The Role of T1 and T2 Mapping on Cardiovascular Magnetic Resonance Imaging in Sudden Cardiac Arrest Survivors

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Abstract

Purpose

Etiology of sudden cardiac arrest (SCA) is identified in less than 30% of survivors without coronary artery disease. We sought to assess the diagnostic role of myocardial parametric mapping using cardiovascular magnetic resonance (CMR) in identifying SCA etiology.

Methods

Consecutive SCA survivors undergoing CMR with myocardial parametric mapping were included in the study. The determination if CMR was decisive or contributory in identifying SCA etiology was made if the diagnosis was unclear prior to CMR, and the discharge diagnosis was consistent with the CMR result. Parametric mapping was considered essential for the CMR diagnosis if the SCA etiology could have not been determined without its utilization, and contributory if the diagnosis could have been potentially based on the combination of cine and LGE imaging, without optimal assessment of the severity and prognosis of the disease (offered by parametric mapping).

Results

Of the 35 patients (mean age 46.9 ± 14.1 years; 57% males) included, diagnosis was based on CMR in 23 (66%) patients. Of those, parametric mapping was essential for the diagnosis of myocarditis and tako-tsubo cardiomyopathy (11/48%) and contributed to the diagnosis in 10 (43%) additional cases.

Conclusion

Inclusion of quantitative T1 and T2 parametric mapping in the SCA CMR protocol has the potential to increase diagnostic yield of CMR and further specify SCA etiology, especially myocarditis. CMR performed early after SCA may aid in the decision-making regarding ICD implantation.

Introduction

Despite best efforts, the etiology of sudden cardiac arrest (SCA) is identified in only 50% of survivors and in less than 30% of those without coronary artery disease (CAD).[1, 2]. SCA might be caused by acute and transient electrical instability in the setting of acute myocardial ischemia or myocarditis, ventricular arrhythmia due to presence of ischemic or nonischemic scar, or primary electrical disease[3–5]. Clinical investigations are aimed to establish the SCA cause and its reversibility to facilitate clinical decision making, including insertion of an implantable cardioverter-defibrillator (ICD)[3, 6].

While the role of T2-weighted imaging and late gadolinium enhancement (LGE) imaging in cardiac magnetic resonance imaging (CMR) in SCA survivors has been explored, there is limited data on the role of quantitative myocardial T1 and T2 relaxation times[2, 3, 7]. Myocardial parametric mapping can be used to identify myocardial inflammation, edema, and diffuse interstitial expansion, which can aid in diagnosing myocarditis and various inflammatory and non-ischemic cardiomyopathies[2, 8].

Quantitative T2 mapping overcomes several limitations of qualitative T2-weighted imaging and provides more accurate evaluation of myocardial edema[8, 9]. The combination of quantitative T2 mapping and LGE is crucial for the differentiation between acute and potentially reversible injuries, such as myocarditis or acute ischemia, and chronic irreversible injuries such as chronic infarct or nonischemic scar[3].

Assessment of interstitial myocardial fibrosis, quantified by extracellular volume fraction (ECV) via T1 mapping, might provide prognostic information in SCA survivors. ECV elevation is associated with adverse outcomes in patients with both ischemic and nonischemic LGE across a spectrum of left ventricular function[10–13]. Importantly, ECV has been shown to be more strongly associated with adverse outcomes than nonischemic LGE, even after adjusting for left ventricle ejection fraction (LVEF) [13]. Similar associations were found in patients with CAD, dilated cardiomyopathy, and heart failure with preserved ejection fraction[12, 14–16].

The goal of our study was to evaluate the diagnostic role of T1 and T2 mapping in identifying SCA etiology and personalizing management of SCA survivors. Preliminary assessment of the prognostic role of parametric mapping was also intended.

