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Diagnostic value and clinical impact of cardiac magnetic resonance imaging in patients after sudden cardiac arrest: a retrospective study

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ABSTRACT

Introduction Cardiac MRI (CMRI) is an important investigation in cases of unclear cause of sudden cardiac arrest (SCA). It demonstrates diagnostic utility in assessing reversibility and tissue scar burden and ultimately aids in further treatment planning.

Methods A retrospective analysis of all adult patients referred for CMRI after SCA between 2007 and 2022 by local intensive care units in our institution was performed. The patient cohort is highly selective, excluding those who did not reach the hospital, had cerebral oedema or had confirmed acute myocardial infarction as the cause of SCA. Data on clinical presentation, imaging findings and subsequent management were collected and analysed.

Results CMRI was diagnostic in 57 of 65 patients. The most common diagnosis by CMRI was ischaemic cardiomyopathy (28.1%), followed by dilated cardiomyopathy (17.5%) and structurally normal hearts (14%). In cases of myocardial oedema, extracellular volume (ECV) was determined in 10 patients and found to be elevated in 80% after resuscitation, whereas T2 mapping was elevated in only 50% of cases. The number of examinations has increased, whereas the time to examination has decreased over the years. Additionally, CMRI findings led to changes in treatment planning.

Conclusion CMRI after resuscitation is gaining increasing interest and clinical relevance as it provides additional diagnostic information that may be crucial for therapy planning. The sensitivity of ECV in detecting myocardial oedema after cardiac arrest highlights its potential utility over T2 mapping. Future studies should investigate the impact of CMRI on long-term patient outcomes and further refine its role in guiding treatment decisions.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Cardiac MRI (CMRI) is a valuable tool for assessing myocardial structure, function and scarring, but its role in diagnosing unclear cases of sudden cardiac arrest (SCA) is not well defined.

WHAT THIS STUDY ADDS

⇒ This study shows that CMRI provides critical diagnostic insights in unclear SCA cases, identifying conditions like ischaemic cardiomyopathy and myocardial oedema, with extracellular volume proving more sensitive than T2 mapping.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ CMRI's integration into the diagnostic pathway for SCA survivors could improve patient outcomes and guide personalised treatments, while highlighting the need for further research on CMRI-based biomarkers.

INTRODUCTION

The prevention and treatment of patients with sudden cardiac arrest (SCA) is of paramount importance in the field of cardiovascular medicine. This event, characterised by an abrupt and unexpected disruption of circulation (mostly due to a cardiac rhythm disorder), continues to challenge medical practitioners and researchers.[1] Because it can occur in individuals without known heart conditions, SCA remains a difficult problem, requiring dedicated efforts to understand its complexities and develop new methods for detection, prevention and treatment. As cardiovascular medicine advances, cardiac MRI (CMRI) has emerged as a valuable tool, offering insights into the causative pathologies of SCA and improving our approach to understanding different cardiac disorders.[2 3]

SCA highlights the delicate balance of the human cardiovascular system. Most cases are caused by ischaemia in the context of coronary artery disease (CAD), but pre-existing ischaemic scars or various structural heart conditions, as well as inherited arrhythmia syndromes, can also be the causative factor for this problem. What they all have in common is that they lead to an immediate cessation in effective blood flow, which can quickly result in organ failure.[4] The impact of SCA extends

beyond the individual, affecting families, communities and healthcare systems.[5] The need to understand SCA causes, risk factors and indicators has driven continuous research.

CMRI has become an important asset in modern cardiology, as it surpasses traditional imaging techniques by providing detailed views of the heart's structure and function. Using magnetic fields and radiofrequency pulses, CMRI produces high-resolution images that reveal the heart's intricacies from various perspectives. It can capture real-time images of heart motion and blood flow, offering a thorough evaluation that goes beyond surface-level observations to delve into the core of cardiac physiology[6 7] and pathophysiology.[8 9] The comprehensive nature of CMRI allows clinicians and researchers to explore cardiac health in depth. It can assess myocardial tissue viability,[10] measure heart chamber volumes[11 12] and evaluate valvular function,[13 14] offering a detailed assessment that aids clinical decision-making. CMRI can also detect subtle anatomical and pathological changes, including scars from previous heart events.[15] These markers help clinicians understand a person's risk for SCA and guide personalised interventions.

