosis of the subvalvular apparatus and chords leads to atrioventricular valve regurgitation. Diastolic dysfunction is responsible for the severe heart failure (HF) in EMF patients and plays an important role in determining exercise limitations.2 Clinical treatment is often unsatisfactory, and FT resection is the treatment of choice for patients in New York Heart Association (NYHA) functional classes III and IV.3

Clinical Perspective on p 311

Echocardiography is a first-line and the gold standard noninvasive technique for diagnosing EMF.4,5 It enables the identification of the disease in early stages, the quantification of the degree of morphological and hemodynamic compromise, and assessment of changes over time or with surgery.5 However, echocardiography is unable to fully characterize...
FT, and it may not fully differentiate EMF from other cardiac diseases presenting as left ventricular (LV) apical obliteration, such as apical hypertrophic cardiomyopathy,1 cardiac tumors,1,6 apical thrombus, or noncompaction of the ventricular myocardium.7 In patients with predominantly right ventricular (RV) involvement, the differential diagnoses includes Ebstein anomaly1,3 and constrictive pericarditis.1 Contrast ventriculography was at first considered the gold standard method for EMF diagnosis,8 but, with improvements in echocardiographic images quality, it is no longer routinely performed.1 In addition to its risk, endomyocardial biopsy allows the diagnosis in only about 50% of the patients.10

Cardiovascular magnetic resonance (CMR) provides detailed information on ventricular morphology and function, including excellent visualization of the ventricular apex. Late gadolinium enhancement (LGE)-CMR allows the evaluation of the presence of myocardial inflammation, fibrosis, and injury caused by a relative accumulation of gadolinium that occurs in these conditions as the result of slower washout kinetics and the increased extracellular volume.11,12 Precise EMF diagnosis and evaluation of fibrosis may allow surgical intervention in a less advanced stage of the disease to improve quality of life and prognosis.13,14 The goal of the present study was to investigate the role of LGE-CMR in the diagnosis and prognosis of EMF.

Methods

Study Population and Groups

Inclusion criteria were age >18 years, a clinical diagnosis of diastolic HF, defined by the finding of typical symptoms and signs of HF in a patient showing preserved LV ejection fraction (LVEF) and no valvular disease. Also, the echocardiogram showing apical obliteration of one or both ventricles with a hypertrefrangent apex and atrial dilation were used as entry criteria for EMF, as previously demonstrated.11 The exclusion criteria were creatinine clearance <30 mL/min, relative or absolute contraindications to CMR studies, and the presence of other systemic or cardiac diseases.

We prospectively studied 36 patients (29 women; age, 27 to 77 years, 54±12 years) with diastolic HF diagnosis followed at outpatient cardiology clinic. A detailed clinical history, physical examination, complete blood count including eosinophil count, and comprehensive 2-dimensional Doppler echocardiography were carried out, resulting in a diagnosis of EMF. The patients then underwent cine-CMR and LGE-CMR to fully detect and quantify the FT deposition.

The EMF patients were classified on the basis of location of LGE-CMR and were divided into the following subgroups: LV-FT for patients with fibrosis in the LV; RV-FT for patients with fibrosis in the RV; and Bi-FT, for patients with fibrosis in both ventricles. Minimal amounts of scattered or punctuate fibrosis detected by LGE-CMR in the contralateral ventricle were not considered in this classification and could not be added to the FT volume.

Control and Hypertrophy Groups

To evaluate the diagnostic performance of LGE-CMR in EMF patients, we compared the results obtained in this patient population with those acquired from 10 healthy volunteers (all men; age, 44±8 years; all with normal ECGs and echocardiograms; control group) and 15 patients with LV hypertrophy (9 men; age, 37±16 years; all with hypertrophic cardiomyopathy, including 2 apical forms; with LV hypertrophy on ECG and echocardiography; LVH group).

Patients with confirmed EMF were treated with standard HF pharmacological therapy. Warfarin was used if permanent atrial fibrillation or previous thromboembolic events were present. Patients remaining in NYHA functional classes III and IV after medical therapy were offered surgical resection.1 The surgical specimens were processed for histopathologic analyses. The study was approved by our institutional review board, and informed consent was obtained from all study subjects.

