Utility of Cardiovascular Magnetic Resonance in Identifying Substrate for Malignant Ventricular Arrhythmias

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Background—Sudden cardiac death (SCD) and sustained monomorphic ventricular tachycardia (SMVT) are frequently associated with prior or acute myocardial injury. Cardiovascular magnetic resonance (CMR) provides morphological, functional, and tissue characterization in a single setting. We sought to evaluate the diagnostic yield of CMR-based imaging versus non–CMR-based imaging in patients with resuscitated SCD or SMVT.

Methods and Results—Eighty-two patients with resuscitated SCD or SMVT underwent routine non-CMR imaging, followed by a CMR protocol with comprehensive tissue characterization. Clinical reports of non-CMR imaging studies were blindly adjudicated and used to assign each patient to 1 of 7 diagnostic categories. CMR imaging was blindly interpreted using a standardized algorithm used to assign a patient diagnosis category in a similar fashion. The diagnostic yield of CMR-based and non–CMR-based imaging, as well as the impact of the former on diagnosis reclassification, was established. Relevant myocardial disease was identified in 51% of patients using non–CMR-based imaging and in 74% using CMR-based imaging (P < 0.002). Forty-one patients (50%) were reassigned to a new or alternate diagnosis using CMR-based imaging, including 15 (18%) with unsuspected acute myocardial injury. Twenty patients (24%) had no abnormality by non-CMR imaging but showed clinically relevant myocardial disease by CMR imaging.

Conclusions—CMR-based imaging provides a robust diagnostic yield in patients presenting with resuscitated SCD or SMVT and incrementally identifies clinically unsuspected acute myocardial injury. When compared with non–CMR-based imaging, a new or alternate myocardial disease process may be identified in half of these patients. (Circ Cardiovasc Imaging. 2012;5:12-20.)

Key Words: MRI • arrhythmia • sudden cardiac death • diagnosis

Sudden cardiac death (SCD) accounts for 5% to 6% of annual deaths in North America.1,2 Many of these events are attributable to malignant ventricular arrhythmia, in which survivors may be at risk of clinical recurrence.1–5 Efforts to identify arrhythmia precipitants in these patients lead to multiple imaging investigations aimed at the identification of myocardial disease, both acquired and inherited, and the exclusion of obstructive coronary artery disease (CAD).5–8 The diagnostic yield and related impact of imaging on ultimate clinical diagnosis in this population has not been previously examined.

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Current clinical guidelines recommend the routine performance of transthoracic echocardiography and coronary imaging in patients presenting with malignant ventricular arrhythmias.5–8 Additional imaging tests, such as cardiovascular magnetic resonance (CMR) imaging, are recommended to be performed at the discretion of the physician.7,8 However, the spectrum and prevalence of diseases identified by CMR in this population have not been established. Therefore, uncertainty exists regarding the clinical role of CMR in this setting.

CMR tissue characterization imaging incorporates an evaluation of irreversible tissue injury using delayed-enhancement (DE) imaging and the identification of current or recent myocardial injury using T2-weighted (“edema”) imaging. Through a combination of these established and validated techniques, distinct patterns of acute or chronic injury may be identified.9–16 The pattern and distribution of this injury offer reliable identification of disease etiology,9 whereas the extent of irreversible tissue injury has been associated with future arrhythmia risk in ischemic17–20 and nonischemic21–26 cohorts. In addition, fatty replacement of myocardium can be identified through the use of T1-weighted imaging, a tool commonly used as part of CMR protocols for the detection of...
arrhythmogenic right ventricular cardiomyopathy (ARVC),27–29 The combination of these 3 tissue imaging techniques with CMR cine imaging, a gold standard for characterization of cardiac morphological features and function, offers a potentially robust tool set for the identification of arrhythmogenic substrate.

In this study, we evaluate the diagnostic yield of routine CMR imaging with a comprehensive tissue characterization protocol in patients presenting with resuscitated SCD or sustained monomorphic ventricular tachycardia (SMVT).