Given these capabilities, CMRI has emerged as a crucial tool in investigating the underlying causes of SCA. Our study leverages the advanced imaging provided by CMRI to retrospectively analyse a highly selective cohort of patients who experienced unclear SCA in a single centre. By examining the diagnostic utility of CMRI in these cases, we aim to uncover patterns and insights that can improve the management and prevention of SCA. This investigation highlights the strengths of CMRI and underscores its potential to transform clinical practices and enhance patient outcomes in the realm of cardiac arrest.

MATERIALS AND METHODS

Study population

This study retrospectively included all adult patients (≥18 years) assigned to CMRI after SCA from all local intensive care units (ICU) at the Paracelsus Medical University of Salzburg, Austria, from 1 January 2007 to 31 August 2022. It is crucial to highlight that the patient cohort under investigation represents a highly selective group. Specifically, the analysis excludes patients who did not survive to reach the hospital, those who suffered significant neurological impairment such as cerebral oedema and individuals with confirmed acute myocardial infarction with ST-segment elevations as the primary cause of SCA.

Ethics declaration

The study was performed according to the Declaration of Helsinki and the standards of Good Clinical Practice. The informed consent requirement was waived by the ethics committee as this is a retrospective analysis using deidentified data.

Electrocardiography

Electrocardiography (ECG) was used to monitor cardiac electrical activity immediately after SCA in patients admitted to the ICU. A standard 12-lead ECG was performed with a routine paper speed of 50 mm/s. The ECG recordings were used to assess heart rhythm and identify arrhythmias, ischaemic changes and other electrical abnormalities. Additionally, specific attention was paid to ST-segment changes, T-wave abnormalities and the presence of Q waves, which could indicate underlying myocardial ischaemia or infarction. The QT and the corrected QT interval (QTc) were also analysed. The results from the ECG were used to guide immediate clinical management and further diagnostic evaluations. Continuous ECG monitoring was maintained to detect any episodes of ventricular fibrillation (VF), ventricular tachycardia (VT) or other life-threatening arrhythmias.

Echocardiography

Echocardiography was performed by experienced providers on patients on the same day of SCA in the ICU (on average 0.5–2 hours after hospital admission) using a high-resolution ultrasound system with a phased-array transducer. Patients were usually imaged in the supine position, and standard views, including parasternal long-axis, short-axis, apical four-chamber and two-chamber views, were obtained. Measurements provided in this study were performed retrospectively by an experienced cardiologist in training (EB) to reduce interobserver variability. IntelliSpace Cardiovascular version 6.1 (Philips Medical Systems Nederland, Best, The Netherlands) was used for all measurements and calculations. The assessment focused on evaluating left ventricular function by measuring end-diastolic and end-systolic diameters and calculating the left ventricular ejection fraction (LVEF). Right ventricular function was assessed by measuring the right ventricular diameter and tricuspid annular plane systolic excursion (TAPSE). Wall motion was analysed to identify regional wall motion abnormalities. Valvular function was examined for stenosis or regurgitation, and pressure gradients across the valves were measured. Additionally, structural assessments included measurements of intraventricular septum thickness and left ventricular posterior wall thickness.