CMR Imaging Protocol

CMR studies were performed with a 1.5-T magnet (General Electric Medical Systems, SIGNA, CV/I, Waukesha, MN) using a cardiac phased-array surface coil. The cardiac images were obtained during breath-holding for 12 to 15 cardiac beats (10 to 15 seconds on average) at the end of the expiration, synchronized with the ECG. Two specific pulse sequences were used: cine-CMR with the steady-state free precession technique (SSFP) and LGE. Initially, the first images were acquired to evaluate the correct position of the cardiac coil and to define the short- and long-axis views of the LV. To cover the LV from apex to base, 8 to 12 short-axis slices of 8-mm thickness at 2-mm intervals were acquired. The long axis was perpendicular to the short axis, and both axis slices were acquired with the 2 pulse sequences, as previously described. The cine-CMR using the SSFP technique was performed to evaluate ventricular morphology, volumes, ejection fraction, and mass and was used for the following parameters: field of view, 34 to 38 cm; matrix, 256×160; receiver bandwidth, 125 kHz; k-space segmented, 8 to 12 lines; TR, 3.9 ms; TE, 1.4 ms; flip angle, 45°; 3/4 field of view; number of excitations equal to 1; and 20 phases for each cardiac cycle. Patients received an intravenous bolus of 0.2 mmol/kg of gadolinium-based contrast. Ten to 20 minutes after gadolinium injection, images were acquired using the LGE technique, which is a gradient-echo pulse sequence with an inversion-recovery preparatory pulse.11 The parameters used were TR, 7.2 ms; TE, 3.2 ms; matrix, 256×192; flip angle, 20°; bandwidth, 31.2 kHz; inversion time, 150 to 300 ms; number of excitations equal to 2; and acquisition in every heart beat (1 RR).13 After contrast, the inversion time was adjusted to null the signal from normal myocardium (dark myocardium) and clearly shows the FT region as intensely bright.

Image Analysis

The LV and RV volumes and derived stroke volume and EF were obtained by the detection of endocardial and epicardial contours of all short-axis slices at end-diastole and end-systole and application of the Simpson method. Biventricular mass was calculated with Mass Analysis Plus software (MEDIS, Medical Imaging Systems, Leiden, The Netherlands) and corrected by body surface area (BSA).

The FT volume was calculated as a sum of the component volumes derived by planimetry of the 8 to 12 slices obtained from the short-axis view from apex to base at end-diastole (mL/m²) in each ventricle (FT-LV or FT-RV), and indexed by body size (FT/BSA). In the case of biventricular EMF, the FT of each ventricle was combined and corrected by BSA (FT-Bi). FT mass (g/m²) was calculated by the FT volume multiplied by the specific density of myocardium (1.05 g/cm³) and corrected by BSA. The cine SSFP and LGE images were analyzed by 2 cardiologists with extensive experience in interpreting MRI and LGE in several clinical situations, with a deep knowledge of the pulse sequence, its pitfalls, and artifacts. When disagreement between the 2 observers occurred, the final decision was made by consensus.

Histopathologic Analysis

Sixteen patients underwent surgical resection of the FT. The excised tissue obtained from 14 patients was fixed in formalin, underwent decalcification if necessary, and embedded in paraffin. Four-micrometer-thin sections were stained with hematoxylin and eosin and Masson trichrome and analyzed under light microscopy.

Statistical Analysis

All data are expressed as mean±SD or frequency (%) for discrete variables. The likelihood ratio test or χ² test was used to compare EMF groups for sex, NYHA functional class, cardiac rhythm, and mortality. The Pearson correlation coefficient was used to analyze the relation between RV and LV FT/BSA with RV and LV mass/BSA, respectively. Comparisons of normally distributed continuous
variables were performed with the t test and 1-way ANOVA with Bonferroni adjustments for multiple comparisons. The t test was used to compare surgical or clinical treatment and FT/BSA. Cutoff value was determined on the basis of receiver operating characteristic curve, which was generated by logistic regression. The Kaplan–Meier method was used for survival curves, with the log-rank test with a mean RV mass of 22 g/m² and FT/BSA of 19 mL/m² used as the cutoff value. The significant variables were adjusted in the multivariate analysis model by Cox regression. The covariates that were candidates for entry in multivariate model were BSA (P<0.044), RV mass/BSA (P<0.011), and FT/BSA (P<0.001). The significance level predictor to enter the model was 0.05 and to be eliminated from it was 0.10. Normality was determined by the Shapiro-Wilk test. Two-sided P<0.05 was considered statistically significant. Statistical analysis was performed with SPSS v 15 (SPSS Inc, Chicago, IL).