**Methods**

**Patient Population**

A total of 125 patients referred to the Electrophysiology Service between September 2007 and December 2009 with resuscitated SCD or symptomatic SMVT were screened. SCD was defined as death occurring within 1 hour of symptom onset. SMVT was defined as ventricular tachycardia lasting for ≥30 s. Patients experiencing an overt ischemic event during or up to 30 days before presentation (N=14) were excluded during the enrollment period, because cause was considered to be clinically established. Acute coronary syndrome was defined as chest pain or ischemic ST-segment changes (elevation or depression ≥1 mm in ≥2 contiguous leads) combined with an elevation of the serum troponin level. The threshold used for troponin elevation was ≥3 and ≥10 times the upper reference value for those without and with those with resuscitated SCD, respectively. Patients with standard contraindications to contrast-enhanced magnetic resonance imaging (MRI) were excluded, including severe claustrophobia (n=1), a previously implanted cardiac device (n=20), and a glomerular filtration rate ≤30 mmol/L per 1.73 m² (n=8). A total of 82 patients (66% of the screened population) met all inclusion and exclusion criteria and formed the study population.

All patients provided written informed consent. The study protocol was approved by the local institution’s Health Sciences Research Ethics Board.

**Study Design**

The study was designed as a prospective cohort study aimed at evaluating CMR’s diagnostic yield and related impact on ultimate diagnosis category in patients presenting with resuscitated SCD or SMVT. Physicians ordered routine, non–CMR-based imaging tests according to standard clinical practice. After all tests were ordered, a CMR study was performed based on the earliest clinically feasible opportunity. A comparison of diagnostic yield and final patient diagnosis category was performed between blinded evaluations of non-CMR and CMR imaging.

**Non-CMR Imaging: Diagnostic Yield and Diagnosis Category Assignment**

All patients underwent routine non-CMR imaging as clinically directed by the patient’s most responsible physician. The presence of clinically relevant findings from these investigations was determined through adjudication of all clinical reports by an experienced cardiologist blinded to CMR findings. The investigator was provided with a summary of the clinical presentation and relevant electrocardiographic findings to provide clinical context. The investigator assigned each patient to 1 of 7 diagnosis categories based on the combined non-CMR imaging findings, as follows: (1) no known cause (normal), (2) CAD with no left ventricular (LV) dysfunction, (3) CAD with LV dysfunction, (4) unexplained (idiopathic) LV dysfunction, (5) hypertrophic cardiomyopathy (HCM), (6) inflammatory cardiomyopathy, and (7) other. CAD was defined as the presence of ≥1 epicardial coronary artery with ≥70% lumen stenosis detected by invasive angiography or cardiac computed tomography. HCM, acute myocarditis, and ARVC were accepted as being present if combined imaging findings were consistent with published guidelines.15,30,31

**CMR Imaging Protocol**

Patients underwent scanning using a 3-T MRI scanner (N=52, TRIO or Verio, Siemens Medical Systems; Erlangen, Germany) or a 1.5-T scanner (N=28, Avanto, Siemens Medical Systems). All patients underwent the same standardized CMR protocol, inclusive of cine imaging, T2-weighted imaging, and DE imaging. T1-weighted imaging was incrementally performed in patients referred for resuscitated SCD or SMVT arising from the right ventricle with left bundle branch block–like morphological characteristics.

Cine imaging was performed using an Steady State Free Precession-based pulse sequence in sequential short-axis slices from the atrioregional annulus to the apex at 10-mm intervals and in the 4-, 3-, and 2-chamber orientations (typical parameters: slice thickness, 6 mm; gap, 4 mm; TE, 1.5 ms; TR, 3.0 ms). T2-weighted imaging was performed in sequential short-axis orientations at 20-mm intervals using a triple-inversion recovery fast-spin echo pulse sequence performed with and without the use of a chest surface coil (slice thickness, 10 mm; gap, 10 mm; TE, 87 ms [1.5 T] and 55 ms [3.0 T]). T1-weighted imaging was performed using a double-inversion recovery fast-spin echo-pulse sequence in sequential axial orientations from lower than the inferior margin of the right ventricle to the pulmonary artery bifurcation (slice thickness, 5 mm; gap, 5 mm; TE, 27 ms). Ten minutes after administration of 0.2 mmol/kg gadolinium (Magnevist or Gadovist, Bayer Inc; Toronto, Canada), DE imaging was performed in slice orientations matching cine imaging using a standard inversion recovery gradient-echo pulse sequence (matrix, 256×192; slice thickness, 6 mm; gap, 4 mm) with manual adjustment of the inversion time, as previously described.32 For patients with frequent ventricular ectopy, a single-shot, Steady State Free Precession-based inversion recovery–DE pulse sequence was used.

