The Additional Value of Gadolinium-Enhanced MRI to Standard Assessment for Cardiac Involvement in Patients With Pulmonary Sarcoidosis*

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Aim: To determine whether gadolinium-enhanced cardiac MRI (CMR) was of additional diagnostic value to standard assessment in patients with sarcoidosis who underwent evaluation for cardiac involvement.

Methods: We reviewed the findings in patients with pulmonary sarcoidosis who had been assessed with ECG, Doppler echocardiography, 201Tl scintigraphy, and CMR from 2002 to 2004.

Results: Of the 55 evaluated patients, standard evaluation diagnosed cardiac involvement in 13 patients while CMR diagnosed myocardial scarring (mean ± SD, 2.5 ± 1.9 segments) in an additional 6 patients. The extent of delayed enhancement correlated with disease duration (p < 0.05), ventricular dimensions and function (p < 0.001), severity of mitral regurgitation (p < 0.05), and the presence of ventricular tachycardias (p < 0.001). Patients in whom cardiac involvement was diagnosed only with CMR had less myocardial scarring and functional impairment (p < 0.05) compared to patients with a diagnosis made by standard assessment.

Conclusion: CMR provides an accurate estimation of the extent of cardiac involvement and may reveal signs of early infiltration that are not detected by standard assessment. The extent of late enhancement with gadolinium relates to the severity of cardiac involvement and may therefore have prognostic implications.

Key words: Doppler echocardiography; MRI; myocardial fibrosis; myocardial scintigraphy

Abbreviations: 4CH = four-chamber view; CMR = cardiac MRI; CS = cardiac sarcoidosis; EMB = endomyocardial biopsy; IR-GRE = inversion recovery-gradient echo; LGE = late gadolinium enhancing; LV = left ventricular; LVEF = left ventricular ejection fraction; PVC = premature ventricular contraction; SA = short axis; SSFP = steady-state–free precession; VLA = vertical long axis; VT = ventricular tachycardia

Sarcoidosis is a multisystem granulomatous disorder of unknown etiology with symptomatic cardiac involvement in up to 7% of patients. Postmortem studies revealed cardiac involvement in 20 to 30% of patients with sarcoidosis in the United States. In Japan, cardiac involvement is present in as many as 55% of patients, and is responsible for as many as 85% of deaths from sarcoidosis. The clinical features of sarcoid heart disease include congestive heart failure, cor pulmonale, supraventricular and ventricular arrhythmias, conduction disturbances, ventricular aneurysms, pericardial effusion, and sudden death. The diagnosis of cardiac sarcoidosis (CS) is made by the coexistence of noncaseating granulomas on myocardial biopsy or biopsies of any extracardiac tissue (with the exclusion of other causes for granulomatous inflammation such as mycobacterial or fungal infection) and cardiac abnormalities for which other possible causes have been excluded.

Many tests have been used to diagnose and follow-up CS, such as standard and ambulatory ECG, echocardiography, gallium scintigraphy, and thallium scintigraphy. More recently gadolinium-enhanced cardiac MRI (CMR) has been used to demonstrate...
myocardial inflammation, wall motion abnormalities, and fibrosis in patients with CS.10–12 Because of superior image resolution, CMR may be able to demonstrate early signs of myocardial infiltration when scintigraphy or echocardiography fail to do so.13 The additional value of CMR to currently employed techniques—ECG, echocardiography and 201Tl scintigraphy—remains to be determined. We aimed to evaluate the additional value of CMR in disclosing structural and functional cardiac abnormalities in patients with pulmonary sarcoidosis who underwent standard cardiac assessment either because of cardiac symptoms or for screening purposes, and correlated the findings with the techniques used in standard assessment.

**Materials and Methods**

**Study Population**

Between July 2002 and March 2004, 55 patients with histologically proven pulmonary sarcoidosis underwent cardiac assessment in the cardiology departments of the Erasmus Medical Centre (n = 4) and the University Hospital Maastricht (n = 51). The study group consisted of 38 men and 17 women (mean age, 48 years and 47 years, respectively). The patients were mainly white (n = 50; 91%), 4 patients were Asian, and 1 patient was African.