Results

The typical EMF image observed consisted of areas of LGE in the endocardium, mainly in the apex and eventually in the inflow tract of one or both ventricles, frequently as continuous stria, with preservation of both outflow tract orifices and not confined to coronary territory. The CMR LGE pattern had a “V sign” at the ventricular apex, characterized by a 3-layer appearance of myocardium, thickened fibrotic endomyocardium, and overlying thrombus. Typical LGE-CMR images are shown in Figure 1, and cine-CMR of EMF and hypertrophic cardiomyopathy patients are shown in Figure 2 and video 1 (online-only Data Supplement). Clinical characteristics and CMR values from EMF patients are shown in Table 1.
All patients presented apical areas of LGE, 17 in the LV (47%, LV-FT), 7 in the RV (20%, RV-FT), and 12 in both ventricles (33%, Bi-FT) (Table 1). The FT/BSA and FT mass/BSA were 10±1 mL/m² and 10±1 g/m² in LV-FT, 13±9 mL/m² and 14±2 g/m² in RV-FT, and 22±9 mL/m² and 25±8 g/m² in Bi-FT, with greater Bi-FT volume and Bi-FT mass (P<0.001).

In our study group of 36 patients, we considered the comprehensive echocardiogram for the diagnosis criteria of EMF. On a retrospective analysis, we found that 9 of the total 36 patients had had their first echocardiogram with findings that differed from the final EMF diagnosis. The initial diagnoses were normal (n=3), mitral valvulopathy (n=2), dilated cardiomyopathy (n=1), suspected Ebstein anomaly (n=1), apical hypertrophic cardiomyopathy (n=1), and pulmonary hypertension (n=1).

The EMF subgroups were similar in age, sex, BSA, LVEF, LV mass, stroke volume, and LV and RV volumes (Table 1).
Of the 36 patients with EMF, 20 were in NYHA functional classes III and IV (Figure 3). Atrial fibrillation was less common in the LV-FT.

The RV-FT subgroup had higher RVEF compared with those in the LV-FT and Bi-FT subgroups (59±14%, 43±11%, and 43±13%, respectively; P=0.016). Although LV mass/BSA was not different among groups, RV mass/BSA was reduced in the LV-FT subgroup (Table 1). RV-FT and Bi-FT subgroups had a higher percentage of patients in NYHA functional class III/IV (71% and 83%, respectively) than did the LV-FT (29%; P=0.008).

On a retrospective evaluation, greater FT/BSA was found to correspond to a worse NYHA functional class and higher likelihood of surgery (Figure 3 and Figure 4). RV FT/BSA correlated positively with RV mass/BSA (r=0.773; P<0.001). Cutoff values were determined by using the receiver operating characteristic curve and showed that patients with RV mass/BSA >22 g/m² and FT/BSA >19 mL/m² had worse survival rates (Figure 5). In multivariate analysis, FT/BSA was the only independent predictor of mortality, with a relative risk of 10.8 and 95% confidence interval of 2.10 to 55.39 (Figure 6, Table 2).

FT resection was indicated in 20 patients with NYHA functional classes III and IV. Four of these patients refused surgery. Of the 16 patients who underwent surgical resection, 5 died in the first year after surgery and 1 died 3 years after surgery. Two other deaths occurred in the clinical therapy group: One was a patient who had refused surgery and the other was from the clinically treated group. All deaths were from cardiac causes (4 patients with sudden cardiac death and 4 patients from worsening HF). There was a tendency for fewer surgical procedures and lower mortality rates in the LV-FT subgroup (Table 1).

LGE-CMR in Control, Hypertrophy, and EMF Groups
The frequency and/or pattern of LGE in the 3 groups were as follows: in the control group of 10 individuals, no LGE; in the hypertrophy group of 15 patients, 4 LGE (27%), showing small spotty areas of fibrosis limited to the mid layers of the myocardium. The EMF group of 15 patients (100%) had areas of LGE in the endocardium, mainly in the apex and eventually in the inflow tract of one or both ventricles, frequently appearing as continuous stria. A correct diagnosis was reached by consensus of the examiners in all but 1 case, diagnosed as normal when the patient had mild LVH.

Eosinophil Count
Eosinophil counts were <1500/mm³, except in 1 patient (1786/mm³).