**CMR Imaging: Diagnostic Yield and Diagnosis Category Assignment**

All CMR studies were deidentified and blindly evaluated by an experienced interpreter. Cine images were visually evaluated for presence of regional or global left and right ventricular systolic dysfunction. Semiautomated quantitative software (CMR42, Circle International; Calgary, Alberta, Canada) was used to obtain the LV end-diastolic volume, LV end-systolic volume, and ejection fraction (EF) of both the left and right ventricles using sequential short-axis cine images.

T2-weighted images were visually scored for the presence of nonhomogeneous myocardial signal with regional enhancement. If believed to be present, matched images taken without the surface coil were assessed using quantitative signal analysis, as previously described.18 Traced myocardial segments achieving a mean signal intensity >1.9-fold higher than that of reference skeletal muscle were accepted as being abnormal, in accordance with the Lake Louise Consensus Criteria.15

DE images were visually scored for the presence of abnormal hyperenhancement (HE). The dominant pattern of HE (used for scoring of diagnosis category) and any or all secondary patterns of HE were recorded, as follows: (1) subendocardial based (CAD-like), (2) midwall patchy, (3) midwall striae, (4) subepicardial, and (5) diffuse, as previously described.33 Any abnormal HE of the right ventricle, when identified, was also recorded.

T1-weighted images were visually scored for the presence of an abnormal intramyocardial fat signal of either the right or left ventricular myocardium, as previously described.28 After a review of all CMR images, the investigator assigned each patient to 1 of the same 7 diagnosis categories, as previously described. Similarly, the investigator was provided a summary of the clinical presentation and relevant electrocardiographic findings to provide clinical context. For CMR-based imaging, CAD was considered present if subendocardial-based HE was identified in a coronary artery distribution. Standardized CMR-based diagnostic criteria of nonischemic cardiomyopathies were used where available, including standardized criteria for the diagnosis of acute myocarditis14 and ARVC.34 Combined morphological and tissue characteriza-
Table 1. Baseline Patient Clinical and MRI Characteristics

<table>
<thead>
<tr>
<th>Category</th>
<th>All Patients (N=82)</th>
<th>SCD (n=30)</th>
<th>SMVT (n=52)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>52±15</td>
<td>46±14</td>
<td>55±14</td>
<td>0.05</td>
</tr>
<tr>
<td>Female sex</td>
<td>27 (33)</td>
<td>9 (30)</td>
<td>18 (35)</td>
<td>0.81</td>
</tr>
<tr>
<td>Hypertension</td>
<td>27 (33)</td>
<td>7 (23)</td>
<td>20 (38)</td>
<td>0.28</td>
</tr>
<tr>
<td>Hypertension</td>
<td>24 (29)</td>
<td>7 (23)</td>
<td>17 (33)</td>
<td>0.54</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>10 (12)</td>
<td>3 (10)</td>
<td>7 (14)</td>
<td>0.23</td>
</tr>
<tr>
<td>Current smoker</td>
<td>19 (23)</td>
<td>10 (33)</td>
<td>9 (17)</td>
<td>0.59</td>
</tr>
<tr>
<td>History of MI</td>
<td>21 (26)</td>
<td>6 (20)</td>
<td>15 (29)</td>
<td>0.55</td>
</tr>
<tr>
<td>Any prior revascularization</td>
<td>12 (15)</td>
<td>3 (10)</td>
<td>8 (15)</td>
<td>0.76</td>
</tr>
<tr>
<td>Non-CAD structural heart*</td>
<td>3 (4)</td>
<td>0</td>
<td>3 (6)</td>
<td>0.41</td>
</tr>
<tr>
<td>Cardiac MRI parameters</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV mass index, g/m²</td>
<td>72±23</td>
<td>77±21</td>
<td>69±23</td>
<td>0.50</td>
</tr>
<tr>
<td>LV EDV index, mL/m²</td>
<td>88±33</td>
<td>88±32</td>
<td>89±33</td>
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<tr>
<td>LV ESV index, mL/m²</td>
<td>48±33</td>
<td>45±26</td>
<td>48±34</td>
<td>0.99</td>
</tr>
<tr>
<td>LV EF, %</td>
<td>51±19</td>
<td>53±17</td>
<td>50±19</td>
<td>0.59</td>
</tr>
</tbody>
</table>

Continuous data are expressed as mean±SD, and categorical data are expressed as number (percentage).