The diagnosis of pulmonary sarcoidosis was confirmed if the clinical presentation and chest radiographic findings were supported by histologic evidence of noncaseating granulomas by transbronchial biopsy, and the possibility of infection, environmental factors, or hypersensitivity reaction to medication causing granulomatous inflammation had been eliminated. The patients either presented with symptoms of cardiac involvement (n = 12) or were screened for cardiac involvement (n = 43).

Patients underwent a clinical assessment and evaluation of the maximal serum angiotensin-converting enzyme levels (n = 43); 12-lead ECG (n = 55); ambulatory ECG monitoring (n = 47); radiologic chest staging by chest radiography and/or high-resolution CT (n = 55); Doppler echocardiography (n = 55); 201Tl scintigraphy (n = 51); and gadolinium-enhanced CMR (n = 55). Diagnostic coronary angiography was done in 13 patients to exclude coronary artery disease. In four patients, endomyocardial biopsy (EMB) samples were obtained. In group A (standard assessment), the diagnosis of CS was made when EMB demonstrated noncaseating, epithelioid granulomata or, when after the exclusion of other causes, abnormalities were present on the resting or ambulatory ECG (left bundle-branch block, right bundle-branch block, left anterior fascicular block, bifascicular block, atrioventricular block, ventricular tachycardia [VT], premature ventricular contraction [PVC], or pathologic Q waves or ST-T segment changes) and echocardiogram (abnormal wall motion, regional wall thinning, or dilation of the left ventricle) or 201Tl myocardial scintigram (irreversible perfusion defect and/or uptake and/or wall thickening).

In group B (CMR group), the diagnosis of CS was based solely on abnormalities detected by CMR, and the exclusion of other conditions known to cause these abnormalities such as hypertension, coronary artery disease, or valvular heart disease. CMR findings were considered suggestive of CS when impaired systolic left ventricular (LV) function, loss of wall thickness, or gadolinium-enhancing LV lesions were present and other explanations had been excluded. Group C consisted of patients in whom CMR abnormalities suggestive of CS were present but in whom standard assessment revealed no abnormalities. Because of the limited diagnostic yield of EMB and the invasive nature with an associated risk of morbidity, we considered it not justified to systematically subject patients who were screened for CS to this procedure.

**ECG and Ambulatory ECG**

A 12-lead surface ECG was performed (MAC, paper speed, 25 mm/s; Marquette; Milwaukee, WI), and the findings were interpreted by two experienced cardiologists and assessed for the presence of abnormalities suggestive of cardiac involvement. Ambulatory ECGs were performed for 24 to 72 h, and were considered suggestive of CS when evidence of intermittent atrioventricular conduction delay or block (disregarding nightly episodes with Wenkebach periodicity), intermittent bundle-branch blocks, or ventricular arrhythmias including PVCs > 100/24 h or at least one VT was found. VT was defined as a run of PVCs containing at least 3 beats with a rate > 100 beats/min. Sustained VT was defined as VT with a rate > 100 beats/min and lasting for at least 30 s.

**Doppler Echocardiography**

Studies were performed with a phased-array imaging system (Sonos 5500, S3 transducer; Hewlett-Packard; Andover, MA). Two-dimensional, continuous-wave, and color-flow Doppler echocardiography was performed from standard parasternal and apical windows in long and short axes. Pulsed Doppler echocardiography was performed from an apical four-chamber (4CH) view with the sample volume placed at the level of the mitral valve leaflet tips, with the cursor orientated parallel to an imaginary line bisecting the left ventricle from apex to mitral valve. LV internal dimensions and wall thickness were measured at end-diastole according to the recommendations of the American Society of Echocardiography.14 LV mass was calculated and indexed for body surface area.15 Body surface area (meters squared) was derived from the Dubois formula.16 LV hypertrophy was considered present when the LV mass index was > 104 g/m2 for women or > 116 g/m2 for men.17 LV wall motion, thickness, and thickening were determined according to the 17-segment model.18 Thickness of the interventricular septum > 13 mm or wall thickness < 7 mm in any segment were considered abnormal.19 LV diastolic function was evaluated by determining the isovolumetric relaxation time, peak velocity of early and late LV filling, early/late LV filling ratio, deceleration rate of early diastolic flow, left atrial size, and flow patterns in the pulmonary veins. Diastolic function was assessed according to the Canadian Consensus Recommendations.20 LV ejection fraction (LVEF) was calculated using a modified Simpson rule.21 Right ventricular size and function and the presence of pericardial fluid were assessed.