Cardiac MRI

Each case was analysed for CMRI-specific parameters including left and right ventricular morphology and function (including left ventricular strain), wall motion abnormalities, late gadolinium enhancement (LGE), perfusion sequences, mapping sequences, extracellular volumes (ECV) and the diagnoses according to the CMRI report (each if available). T1 and T2 mapping were introduced into the institutional CMRI protocol at a later stage of the study period and were therefore not available for earlier cases. In addition, sequences with insufficient image quality or mapping artefacts were excluded from the analysis to ensure robustness. The interpretation of

native T1, T2 and ECV values was based on published reference values obtained from healthy individuals at our institution. The CMRI studies were performed on 1.5T and 3T scanners (Ingenia 1.5T and Achieva 3T, Philips Healthcare, Best, The Netherlands). A combined 16-channel anterior-posterior coil system was used for the 1.5T scanner, and a 6-channel cardiac radiofrequency coil and MultiTransmit technology were used for the 3.0T scanner. Electrocardiography was used for cardiac gating. Since the examinations were conducted over an extended period, the examination and contrast agent protocols, scanner hardware, software versions and post-processing tools inevitably varied. To reduce interscanner and temporal variability, all available datasets were re-evaluated by a board-certified radiologist subspecialised in cardiovascular imaging (BSc) using a consistent post-processing platform (Philips IntelliSpace). All imaging studies included cine sequences for morphological and functional analyses, nearly all included LGE and perfusion sequences (without stress perfusion) and T1 and T2 mapping sequences were included to varying degrees. Conventional T2-weighted imaging was not part of the diagnostic protocol. Myocardial oedema (ME) was evaluated exclusively using quantitative T2 mapping techniques. All studies were performed by experienced radiographers and radiologists trained in CMRI. All CMRI images were analysed using Philips IntelliSpace (Philips Healthcare, Best, The Netherlands) on a stationary workstation. Re-evaluation of datasets and any missing measurements was performed using this dedicated platform by a board-certified radiologist subspecialised in cardiovascular imaging (BS).

Indication for implantable cardioverter-defibrillator

Implantable cardioverter-defibrillator (ICD) implantation criteria for secondary prevention in patients who have survived SCA were based on established European Society of Cardiology (ESC) guidelines valid at the respective time period,[16 17] with the aim of preventing recurrent life-threatening arrhythmias.

Secondary prevention criteria for ICD implantation:

- Survivors of SCA: patients who have survived a cardiac arrest due to VF or haemodynamically unstable VT not associated with a reversible or transient cause.
- Sustained VT: patients with spontaneous sustained VT causing syncope or significant haemodynamic compromise, and those with sustained VT who have not experienced syncope but have significant cardiac disease and/or reduced LVEF.

Contraindications and considerations for ICD non-implantation:

- Reversible causes: if the SCA was caused by a reversible or transient factor such as acute myocardial infarction, severe electrolyte imbalances, drug toxicity or trauma, and this factor has been corrected.
- Severe comorbidities: patients with severe comorbid conditions that significantly limit life expectancy and where the potential benefits of ICD implantation do not outweigh the risks and burdens of the procedure.
- Poor functional status: patients with poor functional status or severe heart failure (eg, New York Heart Association class IV) where ICD implantation is unlikely to improve quality of life or survival.
- Patient preference: patients who, after thorough discussion with their healthcare provider, choose not to undergo ICD implantation due to personal preferences, understanding the risks, benefits and potential outcomes.

Major adverse cardiac events and follow-up

Major adverse cardiac events (MACE) were defined in this study as significant cardiovascular complications occurring following SCA. These events included recurrent cardiac arrest, sustained VT, sustained VF and death. Appropriate interventions for MACE, such as implantable ICD therapy, were administered based on established guidelines.[16 17] Data regarding the occurrence of MACE and corresponding interventions were collected and analysed to assess their impact on patient outcomes. Follow-up was done for 12 months after SCA.

Statistical analysis

The statistical analysis was conducted using SPSS (V.25.0, SPSS). To assess the normal distribution of variables, the Kolmogorov-Smirnov-Lilliefors test was employed. Normally distributed metric data were presented as mean±SD and analysed using an unpaired Student's t-test. For non-normally distributed metric data, the median and IQR were used, and statistical analysis employed the Mann-Whitney U test. Categorical data were expressed as frequencies/percentages and compared using the χ^2 test.