Materials & Methods

Patients

This is a retrospective cohort study assessing the role of myocardial T1 and T2 mapping in identifying SCA etiology and assessing prognosis. Patients who presented to our institution with SCA of unclear etiology between 2016 and 2019 and underwent CMR within 4 weeks of SCA were included in the study. Patients with an acute ischemic injury and known culprit coronary artery were not included in the analysis. Aborted SCA was defined as ventricular fibrillation or hemodynamically unstable ventricular tachycardia requiring electrical or chemical cardioversion. The study was approved by The Ohio State University’s Institutional Review Board and informed consent was waived.

The electronic medical record was reviewed for demographic and clinical data as well as cardiac testing including electrocardiography (ECG), transthoracic echocardiography (TTE), coronary computed tomography angiography (CCTA), and left heart catheterization (LHC). An ischemic evaluation using LHC and CCTA was available in 33 (94%) patients.

CMR Image Acquisition & Analysis

Clinical CMR images were acquired at 1.5 T (Magnetom Avanto, Siemens Healthineers, Germany) using standardized protocols including cine imaging, pre-contrast & post-contrast T1 mapping, T2 mapping, and LGE imaging[17].

CMR studies were anonymized and analyzed using Neosoft, LLC (Pewaukee, Wisconsin, United States of America). LV volumes and LVEF were measured from short-axis stacks of cine frames that covered the LV. Native and post-contrast myocardial T1 values were measured within the septum on the mid short axis (SAX) maps, and ECV was calculated using the standard formula[18]. T1 values were measured within the non-infarcted myocardium in cases with septal infarct scar. Myocardial T2 values were measured in all AHA segments on the mid SAX as well as long axis (vertical, horizontal and 3-chamber long axis) maps when available[17]. CMR reference values for the myocardium were based on institutionally established normative control data and were as follows: native T1 999 ± 31 ms, ECV 23.8 ± 2.6% and T2 56 ± 2 ms.

The presence, pattern, and extent of LGE was assessed by two level 3 trained CMR readers blinded to clinical information. LGE patterns were described as subendocardial, midwall, subepicardial, and transmural. Presence of LGE was reported according to the American Heart Association [AHA] 17-segment model[19].

The determination if CMR was decisive or contributory in identifying SCA etiology was made if the diagnosis was unclear prior to CMR, and the discharge diagnosis was consistent with the CMR result. Final SCA etiology was obtained from chart review and CMR analysis with confirmation by 2 cardiologists with adjudication by a 3rd author if in disagreement. Myocarditis on CMR was diagnosed using the updated Lake Louise criteria[20]. We followed the current criteria for defining hypertrophic cardiomyopathy, nonischemic cardiomyopathy, ischemic cardiomyopathy, and takotsubo cardiomyopathy, as published elsewhere[21–25].

Parametric mapping was considered essential for the CMR diagnosis if the SCA etiology could have not been determined without its utilization, and contributory if the diagnosis could have been potentially based on the combination of cine and LGE imaging, without optimal assessment of the severity and prognosis of the disease (offered by parametric mapping).

Follow-Up & Outcomes

Patient follow-up was performed by review of the vital status and electronic medical record including ICD interrogation and event monitor results. Follow-up duration was calculated from the date of SCA. The primary outcomes were all-cause mortality or heart transplant, and a composite arrhythmic outcomes including sudden death, VF, sustained VT, and appropriate ICD intervention.

Statistical Analysis

Categorical data are presented as frequency with percentage, and comparison between groups was performed using the chi-square or Fisher's exact test. Continuous variables were presented as mean ±

standard deviation (SD) for normal distributions, and median with 25th and 75th percentiles (Q1-Q3) for non-normal distributions. Normality was tested using skewness, kurtosis, visual inspection of the histogram, QQ plot, and Shapiro-Wilk test. Continuous variable comparisons between groups were performed using the Student's t-test or the Mann-Whitney U test as appropriate. Kaplan-Meier survival curves were drawn to assess differences between groups for time to event data. Time zero was defined as the date of CMR study. A Cox regression model was used to assess the relationship of T1 elevation, T2 elevation, LGE presence, and ECV elevation with clinical outcomes. The hazard ratio and incidence rate ratio were presented as mean and 95% confidence interval. A two-sided p value of < 0.05 was considered statistically significant. The statistical analyses were performed using IBM SPSS Statistics for Windows, version 22.0 (IBM Corp., Armonk, N.Y., USA) and R software, version 4.0.3 (The R Foundation, Vienna, Austria).