**Thallium Myocardial Scintigraphy**

After treadmill peak exercise or during IV infusion of dipyridamole, 201Tl was administered and single-photon emission CT performed on a triple-detector gamma camera (MultiSPECT-3; Siemens; Erlangen, Germany) equipped with low-energy, high-resolution collimators. The images were made in a 64 × 64 matrix (60 frames per 45 s). The thallium scan was considered suggestive of CS when areas with reversed uptake and/or irre-
versible perfusion defects were present, and/or reversible perfusion defects were found in patients with normal coronary arteries at angiography. Regional defects were localized according to the 17-segment model.\(^1\)

**CMR**

Studies were performed using a 1.5-T MRI scanner (Philips; Best, the Netherlands; or General Electric, Milwaukee, WI) with a cardiac-dedicated, phased-array coil. The CMR studies were ECG triggered by standard software and obtained in diastole to minimize artifact due to cardiac motion. Studies consisted of multislice–multiphase steady-state–free precession (SSFP) in 55 patients, spin echo in 54 patients, and fat-saturated, T2-weighted breath-hold sequences in 55 patients. Studies were obtained of the short-axis (SA), vertical long-axis (VLA) and 4CH views. SSFP sequences were performed to assess regional wall motion abnormalities, and T2-weighted studies were performed to assess the presence of myocardial inflammation. Ten minutes after the additional administration of 0.1 mmol/kg gadolinium diethylene-triamine penta-acetic acid (Schering; Berlin, Germany), SA and additional administration of 0.1 mmol/kg gadolinium diethylene-triamine penta-acetic acid (Schering; Berlin, Germany), SA and 4CH images were obtained with spin echo in 54 patients (slice thickness, 8 mm; gap, 0.8 mm; matrix, 512 \(\times\) 512; field of view, 380 mm; voxel size, 0.7 \(\times\) 0.7 \(\times\) 8 mm) and three-dimensional breath hold inversion recovery-gradient echo (IR-GRE) sequences (SA, VLA, 4CH) in 15 patients (slice thickness, 10 mm; no gap; matrix, 256 \(\times\) 256; field of view, 400 mm; voxel size, 1.6 \(\times\) 1.6 \(\times\) 10 mm) to assess for the presence of late gadolinium-enhancing (LGE) lesions. Fourteen patients underwent both spin echo and IR-GRE studies. The inversion time (250 to 400 ms) was determined on an individual basis to obtain optimal nulling of the unenhanced myocardial signal.

Regional differences in LV wall enhancement were measured and localized according to the 17-segment model.\(^1\) The total time required for the investigation was 45 to 60 min. The studies were independently evaluated by four blinded observers—three cardiologists and one radiologist—experienced in performing CMR. The study results were considered to be abnormal when at least two observers described identical abnormalities.

**Coronary Angiography**

Diagnostic coronary angiography (\(n = 13\)) was performed when considered indicated by the managing physician, and was generally done in patients with symptoms and findings suggestive of coronary artery disease.

**Statistical Analysis**

All statistical analyses were performed using statistical software (SSPS version 11.5; SPSS; Chicago, IL). Group data are expressed as mean \(\pm\) SD. Continuous variables were assessed using the Student unpaired \(t\) test for independent samples or the Mann-Whitney test were appropriate, and all categorical variables were assessed using the \(\chi^2\) test. Statistical significance was defined as \(p < 0.05\).

**Results**

**Findings at Standard Evaluation**

Standard assessment diagnosed CS in 13 patients. The demographic and clinical characteristics of the patients are presented in Table 1. During a mean