EDV indicates end-diastolic volume; ESV, end-systolic volume; MI, myocardial infarction.

*Three patients had an established non-CAD diagnosis (hypertrophic cardiomyopathy, dilated cardiomyopathy, and tetralogy of Fallot).

Statistical Analyses were performed using a commercially available software program (GraphPad Prism, Version 5.0, La Jolla, CA). For comparisons between subgroups, differences in continuous variables were analyzed using the 2-sample t test, and differences in categorical variables were analyzed using the χ² test. All P values used were 2 sided, with P<0.05 considered significant.

Results

Eighty-two consecutive patients were enrolled. Thirty-one patients (38%) presented with resuscitated SCD, with 51 (62%) presenting with symptomatic SMVT. Baseline patient and MRI-derived characteristics are shown in Table 1. The age of the population was 52±15 years, with 27 (33%) being female. Of patients presenting with resuscitated SCD, an electrocardiographic rhythm strip of the presenting rhythm was available in 27, showing ventricular fibrillation in 22, SMVT (pulseless) in 3, and polymorphic VT in 2. A history of myocardial infarction was present in 21 patients (18%).

Non-CMR Imaging: Findings and Diagnosis Assignment

The type and frequency of non-CMR imaging testing performed by the most responsible physician is shown in Table 2. All patients (100%) underwent transthoracic echocardiography, with invasive coronary angiography performed in 90% of patients with SCD and 69% of patients with SMVT. Single-positron emission computed tomography perfusion imaging and cardiac computed tomographic imaging were performed for the exclusion of CAD in 5 and 2 patients, respectively.

The diagnostic yield of the various non-CMR imaging modalities is shown in Table 2. After blinded adjudication of all non-CMR imaging, a “normal” diagnosis category (ie, no abnormal findings) was assigned in 40 patients (49%). Figure 1 illustrates the rate of nonnormal diagnosis category assignments. “Unexplained LV dysfunction” was categorized in 18 (22%), “obstructive CAD with LV dysfunction” in 15 (18%),
“CAD without LV dysfunction” in 6 (7%), “HCM” in 1 (1%), and “other” in 2 (2%). In the latter category, 1 patient had findings consistent with takotsubo cardiomyopathy, with the other having findings typical for tetralogy of Fallot repair.

CMR Imaging Findings and Diagnosis Assignment

CMR imaging was successfully performed in all patients at a time from the documented arrhythmia of 12±7 days (range, 1–28 days) for resuscitated SCD and 21±19 days (range, 2–62 days) for SMVT. The LV EF was 51±19%, with 49 patients (60%) having an LV EF ≥50% (Table 1). No significant differences in LV volumes or EF were identified between those presenting with resuscitated SCD and those presenting with SMVT.

The prevalence of clinically relevant findings by CMR imaging was 74% and is listed according to MRI pulse sequence in Table 2. DE imaging was abnormal in 46 patients (56%) and demonstrated an isolated ischemic pattern of HE in 28 (34%) and a nonischemic pattern of HE in 26 (32%). A combination of ischemic and nonischemic HE was seen in 9 patients (11%). T2-weighted imaging demonstrated regional signal abnormalities in 14 patients (17%), all but 1 being associated with regionally matched HE. This HE was subendocardial in 5 patients (ie, consistent with acute myocardial infarction) and subepicardial in 9 patients (ie, consistent with acute myocarditis) (Figure 2). One patient had an isolated regional increase in T2 signal without HE (Figure 3). Of 14

![Figure 1. Relative distribution of diagnosis categories assigned to all patients before cardiovascular magnetic resonance (CMR) imaging (non-CMR diagnosis, left) and after CMR imaging (CMR diagnosis, right). CAD indicates coronary artery disease; HCM, hypertrophic cardiomyopathy; Inflam, inflammatory cardiomyopathy; LVD, left ventricular dysfunction.](image1)