Results

Baseline Characteristics

A total of 35 patients were included in the analysis (Fig. 1). The mean age of the cohort was 46.9 ± 14.1 years, 57% male (Table 1). The most common primary arrhythmia was ventricular fibrillation/ventricular tachycardia in 33 patients (94%) followed by pulseless electrical activity in 2 patients (6%). CMR was performed within a median 5.5 (IQR: 2-7.5) days after the SCA.

CMR results

CMR findings are summarized in Table 1 for the whole cohort as well as are stratified by the primary outcome of all-cause mortality, heart transplant and arrhythmic outcomes, and detailed CMR data for each patient are presented in Supplemental Table 1. Elevation of T1 was seen in 18 (51%) patients (mean T1 1069 ± 60 ms), ECV in 16 (46%) patients (mean ECV 30 ± 7%), and T2 in 22 (63%) patients (mean T2 65 ± 10 ms). T2 was most frequently elevated in the mid inferolateral wall (12/34%) Fig. 2). LGE was present in 32 (91%) patients with a median of 5 (IQR 4–8) left ventricular segments affected (Table 1, Supplemental Table 1). The prevalence of LGE was highest in the basal (66%) to mid inferoseptal wall (63%).

Clinical impact of CMR

SCA etiology was established based on clinical data, ECG, TTE, CCTA, and LHC in 8 (23%) patients (Fig. 1, Table 1). CMR provided the most probable SCA etiology in an additional 23 (66%) patients with parametric mapping abnormalities in 21 (60%). Myocarditis (10), hypertrophic cardiomyopathy (4), nonischemic cardiomyopathy (5), ischemic cardiomyopathy (3), and takotsubo cardiomyopathy (1) were found among the 23 patients whose diagnoses were determined by CMR (Table 1, Supplemental Table 1).

Parametric mapping was essential for the diagnosis of myocarditis and takotsubo cardiomyopathy in 11 patients and contributed to the diagnosis in an additional 10 patients among the 23 cases with diagnoses determined by CMR. The etiology of SCA remained unknown in 4(11%) patients despite extensive testing including CMR with parametric mapping.

Parametric Mapping in Myocarditis

In the 10 myocarditis patients, T2 signal elevation (mean highest T2 of 73 ± 12 ms) and LGE were most commonly seen in the inferolateral wall consistent with prior reports (Table 1, Supplemental Table 1, Fig. 2)[20]. T1 was elevated in 6 patients (mean T1, 1084 ± 56 ms), ECV in 3 patients (mean ECV, 28.1 ± 5.6%), and left ventricular ejection fraction (LVEF) was depressed (< 50%) in 3 patients. Diagnosis was confirmed with an endomyocardial biopsy in 1 patient. One patient had influenza infection; 8 had troponin elevation (0.76, IQR 0.19–15.5, normal < 0.11 ng/L).

Parametric Mapping and in Takotsubo Cardiomyopathy

T1 (1044 ms) and ECV (28.1%) were normal in the tako-tsubo cardiomyopathy patient with myocardial edema/inflammation in the mid and apical segments (mean T2, 77 ms) that extended beyond the septal midmyocardial LGE.

Parametric Mapping in Other Etiologies of SCA

In the 4 patients with hypertrophic cardiomyopathy, native T1 was elevated in 1, and ECV in 2 patients. Two patients demonstrated T2 signal elevation, with LGE most commonly seen in the septum (Table 1, Supplemental Table 1).

Native T1 elevation was observed in 4 out of 5 patients with non-ischemic cardiomyopathy, with ECV and T2 being borderline abnormal in 1 patient. Septal and inferolateral walls were the most common locations of nonischemic LGE.