<table>
<thead>
<tr>
<th>Variables</th>
<th>CS by Standard Assessment (Group A, (n = 13))</th>
<th>CS Only by CMR (Group C, (n = 6))</th>
<th>(p) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, yr</td>
<td>49 (\pm) 6</td>
<td>50 (\pm) 10</td>
<td>NS</td>
</tr>
<tr>
<td>Male/female gender</td>
<td>9 (69)/4 (31)</td>
<td>4 (67)/2 (33)</td>
<td>NS</td>
</tr>
<tr>
<td>Time since diagnosis, yr</td>
<td>9 (\pm) 8</td>
<td>5 (\pm) 2</td>
<td>NS</td>
</tr>
<tr>
<td>Follow-up since cardiac assess, mo</td>
<td>18 (\pm) 10</td>
<td>21 (\pm) 7</td>
<td>NS</td>
</tr>
<tr>
<td>Modified New York Heart Association functional class 0</td>
<td>7/5/0/0/1</td>
<td>6/0/0/0/0</td>
<td>NS</td>
</tr>
<tr>
<td>Pulmonary stage 0/ I/II/III/IV, No.</td>
<td>0/3/4/6/0</td>
<td>1/3/0/1/1</td>
<td>NS</td>
</tr>
<tr>
<td>Palpitations</td>
<td>4 (31)</td>
<td>4 (67)</td>
<td>NS</td>
</tr>
<tr>
<td>Maximal serum angiotensin-converting enzyme, IU/L</td>
<td>49 (\pm) 40</td>
<td>31 (\pm) 12</td>
<td>NS</td>
</tr>
<tr>
<td>LV EF, %</td>
<td>45 (\pm) 17</td>
<td>60 (\pm) 6</td>
<td>NS</td>
</tr>
<tr>
<td>Patients with late enhancement, No.</td>
<td>11</td>
<td>6</td>
<td>NS</td>
</tr>
<tr>
<td>Segments per patient, No.</td>
<td>4.1 (\pm) 4.2</td>
<td>2.5 (\pm) 1.9</td>
<td>NS</td>
</tr>
<tr>
<td>Range</td>
<td>0–12</td>
<td>1–6</td>
<td></td>
</tr>
<tr>
<td>Localization of gadolinium enhancement</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basal segments (1–6)</td>
<td>30 (57)</td>
<td>10 (67)</td>
<td>NS</td>
</tr>
<tr>
<td>Equatorial segments (7–12)</td>
<td>19 (36)</td>
<td>5 (33)</td>
<td>NS</td>
</tr>
<tr>
<td>Apical segments (13–17)</td>
<td>4 (7)</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Transmural distribution of gadolinium enhancement</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subendocardial layer</td>
<td>9 (82)</td>
<td>6 (100)</td>
<td>NS</td>
</tr>
<tr>
<td>Middle layer</td>
<td>9 (82)</td>
<td>5 (67)</td>
<td>NS</td>
</tr>
<tr>
<td>Subepicardial layer</td>
<td>9 (82)</td>
<td>2 (33)</td>
<td>NS</td>
</tr>
<tr>
<td>All three layers</td>
<td>9 (82)</td>
<td>2 (33)</td>
<td>NS</td>
</tr>
<tr>
<td>Confluent transmural</td>
<td>3 (27)</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Regional wall motion abnormalities, No.</td>
<td>7</td>
<td>0</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Loss of wall thickness &lt; 7 mm, No.</td>
<td>2</td>
<td>0</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Data are presented as mean \(\pm\) SD or No. (%) unless otherwise indicated. NS = not significant.

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**Table 1—Summary of the Clinical and Demographic Patient Characteristics and Findings With CMR**

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follow-up of 19 ± 7 months, of the patients with CS diagnosed by standard assessment, one patient died suddenly, two patients received a DDD-R pacemaker because of third-degree atrioventricular block, and four patients received a pacemaker/implantable cardioverter defibrillator because of ventricular tachyarrhythmias. The follow-up of patients with CS diagnosed solely because of CMR abnormalities (group C) was uneventful.

Findings With CMR

The findings with CMR are presented in Table 1. In 37 studies (67%), four of four observers agreed on the absence or the presence and localization of late enhancement; in 12 studies (22%), three of four observers agreed; and in 6 studies (11%), two of four observers agreed. LGE lesions were present in 17 patients. Seven of 17 patients underwent evaluation with SE, 9 patients underwent evaluation with spin echo and IR-GRE, and 1 patient underwent IR-GRE only. In 6 of the patients undergoing spin echo, two observers agreed on the presence and localization of LGE; in one of the patients undergoing spin echo, three observers agreed; in 4 of the 10 patients who underwent both spin echo and IR-GRE or only IR-GRE, three observers agreed; and in the remaining 6 patients, all observers agreed on the presence and localization of LGE.

In 19 patients (group B), including all patients with a diagnosis made by standard assessment (group A), the CMR examinations showed abnormalities that were suggestive of CS. In six patients, late enhancement involved 1 to 6 segments (mean, 2.5 ± 1.9 segments), one of whom also had impaired systolic LV function (LVEF 52%) diagnosed by CMR, while standard assessment was normal (Figs 1–4).