![Figure 2. Example cases of cardiovascular magnetic resonance (CMR) leading to detection of otherwise unrecognized acute injury. Acute ischemic injury is seen in 2 patients who underwent echocardiography and coronary angiography, both described as normal. CMR shows abnormal delayed enhancement (DE) associated with increased signal on T2-weighted (T2w) imaging, consistent with acute injury. Acute inflammatory injury is seen in 2 patients with idiopathic LV dysfunction diagnosed by echocardiography with normal coronary angiography. CMR shows epicardial-based DE with associated increased signal on T2w imaging, consistent with an acute inflammatory process (ie, myocarditis). The white triangles indicate associated pericardial effusion.](image2)
patients (17%) demonstrating abnormal T2 and HE (ie, recent myocardial injury with myocyte necrosis), 7 (47%) had an elevation in the serum troponin I level (mean, 0.7 ng/mL; range, 0.4–1.0 ng/mL). In all 7 patients, invasive coronary imaging failed to demonstrate obstructive CAD.

Forty-two patients underwent T1-weighted fat imaging according to the prespecified CMR protocol (30 with resuscitated SCD and 12 with left bundle branch block-like SMVT) (Table 2). T1 imaging demonstrated abnormal intramyocardial fat in 3 patients (7%), all presenting with left bundle branch block-like SMVT. In all 3 of these patients, cine imaging of the RV was sufficiently abnormal to meet either minor (n=1) or major (n=2) ARVC task force MRI criteria.31 Abnormal RV HE was also seen in 1 of these patients (Figure 4). Abnormal HE of the RV was seen in 2 additional patients, as part of extensive biventricular HE.

Figure 3. A 57-year-old man with resuscitated sudden cardiac death (SCD) classified as idiopathic left ventricular (LV) dysfunction by non–cardiovascular magnetic resonance (CMR) imaging with septal hypokinesia by echo (LV ejection fraction [EF], 30%) and nonobstructive coronary artery disease (CAD) by invasive angiography (30%–40% proximal left anterior descending artery lesion). CMR imaging, performed 4 days after cardiac arrest, showed persistent hypokinesia of the septum (top left, systolic phase cine), an increased T2 signal throughout the same territory (bottom row, white arrows), and normal delayed enhancement imaging (top right), suggesting transient injury without significant necrosis. The peak troponin I increase was 0.06 ng/mL. Follow-up echocardiography at 3 months showed marked functional improvement, with an estimated LV EF of 50% to 55%.

Figure 4. Examples of right ventricular disease. Patient 1 was a 35-year-old man with unstable monomorphic ventricular tachycardia. Echocardiography and invasive catheterization results were both interpreted as normal. The cardiovascular magnetic resonance (CMR) image shows a focal right ventricular aneurysm (top) with associated transmural scar (bottom), consistent with arrhythmogenic right ventricular cardiomyopathy. Patient 2 was a 52-year-old man with ventricular tachycardia diagnosed as having idiopathic left ventricular (LV) dysfunction after echocardiography and catheterization. The CMR shows a right and left ventricular epicardial-based scar, consistent with cardiac sarcoid (confirmed by endomyocardial biopsy).
most consistent with chronic myocarditis (Figure 4). Cine imaging abnormalities were seen in the absence of any other CMR abnormality in 4 patients (5%), having right ventricular dysfunction consistent with ARVC (2 meeting minor and 1 meeting major task force MRI criteria) and 1 having LVNC.

The frequency and distribution of patient diagnosis categories assigned by CMR imaging are shown in Figure 1. Overall, CMR imaging identified substrate for ventricular arrhythmia in 61 patients (74%), including 20 (65%) of the 31 patients with SCD and 41 (80%) of the 51 patients with SMVT. CMR was categorized as normal in 21 patients (26%), unexplained LV dysfunction in 5 (6%), “CAD with LV dysfunction” in 29 (35%), “inflammatory cardiomyopathy” in 17 (21%), HCM in 1 (1%), and other in 9 (11%). Of patients categorized as having “inflammatory cardiomyopathy,” 3 were interpreted as being consistent with cardiac sarcoid. Of patients categorized as other, 6 were interpreted as being consistent with ARVC, 1 with LVNC, and 2 with midwall fibrosis of unknown cause.