CMR based diagnosis of ischemic cardiomyopathy, was associated with native T1 and ECV elevation in 2/3 patients, whereas T2 was elevated in all 3/3 patients. Infarct scar in 2/3 patients corresponded to segments with T2 elevation and extended to other neighboring segments with no evidence of myocardial edema/inflammation.

Native T1 was elevated in 2 of 3 patients, and ECV in all 3 patients whose diagnosis of ischemic cardiomyopathy was not based on CMR. CMR of 3/3 patients was noticeable for an infarct scar that corresponded to segments with T2 elevation and extended to other neighboring segments with no evidence of myocardial edema/inflammation.

Two out of 5 patients with the primarily electric etiology of SCA (hypokalemia with QT prolongation, Brugada syndrome) presented with T1 and T2 elevation. Myocardial edema/inflammation, when present, was demonstrated in 1–2 AHA segments. ECV elevation was observed in 1 of 5 patients.

Among 4 patients with unclear SCA etiology, native T1 was elevated in 1 patient with ECV elevation in 3 patients. There was no evidence of T2 elevation.

ICD Implantation

ICD was implanted in 29 (83%) patients. ICD was not implanted in 4 (11%) patients due to reversible SCA cause, whereas 2 (6%) patients refused ICD implantation.

Primary Outcomes

Three (9%) patients were lost to follow-up. Over the median follow-up of 33.1 months (IQR 25.4–43.1) from SCA, 5 (16%) patients died or underwent a heart transplant, and 12 (38%) met the composite arrhythmic outcome (Supplemental Table 2). No significant differences in baseline characteristics, comorbidities, SCA etiology & mechanism as well as CMR parameters were demonstrated between patients that met and did not meet both primary outcomes (Table 1, Supplemental Fig. 2–3). There was no significant association between T1 elevation and all-cause mortality or heart transplant (HR: 3.25, 95% 0.36–29.7, p = 0.30), or arrhythmic outcomes (HR: 1.37, 95% CI: 0.41–4.62, p = 0.61); T2 elevation and all-cause mortality or heart transplant (HR: 1.84, 95% CI:0.19–17.7, p = 0.60), or arrhythmic outcomes (HR: 0.79, 95% CI: 0.24–2.60, p = 0.70); ECV elevation and all-cause mortality or heart transplant (HR: 0.84, 95% CI: 0.14–5.01, p = 0.84), or arrhythmic outcomes (HR: 1.23, 95% CI: 0.39–3.86, p = 0.72); (Supplemental Fig. 1).

Discussion

We investigated the diagnostic role of myocardial parametric mapping in SCA survivors. Our data confirms and extends previous observations that CMR with parametric mapping has diagnostic value in assessing SCA etiology compared to non-CMR based evaluation[3, 4]. We show that parametric mapping was essential for the diagnosis in 11 patients (myocarditis and tako-tsubo) and contributed to and hence clarified the diagnosis in additional 10 patients. Our data also suggests that myocarditis may be an underdiagnosed cause of SCA in adults. We did not find any association between T1, T2, and ECV elevation and primary outcomes of all-cause mortality and heart transplant as well as arrhythmic outcomes.

Our findings regarding the diagnostic value of CMR are in agreement with previously published data.[3, 4] Prior reports suggest that LVEF alone has limited sensitivity and specificity for predicting SCA, since 80% of SCA occurs in the setting of LVEF > 35%[26]. In large studies on SCA survivors with inconclusive ischemic evaluation, CMR contributed to the diagnosis in 49–69% and was decisive in 28–30% of cases, depending on the definition of the study group, despite lack of utilization of parametric mapping[7, 27]. Similar to our study, dilated cardiomyopathy, myocarditis, ischemic cardiomyopathy, and hypertrophic cardiomyopathy were the most common SCA etiologies, while 31–36% of cases had unclear etiology[7, 27].