The amount of myocardial enhancement in group A did not significantly differ from group C. The transmural extent of enhancement and the number of enhancing segments both showed a negative correlation with LV function (p < 0.05).

The presence of late enhancement correlated with a primary cardiac presentation (p < 0.001) and the duration of disease (p < 0.05). The presence of myocardial enhancement did not correlate with the extent of pulmonary disease. The bull’s-eye representation reflecting the distribution of LV enhancement is shown in Figure 1. Late enhancement predominantly involved the basal segments of the left ventricle (59%), with the anteroseptal and anterolateral segments most affected (43%) [Figs 5–7].

Correlation of Standard Evaluation With CMR Findings

Figure 2 illustrates the correlations between the different investigations and CMR.

ECG

Resting ECG findings are presented in Table 2. The presence of regional wall motion abnormalities (p < 0.001), impaired LV function (p < 0.001), and the segmental extent (p < 0.05) of late enhancement all correlated with the ECG abnormalities. The transmural extent and distribution of late enhancement did not determine the presence of conduction abnormalities on the ECG. Transmural enhancement (p < 0.001) and segmental extent of enhancement (p < 0.05) correlated with the presence of Q waves.

The presence of impaired LV function (p < 0.05) and regional wall motion abnormalities (p < 0.001) correlated with the abnormalities found with ambu-
latory ECG mentioned in Table 2. The extent of segmental enhancement (p < 0.001) and transmural enhancement correlated with the presence of nonsustained ventricular arrhythmias.

**Doppler Echocardiography**

Doppler echocardiographic findings are presented in Table 3. The segmental and transmural extent of enhancement correlated with regional wall motion abnormalities (p < 0.05), mitral regurgitation (p < 0.001), LV dimensions (p < 0.05), and impaired diastolic and systolic LV and right ventricular function (p < 0.05).

**Thallium Scintigraphy**

$^{201}$Tl scintigraphy demonstrated reversed distribution or irreversible perfusion defects in six patients. The presence of perfusion defects correlated with
late enhancement and impaired systolic LV function (p < 0.05). The localization of perfusion defects and late enhancement matched in four of the six patients. In one patient, reversed distribution was present in the apex, while ECG, echocardiography, and CMR findings were normal.

**Figure 3.** CMR study (spin echo sequence, SA view) demonstrates late enhancement (arrows; segments 1, 4, and 5) in a patient in whom a standard assessment did not reveal abnormalities.

**Figure 4.** CMR study (spin echo sequence, SA view) demonstrates late enhancement (arrows; segments 7 and 12) in a patient in whom standard assessment did not initially reveal abnormalities.

**Figure 5.** CMR study. *Left:* Three-dimensional breath-hold inversion recovery, fast IR-GRE (SA view). *Right:* SSFP (4CH view) demonstrates loss of wall thickness and late enhancement (segments 11 and 12) in a patient who survived out-of-hospital cardiac arrest secondary to ventricular fibrillation. Arrowheads (*left*) indicate loss of wall thickness. Arrow (*right*) demonstrates late enhancement.

**Figure 6.** CMR study (spin echo sequence, SA view) demonstrates LV dilation and late enhancement (arrow; segment 6) in a patient who presented with heart failure and left bundle-branch block.

**Discussion**

The diagnostic potential of CMR for detecting CS has been demonstrated by several case reports and single-center patient series. Patel et al. assessed 58 mainly African-American sarcoidosis patients without cardiac symptoms and reported a twofold-higher rate of cardiac involvement with gadolinium-enhanced CMR compared to evaluation with ECG and echocardiography. Sköld et al. reported on 18 subsequent patients with pulmonary
sarcoidosis who had been systematically evaluated for cardiac involvement with ECG, Doppler echocardiography, and CMR.