Histology
Of the 16 surgical cases, pathological data were not available in 2 cases. The remaining 14 cases had histological features typical of EMF. Extensive endocardial fibrous thickening irregularly penetrated the subendocardial myocardium. Additionally, small-vessel proliferation and mild-to-moderate inflammatory infiltrate with scarce eosinophils were present in the endocardium (Figure 1). Fibrosis was present in all specimens; superficial thrombosis was present in 9 of 14
Table 2. Results of Multivariate Cox Regression

<table>
<thead>
<tr>
<th>Variable</th>
<th>Estimated Parameter</th>
<th>Standard Error</th>
<th>P</th>
<th>Relative Risk</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>FT/BSA, &gt;19 mL/m²</td>
<td>2.38</td>
<td>0.83</td>
<td>0.004</td>
<td>10.79</td>
<td>2.10</td>
</tr>
</tbody>
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(64%) cases and calcification was present in 6 of 14 (43%) cases.

Discussion

The utility of LGE in the diagnosis of EMF has been limited to a few case reports.14,16,17 The present study demonstrates the typical LGE-CMR in a relatively large number of patients with EMF on echocardiography. In addition, the histological analysis confirmed the LGE-CMR diagnosis in all 14 patients who underwent surgical FT resection.

In all patients, LGE regions were observed only in the endocardium, appearing as a continuous area, commonly extending from the subvalvar region to the apex of the ventricles, where it was usually more prominent. Also, a 3-layered appearance (V sign at the apex) was typical of this disease with an inner layer showing no perfusion due to presence of thrombus, a middle layer of LGE reflecting the FT and an outer layer of almost normal myocardium. In our surgical subgroup, all EMF diagnosed by LGE-CMR was confirmed by histopathology. These data suggest that LGE-MR may play a role in EMF diagnosis.

The patients’ first echocardiography report differed from the final EMF diagnosis in 9 patients. Considering the low prevalence of EMF in the population, an inexperienced examiner may miss the EMF diagnosis. This might suggest that in the event of suspected EMF, apical hypertrophy, or restrictive cardiomyopathy with apical filling, an LGE-CMR study might help in defining the correct diagnosis by delineating clearly the morphology of the apex and characterizing the FT.

RV involvement evaluated invasively by ventricular angiography is a predictor of a worse prognosis in EMF patients.9 Tissue characterization obtained with LGE-CMR allows for a critical evaluation of the fibrotic deposition occurring in the right ventricle. The RV-FT subgroup had higher RVEF compared with the other groups; probably the small RV cavity in these patients is the main factor leading to a higher EF.

Previous studies demonstrated that atrial fibrillation is a marker of a worse prognosis in this disease18 and that RV-FT and Bi-FT subgroups have a poorer evolution.9 In our study, RV-FT and Bi-FT subgroups presented a higher incidence of atrial fibrillation, which might indicate that this arrhythmia may be a contributing factor of worse prognosis in these subgroups.

CMR allowed the noninvasive quantification of cardiac volumes. A noteworthy finding is the significantly reduced LV stroke volumes (33±10 mL) in all patients and subgroups compared with the normal range (104±21 mL),19 which, associated with preserved LVEF, is typical of restrictive cardiomyopathy.

Endomyocardial Biopsy and LGE-CMR in EMF

In some institutions, RV endomyocardial biopsy is part of the EMF diagnostic workup, providing the diagnosis and excluding other restrictive cardiomyopathies, particularly infiltrative and storage diseases. However, it has been previously demonstrated that endomyocardial biopsy allows the diagnosis in only about 50% of patients.10 Our data indicate that a biopsy may not be required for establishing the diagnosis of EMF if a LGE-CMR study provides reliable evidence based on criteria we describe here. We also propose LGE-CMR as the best noninvasive method for diagnosing EMF.

Eosinophils, Thrombus, and Calcification

A high eosinophil count may be found in the initial stages of the disease.1 Our patients, who had normal or mild elevation of eosinophil counts, probably were in the later fibrotic phase of the disease, as corroborated by the histopathologic analysis of surgical specimens showing a low number of inflammatory cells. Although it has not been already demonstrated, we speculate that earlier endomyocardial alterations, such as the initial inflammatory process, may be detected with LGE-CMR performed at the initial stage of the disease. The presence of thrombus may be detected by LGE-CMR as regions with a dark appearance surrounded by bright contrast-enhanced blood.20 With LGE-CMR, we observed the presence of these dark-appearing regions. Areas of calcification, which were found in the histological analysis, are difficult to distinguish from thrombus on the basis of LGE images only (with the same dark appearance). However, CMR can differentiate thrombus from calcification because the acute thrombus that will appear bright on both T1- and T2-weighted images and subacute thrombus that will be bright on T1 and with a low signal on T2, due to the paramagnetic effect of methemoglobin and shortening of T2 relaxation times.21 Chronic organized thrombus has low signal intensity on both T1 and T2 images as the result of decreased water content and/or concomitant calcification and might be more difficult to differentiate from calcification alone.22 The specific techniques described above were not performed in our study.