RESULTS

Study cohort

In total, 65 patients with CMRI during their hospitalisation after cardiac arrest were identified in this retrospective investigation. In eight patients, the scan was aborted because of tachyarrhythmia (three patients, 4.6%) or claustrophobia (five patients, 7.7%). In 57 patients, the imaging studies were considered diagnostic and consecutively included in this analysis.

Baseline characteristics

The study analysed participants' clinical characteristics before experiencing SCA, as shown in table 1. The average age was 52.6 years, with 68.4% being male. Median height was 180.0 cm, average weight was 81.3 kg and mean body surface area was 2.0 m^2. Notably, 61.4% had no prior cardiac history with an age of 49.0±14.5 years and 68.6% being male. Among those with prior medical conditions, 12.3% had CAD, 8.8% had previous myocardial infarction, 10.5% suffered from heart failure, 40.4% had arterial hypertension, 7.0% had a history of diabetes mellitus and 31.6% had hypercholesterolaemia. Additionally, 10.5% were current smokers, with no cases of peripheral

Table 1 Patients' clinical characteristics before experiencing SCA

Age (years), mean±SD	52.6±14.8
Sex (male), n (%)	39/57 (68.4)
Height (cm), median±IQR	180.0±18.0
Weight (kg), mean±SD	81.3±15.7
BSA (m^2), mean±SD	2.0±0.2
Negative, cardiac anamnesis, n (%)	35/57 (61.4)
CAD, n (%)	7/57 (12.3)
MI, n (%)	5/57 (8.8)
HF, n (%)	6/57 (10.5)
aHT, n (%)	23/57 (40.4)
DM, n (%)	4/57 (7.0)
HC, n (%)	18/57 (31.6)
Current smoker, n (%)	6/57 (10.5)
PAD, n (%)	0/57 (0.0)
COPD, n (%)	1/57 (1.8)

aHT, arterial hypertension; BSA, body surface area; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; DM, diabetes mellitus; HC, hypercholesterolaemia; HF, heart failure; MI, myocardial infarction; PAD, peripheral artery disease; SCA, sudden cardiac arrest.

artery occlusive disease and 1.8% with chronic obstructive pulmonary disease.

ECG, laboratory parameters, echocardiography and coronary angiography

A detailed overview of ECG, laboratory parameters, echocardiography and coronary angiography (CAG) findings is demonstrated in table 2. Initially, 86.0% of patients had VF as their initial heart rhythm during cardiopulmonary resuscitation (CPR), with 10.5% experiencing VT and 3.5% having Torsades de Pointes tachycardia. After the return of spontaneous circulation, 87.7% had sinus rhythm and 12.3% had atrial fibrillation. Left bundle branch block was observed in 10.5% and right bundle branch block in 3.5%. The average QRS duration was 105.8 ms, and the QTc was 552.8 ms.

Relevant laboratory parameters—especially cardiac biomarkers—revealed a troponin T of 308±55 pg/mL and an N-terminal pro-B-type natriuretic peptide of 1672±593 pg/mL.

Echocardiographic assessments revealed a mean LVEF of 46.5%, a left ventricular end-diastolic diameter (LVEDD) of 50.2 mm, an interventricular septal diameter (IVSD) of 11.9 mm and a left ventricular posterior wall diameter of 11.1 mm. Aortic valve insufficiency was present in 17.6% of cases, with varying degrees of severity. Mitral valve insufficiency occurred in 63.2%, and tricuspid valve insufficiency was found in 57.9%. One patient had moderate aortic stenosis. The mean TAPSE was 19.8 mm, while the median systolic pulmonary artery pressure (sPAP) was 35.0 mm Hg. The median TAPSE/