Our study is the first to evaluate the value of comprehensive CMR assessment in addition to resting and ambulatory ECGs, Doppler echocardiography, and 201Tl scintigraphy. We found CMR to be highly sensitive in detecting abnormalities that suggest cardiac involvement in patients with histologically confirmed pulmonary sarcoidosis. Our findings demonstrate a strong correlation between ECG and echocardiographic abnormalities and the amount of LV enhancement with CMR. The distribution of enhancement correlates well with the localization of inflammation and fibrosis reported in postmortem studies of patients with CS.4,5

Limited, nontransmural, or patchy myocardial scar tissue—frequently found at postmortem evaluation of CS patients—may remain undetected by ECG, ultrasound, or scintigraphy but can be detected by CMR due to its high image resolution and IR-GRE sequences that increase signal intensity differences by almost 500%.13,30 The additional value of CMR to standard assessment was demonstrated in six patients (11%) in whom LV involvement was only diagnosed by CMR. Since disease in this group was of shorter duration in comparison to the group with a diagnosis by standard assessment, and myocardial involvement was limited, the findings in these patients may well reflect early involvement. The follow-up of this group, though uneventful, may be too short (21 ± 7 months) to draw firm conclusions concerning the prognostic relevance of the CMR findings.

ECG is considered an appropriate screening test for CS; however, in 25% of patients with gross cardiac infiltration at postmortem assessment, ECG abnormalities had not been present during life.4,31,32 Ambulatory ECG is reported to have a sensitivity of 67% and specificity of 80% for the diagnosis of CS.33 Abnormalities on two-dimensional echocardiograms, increase or loss of wall thickness, ventricular dilation, functional impairment, mitral regurgitation, or the presence of pericardial effusions have been detected in 14 to 41% of patients with sarcoidosis, even in the absence of ECG abnormalities and clinical symptoms.34–39 Impaired diastolic function was found in 14 to 56% of patients.27,37 In our study population, unexplained diastolic relaxation abnormalities were present in 19 patients (35%) without evidence of cardiac involvement. CMR did not re-

Table 2—Summary of ECG Findings*

<table>
<thead>
<tr>
<th>Variables</th>
<th>CS by Standard Assessment (Group A, n = 13)</th>
<th>CS Only by CMR (Group C, n = 6)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Twelve-lead ECG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>1 (7%)</td>
<td>6 (100%)</td>
<td>&gt; 0.001</td>
</tr>
<tr>
<td>Abnormal</td>
<td>12</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Left bundle-branch block</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Right bundle-branch block</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Bifascicular block</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Left anterior fascicular block</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Grade III atrioventricular block</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Pseudoinfarct pattern</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>ST-T segment abnormalities</td>
<td>3</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Ambulatory ECG</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Abnormal</td>
<td>6 (46%)</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>PVC &gt; 100/24 h</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Nonsustained VT</td>
<td>3</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Sustained VT</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Paroxysmal atrial fibrillation</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Atrioventricular nodal reentrant tachycardia</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

*Data are presented as No. (%) or No. See Table 1 for expansion of abbreviation.
veal scar tissue in this group and, unlike a previous study, its presence did not correlate with disease duration. 201Tl scintigraphy has been most extensively studied and revealed perfusion defects diagnostic of myocardial fibrosis or granulomatous infiltration in 13 to 32% of sarcoidosis patients. Both ECG and echocardiography have demonstrated prognostic value in patients with CS. The presence of sustained VTs, New York Heart Association functional class, and LV dilation were found to be strong independent predictors of mortality.

The correlation between the presence of nonsustained and sustained VTs on Holter monitoring and the extent of LV enhancement with CMR in sarcoidosis has not been reported before analog three-channel Holter recorder with amplitude modulation at a speed of 1 mm/s; GE Medical Systems; Antwerp, Belgium). Tapes were analyzed on a digital system (Marquette 8000; Marquette; Antwerp, Belgium). Myocardial scar tissue is considered to be the substrate for ventricular tachyarrhythmia due to electrical reentry. In our study population, the presence of VTs did not correlate with substrate in a particular myocardial layer, since delayed enhancement involved parts of all three myocardial layers in most patients. Further study is needed to elucidate the diagnostic and prognostic relevance of CMR in the management of this condition.

**CONCLUSIONS**

CMR is a useful technique for the assessment of cardiac involvement in sarcoidosis, since the findings correlated strongly with standard assessment and the diagnosis of CS according to accepted guidelines. CMR may be able to demonstrate disease at an early stage, when standard assessment fails to do so. In addition, extent of late enhancement with gadolinium correlated with ventricular dilatation, functional impairment, and the presence of ventricular arrhythmias, all known markers of sudden death. CMR-based early detection and monitoring of CS may represent a useful strategy to determine the optimal timing of medical intervention.

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