We further demonstrate that utilization of parametric mapping has potential to decrease the number of cases with unclear etiology of SCA and hence influence clinical decision-making. Myocarditis, a common SCA cause, remains underdiagnosed[28–33]. Since endomyocardial biopsy is not recommended in every case of suspected myocarditis, CMR has become the gold standard non-invasive diagnostic method enabling tissue characterization of the entire myocardium[8, 32, 34–37]. The diagnosis of myocardial inflammation is based on updated Lake Louis criteria including T1 and T2 mapping as well as late gadolinium enhancement imaging[38]. Combination of T2-weighted imaging with LGE imaging in studies on SCA survivors with inconclusive ischemic evaluation, resulted in diagnosing myocarditis in 6–13% of cases[27, 39]. Utilization of T2 mapping, that provides more accurate and quantitative assessment of myocardial edema, could potentially explain the higher percentage of myocarditis in our study (28%)[8, 9].

T2 mapping contributed to a more certain diagnosis of other etiologies by either excluding myocarditis as a cause of SCA; confirming the diagnosis suspected based on ischemic evaluation and echocardiography as in takotsubo cardiomyopathy; or assessing the acuity and potential reversibility of the process as in MINOCA and ischemic cardiomyopathy[8]. These results have the potential to influence decisions regarding ICD implantation[27].

Other studies have also shown that T2 mapping is useful not only for making the diagnosis but also for risk stratification in certain diagnoses[8]. The degree of T2 elevation is a reliable predictor of major adverse cardiac events and heart failure hospitalization in patients with myocarditis[8, 35]. T2 elevation in hypertrophic cardiomyopathy can be used as a marker for arrhythmogenicity, and in dilated cardiomyopathy to identify patients with low probability of reverse remodeling [8, 40].

ECV elevation, which was present in a significant subset of patients in our study, has been associated with adverse outcomes in various cardiomyopathy[11–16, 41]. ECV in our population was highest in hypertrophic and ischemic CMP as well as MINOCA patients.

We attempted to analyze the prognostic role of parametric mapping aware of the study limitations. Heterogeneity of included SCA etiologies is the most likely explanation of the lack of association between T1, T2, or ECV elevation and primary outcomes in our study. Our study was limited by a small sample size, which got even smaller as specific SCA etiologies were evaluated. Further studies that address specific etiologies of SCA, including our future expanded cohort, may be able to characterize association of parametric mapping abnormalities with outcome.

Limitations

This is a single center small retrospective study. SCA etiologies were determined based on clinical data, and endomyocardial biopsy was not performed in most cases to confirm CMR-based diagnosis of myocarditis. The accuracy of the endomyocardial biopsy, however, is limited by sampling error due to the patchy nature of the inflammatory process[28, 34, 42, 43].

Since myocardial edema and inflammation observed on CMR could potentially be secondary to SCA (myocardial necrosis secondary to transient hypoxemia) or cardiopulmonary resuscitation, the data must be analyzed with caution and in correspondence with typical LGE pattern[3, 20]. Since myocarditis in our study was diagnosed based on the updated Lake Louis criteria which includes T1 and T2 mapping and LGE, it is unlikely to affect results.

Distribution of SCA etiologies is not representative for the general population since only patients who survived SCA and had no clear cause of SCA were included in the study. Small sample size with very small subgroups of each SCA etiology limited analysis of the prognostic utility of parametric mapping.

Conclusions

To conclude, inclusion of quantitative T1 and T2 parametric mapping in the SCA CMR protocol has the potential to increase diagnostic yield of CMR and further specify SCA etiology, especially myocarditis. CMR performed early after SCA may aid in the decision-making regarding ICD implantation. Further studies are needed to investigate the prognostic role of parametric mapping in SCA survivors.

Declarations

Disclosures: N/A

Acknowledgments: N/A

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Table 1

Table 1. Baseline clinical and imaging characteristics of Sudden Cardiac Arrest Survivors.