Table 2 ECG, laboratory parameters, echocardiography and coronary angiography data of study cohort

ECG	
Initial heart rhythm at CPR, n (%)	
VF	49/57 (86.0)
VT	6/57 (10.5)
TdP	2/57 (3.5)
Heart rhythm after ROSC, n (%)	
SR	50 (87.7)
AF	7 (12.3)
LBBB, n (%)	6/57 (10.5)
RBBB, n (%)	2/57 (3.5)
QRS (ms), mean±SD	105.8±19.2
QTc (ms), mean±SD	552.8±44.4
Laboratory parameters	
Sodium (mmol/L), mean±SD	138.3±8.5
Potassium (mmol/L), mean±SD	4.3±1.1
Troponin T (pg/mL), mean±SD	308±55
NT-proBNP (pg/mL), mean±SD	1672±593
Transthoracic echocardiography	
LVEF (%), mean±SD	46.5±11.0
LVEDD (mm), mean±SD	50.2±10.8
IVSD (mm), mean±SD	11.9±3.2
LVPWD (mm), mean±SD	11.1±1.8
AVI, n (%)	
I°	9/57 (15.8)
II°	1/57 (1.8)
III°	0/57 (0.0)
MVI, n (%)	
I°	27/57 (47.4)
II°	5/57 (8.8)
III°	4/57 (7.0)
TVI, n (%)	
I°	26/57 (45.6)
II°	6/57 (10.5)
III°	1/57 (1.8)
AS, n (%)	
I°	0/57 (0.0)
II°	1/57 (1.8)
III°	0/57 (0.0)
TAPSE (mm), mean±SD	19.8±6.3
sPAP (mm Hg), median±IQR	35.0±23.5
TAPSE/sPAP (mm/mm Hg), median±IQR	0.8±1.3
Wall motion disorders, n (%)	35/57 (61.4)
Coronary angiography	
Coronary angiography done, n (%)	54/57 (94.7)
CAD, n (%)	19/57 (33.3)

Continued

Table 2 Continued

CAD 1 vessel, n (%)	10/57 (17.5)
CAD 2 vessels, n (%)	7/57 (12.3)
CAD 3 vessels, n (%)	2/57 (3.5)
Intervention necessary, n (%)	4/57 (7.0)

AF, atrial fibrillation; AS, aortic stenosis; AVI, aortic valve insufficiency; CAD, coronary artery disease; CPR, cardiopulmonary resuscitation; I°-III°, grade 1-3; IVSD, interventricular septum thickness in diastole; LBBB, left bundle branch block; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVPWD, left ventricular posterior wall diameter; MVI, mitral valve insufficiency; NT-proBNP, N-terminal pro-B-type natriuretic peptide; QTc, corrected QT interval; RBBB, right bundle branch block; ROSC, return of spontaneous circulation; sPAP, systolic pulmonary artery pressure; SR, sinus rhythm; TAPSE, tricuspid annular plane systolic excursion; TdP, Torsades de Pointes; TVI, tricuspid valve insufficiency; VF, ventricular fibrillation; VT, ventricular tachycardia.

sPAP ratio was 0.8. Wall motion abnormalities were identified in 61.4% of participants.

CAG was performed in 94.7% of the patients, revealing that 33.3% had CAD. Overall, 17.5% had single-vessel disease, 12.3% had two-vessel disease and 3.5% had three-vessel disease. Interventions were necessary in 7.0% of cases.

CMRI data

Over the last 15 years, the number of CMRI scans after CPR has continuously increased in our institution, as demonstrated in figure 1. Simultaneously, the time to scan after CPR has continuously decreased, now at an average of 2 days to scan after CPR in the year 2022. The median time to scan was 5 days.

An overview of the CMRI data collected is shown in table 3. Of all CMRI scans, 54.4% were conducted using a 1.5T machine, while 45.6% used a 3.0T machine. The average heart rate during the scans was 70.7 beats per minute.