**Impact of CMR Imaging on Diagnosis Category**

Compared with non-CMR imaging, a new or alternate myocardial disease process was identified by CMR imaging in 41 (49%) of 82 patients (Figure 5). Twenty patients (24%) categorized as normal by non-CMR imaging had relevant cardiovascular disease process identified by CMR imaging, inclusive of tissue characterization, has diagnostic utility for the detection of myocardial substrate in patients with malignant ventricular arrhythmia, particularly unsuspected acute injury, incremental to that provided by routinely ordered non-CMR imaging.

Current diagnostic algorithms for patients presenting with malignant ventricular arrhythmia recommend the routine performance of transthoracic echocardiography and invasive coronary angiography with the selective use of additional imaging, such as CMR. However, CMR’s identification of myocardial disease in this clinical setting has not been previously described. The results of this study suggest that CMR has a broad capacity to identify relevant, but clinically unsuspected, disease in patients with SCD or SMVT, such as acute myocarditis and acute ischemic injury. Because these conditions may be considered discreet or transient catalysts of ventricular arrhythmia, their detection may be of clinical importance.

The clinical adoption of CMR imaging in the tertiary care setting has expanded in response to physician awareness and improved clinical access. DE CMR imaging for the identification of irreversible myocardial injury has been extensively validated for the detection of such ischemic and nonischemic forms of myocardial fibrosis. A role for quantitative scar signal analysis for the prediction of future arrhythmic events in these populations has also emerged.

**Discussion**

To our knowledge, this study is the first to evaluate the diagnostic utility of cardiac imaging in patients presenting with resuscitated SCD or SMVT. Our results support that CMR imaging, inclusive of tissue characterization, has diagnostic utility for the detection of myocardial substrate in patients with malignant ventricular arrhythmia, particularly unsuspected acute injury, incremental to that provided by routinely ordered non-CMR imaging.
T1-weighted imaging detects abnormal intramyocardial fat in patients with ARVC, a condition closely associated with malignant arrhythmic events. In combination, these techniques provide a comprehensive evaluation for the detection of myocardial substrate for malignant ventricular arrhythmias.

In this study, acute myocardial injury was confirmed by T2-weighted imaging in one third of patients presenting with resuscitated SCD. The lower prevalence of acute injury patterns in the SMVT population (8%) may, in part, have been due to a longer median interval between arrhythmia occurrence and the completion of CMR testing (21 versus 12 days). When present, T2 abnormalities were typically associated with an ischemic HE pattern (subendocardial based) or an inflammatory HE pattern (subepicardial based), allowing for discrimination of injury mechanism. Of interest, in those with the former pattern, all demonstrated normal invasive coronary angiography and (by CMR) no evidence of cardiac thrombus to suggest an embolic cause. Four of these patients were returned to the cardiac catheterization suite for ergonovine provocation testing, which revealed regional coronary spasm within a related vascular territory in 3 of them.

A 50% improvement in the identification of relevant myocardial disease was observed through the use of CMR imaging in this population, leading to a robust 75% diagnostic yield. This increase was principally realized through more sensitive detection of acute and healed myocardial disease from both ischemic and nonischemic causes. The prevalence of such myocardial diseases in patients with SCD has been previously reported. In a large autopsy-based study of 453 patients dying from SCD, 123 (27%) demonstrated pathological findings of non–CAD-related structural heart disease: 38 had dilated cardiomyopathy/idioptic myocardial fibrosis, 39 had acute myocarditis, 28 had HCM, 10 had ARVC, and 6 had ischemic injury in the setting of normal coronary arteries, presumed to be due to coronary spasm. The results of the current study are, therefore, consistent with these prior pathological observations.

The identification of unsuspected acute myocardial injury in one third of patients presenting with SCD raises important considerations. Such injury provides a biologically plausible catalyst for the malignant ventricular arrhythmias experienced by these patients. Current clinical guidelines do not recommend the prescription of implantable cardiac defibrillators for patients experiencing unstable ventricular arrhythmias in the setting of acute myocardial injury.5 Future evaluation of arrhythmia recurrence in patients with CMR evidence of acute myocardial injury is, therefore, of particular interest and may present insights into the appropriate use of implantable defibrillator therapy in this population.