Abbreviations: BSA - body surface area; SCA – sudden cardiac arrest; MINOCA - myocardial infarction with non-obstructive coronary arteries; CMR – cardiovascular magnetic resonance; LVEDVI – left ventricular end-diastolic volume index; LVESVI - left ventricular end-systolic volume index; LVEF – left ventricular ejection fraction; LGE – late gadolinium enhancement imaging; AHA – American Heart Association.

Continuous variables are expressed as mean ± standard deviation with normal distribution and median and interquartile range (25th-75th percentiles) with non-normal distribution. Categorical variables are presented as n (%).

*Calculated from the cohort having primary outcome data (all-cause mortality, heart transplant and arrhythmic outcomes)

Characteristic	Whole cohort (n=35)	Primary Outcome*		P value
		Yes (n=15)	No (n=17)	
Age, mean (SD), y	46.9 ± 14.1	50.4 ± 15.0	46.1 ± 13.3	0.39
Male, No. (%)	20 (57)	9 (60.0)	8 (47.1)	0.46
BSA, mean (SD), m^2	2.1±0.3	2.1 ± 0.4	2.0 ± 0.2	0.32
Obesity, No. (%)	13 (37)	7 (46.7)	5 (29.4)	0.31
Hypertension, No. (%)	24 (69)	13 (86.7)	10 (58.8)	0.12
Hyperlipidemia, No. (%)	14 (40)	6 (40.0)	6 (35.3)	0.78
Diabetes mellitus, No. (%)	6 (17)	2 (13.3)	3 (17.6)	0.99
Atrial fibrillation, No. (%)	6 (17)	2 (13.3)	4 (23.5)	0.66
Coronary artery disease, No. (%)	16 (46)			
Stroke, No. (%)	4 (11)	2 (13.3)	2 (11.8)	0.99
Chronic kidney disease, No. (%)	4 (11)	2 (13.3)	2 (11.8)	0.99
Chronic obstructive pulmonary disease, No. (%)	2 (6)	2 (13.3)	0 (0.0)	0.21
SCA mechanism, No. (%)				
Ventricular fibrillation/ventricular tachycardia	33 (94)	14 (93.3)	16 (94.1)	0.99
Pulseless electrical activity	2 (6)	1 (6.7)	1 (5.9)	0.99
Suspected SCA etiology, No. (%)				0.65
Suspected SCA etiology by CMR, No. (%)				
Myocarditis (parametric mapping essential for the diagnosis)	10 (29)	4 (27)	5 (29)	
Hypertrophic cardiomyopathy	4 (11)	1 (7)	3 (18)	
Non-ischemic cardiomyopathy	5 (14)	2 (13)	3 (18)	
Ischemic cardiomyopathy	3 (9)	3 (20.0)	0 (0.0)	
Tako-tsubo cardiomyopathy (parametric mapping essential for the diagnosis)	1 (3)	0 (0)	1 (6)	
Suspected SCA etiology based on other tests, No. (%)				
Ischemic cardiomyopathy	3 (9)	1 (7)	1 (6)	
Primarily electric etiology	5 (14)	3 (20)	2 (12)	
Unclear cause	4 (11)	1 (7)	2 (12)	
CMR parameters, mean (SD)				
LVEDVI, mean (SD), ml/m2	87 (27)	91 (23)	85 (30)	0.52
LVESVI, mean (SD), ml/m2	51 (25)	55 (25)	49 (27)	0.52