Differentiating EMF From Other Cardiomyopathies Using LGE-CMR

FT deposition is also a frequent finding in Chagas disease, with a heterogeneous midwall and subepicardial distribution preferably in the apex and basal inferolateral segments, although it can appear in a pattern indistinguishable from alterations found in coronary artery disease.15 This pattern of FT distribution and appearance is clearly distinct from the homogeneous and exclusively endomyocardial distribution found in our EMF patient study population. In EMF patients, the apical systolic inward motion is commonly preserved,23 differing from the dysskinetic motion of thrombotic apical
obliteration occurring in patients with coronary artery disease or Chagas disease.\(^{12}\)

Other conditions, such as amyloidosis, sarcoidosis, and subendocardial infarction, could cause subendocardial LGE.\(^ {12}\) The most important aspect that clearly differentiates EMF from other causes is that the LGE is subendocardial but on top of an apical filling mass and frequently extends to the inflow tract involving subvalvar apparatus. LGE in EMF is also heterogeneous with hypointense areas that can be associated with thrombus or calcification. In subendocardial infarction, however, the LGE is related to a coronary artery distribution, there is no apical filling mass, and when involving the apex is usually associated with wall thinning and wall motion abnormality. LGE in cardiac amyloidosis often demonstrates global, inhomogeneous, subendocardial hyperenhancement associated with a global increase in wall thickness and systolic dysfunction. LGE in sarcoidosis will appear as areas of myocardial enhancement in regions with granulomas. These areas often show wall thinning and wall motion abnormalities. They are usually located in the septum but can affect other walls, with midwall, epicardial, or transmural involvement, whereas papillary muscle and the RV wall are rarely affected. In LV noncompaction, thickened apical segments of the myocardium have a double-layered appearance with marked trabeculations and deep intratrabecular recesses located in the mid and apical portions of the ventricle. Nontransmural LGE of the prominent trabeculations suggesting areas of fibrosis is more frequent than transmural LGE.\(^ {12}\)

LGE-MRI and Surgery in EMF Patients

Surgical resection of FT is the recommended treatment for patients in NYHA functional classes III and IV.\(^ {24,25}\) The FT distribution and localization obtained with LGE-CMR may contribute to surgical planning, offering anatomical details that may guide the surgical procedure. In particular, the extent of apical calcification may be of special interest, leading the surgeon to choose a different approach. The increased mortality rate observed in patients with FT/BSA >19 mL/m\(^2\) may be related to the severity of LV filling restriction and to the irreversible myocardial damage triggering arrhythmic events. The follow-up assessment of the quantification of FT by LGE-CMR, including after surgery, may contribute to a better understanding of the disease progression and recurrence.\(^ {26}\)

Conclusions

Our study provides evidence that LGE-CMR is useful in the diagnosis of EMF by characterization of FT distribution, which is the hallmark of the disease. A consistent pattern of LGE was observed, with FT present only in the subendocardium, appearing as a continuous area, commonly extending from the subvalvar region to the apex of the ventricles, where it was usually more prominent. Morphological and tissue characterization by LGE-CMR leads to a confident diagnosis of EMF. Our initial data suggest that parameters evaluated by CMR, such as FT/BSA, can convey prognostic information.

Acknowledgment

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Disclosures

None.

References

We evaluated the role of late gadolinium enhancement (LGE) cardiovascular magnetic resonance (CMR) for the diagnosis of endomyocardial fibrosis (EMF) using surgical specimens as the standard method. LGE-CMR confirmed the diagnoses of EMF patients on the basis of areas of LGE that were confined to the endocardium as continuous stria from the inflow tract to the apex. Histopathology of fibrous tissue in 14 patients showed typical features of EMF. Our study provides evidence that LGE-CMR is a reliable diagnostic tool to confirm EMF.
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Supplemental Material

Video. Four-chamber, long-axis, cine-CMR showing left ventricular apical obliteration (upper left), right ventricular (upper right), biventricular (lower left) by endomyocardial fibrosis and cine-CMR of a patient with hypertrophic cardiomyopathy (lower right) showing the classic spade-shaped ventricular cavity in systole for comparison.