**Study Limitations**

This study was designed to evaluate the diagnostic utility of CMR imaging in patients with malignant ventricular arrhythmias. Although the impact of CMR findings on therapeutic decision-making and clinical outcomes is desirable, this was beyond the scope of the current study and should be a focus for future investigation.

The diagnostic yield of routine, non–CMR-based imaging in this study must be considered in recognition that, as a single-center study, it is inherently exposed to local practice bias. Tests that were clinically ordered may not reflect practice at other academic centers. Confirmation of these findings within a larger multicenter study is, therefore, recommended.

CMR imaging in this study, although on average being performed soon after clinical presentation, was delayed in some because of transfer from referring hospitals or other limitations related to clinical care. The sensitivity of T2-weighted imaging in this circumstance is expected to be reduced and may, therefore, have underrepresented its utility. However, these delays are expected at all centers and represent true clinical practice, making the results generalizable.

Two patients (2%) were misclassified as normal by CMR imaging but had CAD with normal LV function by non–CMR-based imaging. The interpretation of CAD was limited for CMR imaging to the visualization of “subendocardial-based HE in a coronary distribution” and, therefore, was unable to identify these 2 patients without any HE. This limitation may have been avoided through the incremental performance of either stress perfusion CMR or coronary magnetic resonance angiography, both having utility in this setting. However, the addition of such components to the described CMR protocol may be questioned with respect to widespread clinical feasibility.

Both 1.5- and 3-T MRI scanners were used for this study. A well-performed comparative study of T2-weighted imaging between these field strengths in acute cardiac injury has not been performed. Therefore, extrapolation of conventional interpretive approaches for T2 imaging at 3 T requires further validation.

Finally, relatively few patients underwent electrophysiological studies during their initial diagnostic workup. Therefore, the results of this testing were not incorporated into the study.

**Conclusions**

In conclusion, CMR imaging provides robust identification of myocardial disease in patients presenting with resuscitated SCD or SMVT. Compared with routine non–CMR-based imaging, CMR imaging incrementally identifies relevant substrate for malignant ventricular arrhythmias and leads to a reclassification of diagnosis category in half of the patients. These findings suggest that CMR imaging should be considered early in the diagnostic evaluation of patients presenting with malignant ventricular arrhythmia.

**Acknowledgments**

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Patients presenting with malignant ventricular arrhythmias outside of the setting of acute coronary syndrome pose a diagnostic and therapeutic challenge to the practicing clinician. One of the primary aims of cardiac imaging in this setting is the identification of underlying myocardial disease to offer plausible explanations for arrhythmia occurrence. However, the ability of diagnostic imaging to identify myocardial substrate for arrhythmia, the spectrum of such substrate that may be identified, and the incremental impact of these findings on diagnosis category are unknown. This study evaluated the diagnostic findings of routinely performed imaging other than cardiac magnetic resonance (CMR) versus comprehensive CMR to evaluate their respective diagnostic yield and impact on clinical diagnosis category in patients presenting with resuscitated sudden cardiac death (SCD) or sustained monomorphic ventricular tachycardia. In this population, a 50% increase in diagnostic yield of relevant myocardial disease was appreciated using CMR versus non-CMR imaging. This incremental yield translated into a new alternate diagnosis category being assigned in half of patients. For patients experiencing resuscitated SCD, approximately one third, by CMR, had unsuspected acute myocardial injury, offering identification of acute precipitants for otherwise unexplained cardiac arrest. In these patients, such findings may have incremental yield translated into a new alternate diagnosis category being assigned in half of patients. For patients experiencing resuscitated SCD, one third, by CMR, had unsuspected acute myocardial injury, offering identification of acute precipitants for otherwise unexplained cardiac arrest. In these patients, such findings may have important implications for therapeutic decision making and warrant further investigation. Overall, this study identifies that early CMR may be an efficient and high-yield diagnostic approach for the evaluation of patients presenting with malignant ventricular arrhythmias.
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