LVEF, mean (SD), %,	49 (13)	46 (14)	52 (13)	0.20
T1 elevation, No. (%)	18 (51%)	10 (66.7)	7 (41.2)	0.15
Mean T1, mean (SD), ms	1069 ± 60	1085 ± 63	1059 ± 57	0.22
ECV, mean (SD), %	30 ± 7	30 ± 7	29 ± 7	0.69
T2 elevation, No. (%)	22 (63%)	9 (60.0)	11 (64.7)	0.78
Mean T2, mean (SD), ms	65 ± 10	64 ± 10	65 ± 11	0.71
LGE presence, No. (%)	32 (91)	13 (86.7)	16 (94.1)	0.59
LGE midwall, No. (%)	24 (69)	8 (53.3)	15 (88.2)	0.03
LGE subepicardial, No. (%)	7 (20)	2 (13.3)	4 (23.5)	0.66
LGE subendocardial, No. (%)	7 (20)	4 (26.7)	2 (11.8)	0.38
LGE transmural, No. (%)	4 (11.4)	3 (20.0)	1 (5.9)	0.32
AHA segments, median (IQR)	5 (4-8)	4 (3-7)	5 (4-10)	0.22

Figures

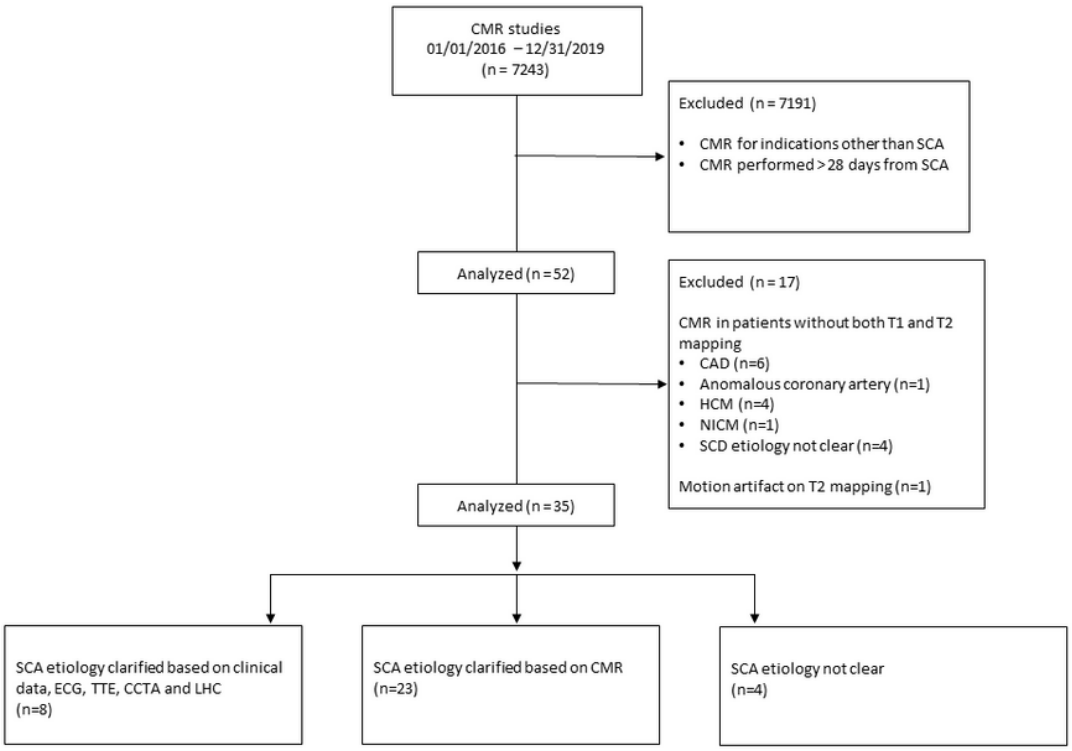

Figure 1

Flow diagram of the analyzed studies.

CMR – cardiovascular magnetic resonance; SCA – sudden cardiac arrest; CAD – coronary artery disease; HCM – hypertrophic cardiomyopathy; NICM – nonischemic cardiomyopathy; ECG – electrocardiogram; TTE – transthoracic echocardiography; CCTA – coronary computed tomography angiography; LHC – left heart catheterization.

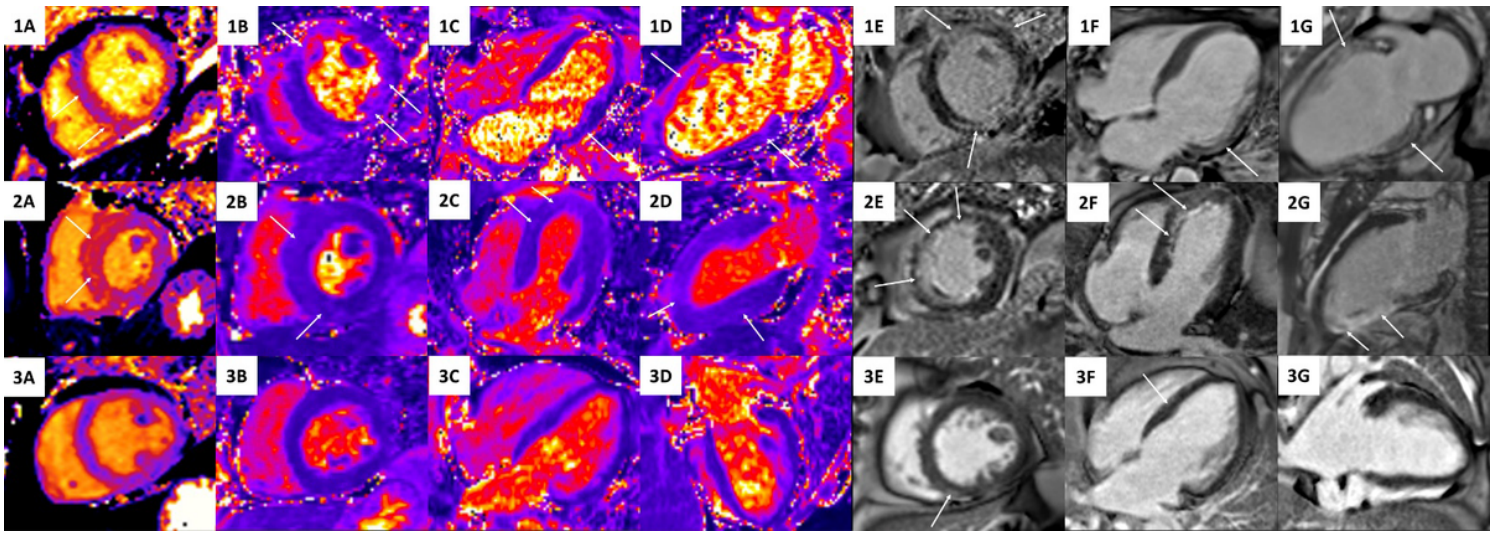

Figure 2

Cardiovascular Magnetic Resonance Findings in Sudden Cardiac Death Survivors.

T1 mapping: Panel A (mid short axis map view); **T2 mapping:** Panel B (mid short axis map view), C (horizontal long axis view), D (vertical long axis view); **Late gadolinium enhancement imaging:** Panel E (mid short axis map view), F (horizontal long axis view), G (vertical long axis view).
Panel 1, 68-year-old male: T1 signal elevation (Panel 1A; white arrows) (1127 ms; site specific normal <1060 ms), extensive myocardial edema/inflammation (Panel 2A; black arrows) (up to 92 ms in the anterior wall; site specific normal <60 ms) with corresponding prominent midmyocardial-subepicardial nonischemic fibrosis in the anterior, inferior, and lateral walls (Panel 3A-C; white arrows).
Panel 2, 55-year-old male: Nontransmural infarct scar in the septal and inferior walls (Panel 3A-C; white arrows), T1 signal elevation (Panel 1A; white arrows) (1159 ms) and corresponding myocardial edema/inflammation on T2 mapping (Panel 2A; black arrows) (up to 69 ms in the septal wall).
Panel 3, 58-year-old female: Normal native T1 values (Panel 1A) with no evidence of myocardial edema/inflammation (Panel 2A) and mild non-specific midwall non-ischemic fibrosis (Panel 3A-C; white arrows).

Supplementary Files

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