The mean left atrial depth was 60.5 mm and the mean right atrial depth was 62.7 mm. The mean LVEF was 50.5%, with a median left ventricular stroke volume of 90.3 mL and a mean left ventricular cardiac output of 6.3 L/min. The mean left ventricular end-diastolic volume was 191.6 mL, and the left ventricular end-systolic volume had a median of 71.1 mL. The average LVEDD was 55.8 mm, and the IVSD had a median of 12.0 mm. The mean right ventricular ejection fraction was 59.2%, with a mean right ventricular stroke volume of 97.4 mL and a mean right ventricular cardiac output of 6.8 L/min. The mean right ventricular end-diastolic volume was 166.3 mL, and the mean right ventricular end-systolic volume was 68.9 mL. The average right ventricular end-diastolic diameter was 45.2 mm. Left ventricular global radial strain peak was on average 22.9%, with a mean time to peak (TTP) of 297.0 ms. Left ventricular global circumferential strain peak averaged −10.5%, with a TTP of 301.0 ms, while left ventricular global longitudinal strain peak averaged −10.4%, with a TTP of 333.0 ms. T1 and T2 mapping were available for 43.9% and 63.2% of patients, respectively. Myocardial fibrosis was observed in 36.0% of diagnostically useable T1 sequences and ME in 21.1% of diagnostically useable T2 sequences. ECV was available in 17.5% and elevated with ≥30% in 80% of these. Pericardial effusion was found in 29.8% of participants, and wall motion disorders were present in 47.4%. LGE was available for 61.4%, showing a non-ischaemic pattern (mid-wall and/or subepicardial enhancement) in 25.7%, an ischaemic pattern (subendocardial or transmural enhancement) in 71.4% and a mixed pattern in 2.9%. Perfusion deficits were noted in 31.6% of the patients.

Final diagnosis

Diagnoses made by the imaging studies are visualised in table 4, revealing the most common diagnoses in our study cohort as follows: ischaemic cardiomyopathy (28.1%), dilated cardiomyopathy (17.5%) and structurally normal hearts (14%). Six cases of hypertrophic cardiomyopathy (10.5%) and three cases of myocarditis (5.3%) were diagnosed via CMRI. Four more cases (7.0%) were clinically diagnosed as long QT syndrome beforehand with the question to the CMRI scan to exclude other structural heart diseases. Other diagnoses included Morbus Barlow (3.5%), Takotsubo cardiomyopathy (1.7%) and sarcoidosis (1.7%). One case of sarcoidosis and one case of myocarditis were confirmed by endomyocardial biopsy (EMB). For six cases (10.5%), the diagnosis remained unclear.

Medication and outcome

The study also provided data on medications administered after SCA and patient outcomes (table 4). Regarding medications post-SCA, 26.3% of the patients were on oral anticoagulants, 43.9% on aspirin and 21.1% on P2Y12 inhibitors. ACE inhibitors or angiotensin II receptor blockers were prescribed to 49.1%, beta blockers to 84.2% and mineralocorticoid receptor antagonists to 28.1%. Sodium-glucose cotransporter-2 inhibitors were used by 1.8% of patients, class Ic antiarrhythmics by 3.5% and class III antiarrhythmics by 12.3%.

In terms of outcomes, EMB was used in 5.3% of cases, while ICD implantation was performed in 68.4% of patients. No patients received cardiac resynchronisation therapy. The mortality rates were 5.3% at 1 year and 8.8% at one and a half years. Major adverse cardiac events (MACE) occurred in 14.0% of patients at 1 year.

DISCUSSION

In our study, we aimed to delineate the role of CMRI in diagnosing and managing patients after unclear SCA. In addition, the aim was to establish a diagnostic work-up flow chart as illustrated in figure 2, where we present a structured and comprehensive approach, integrating multiple diagnostic modalities to ensure a thorough evaluation of each patient.

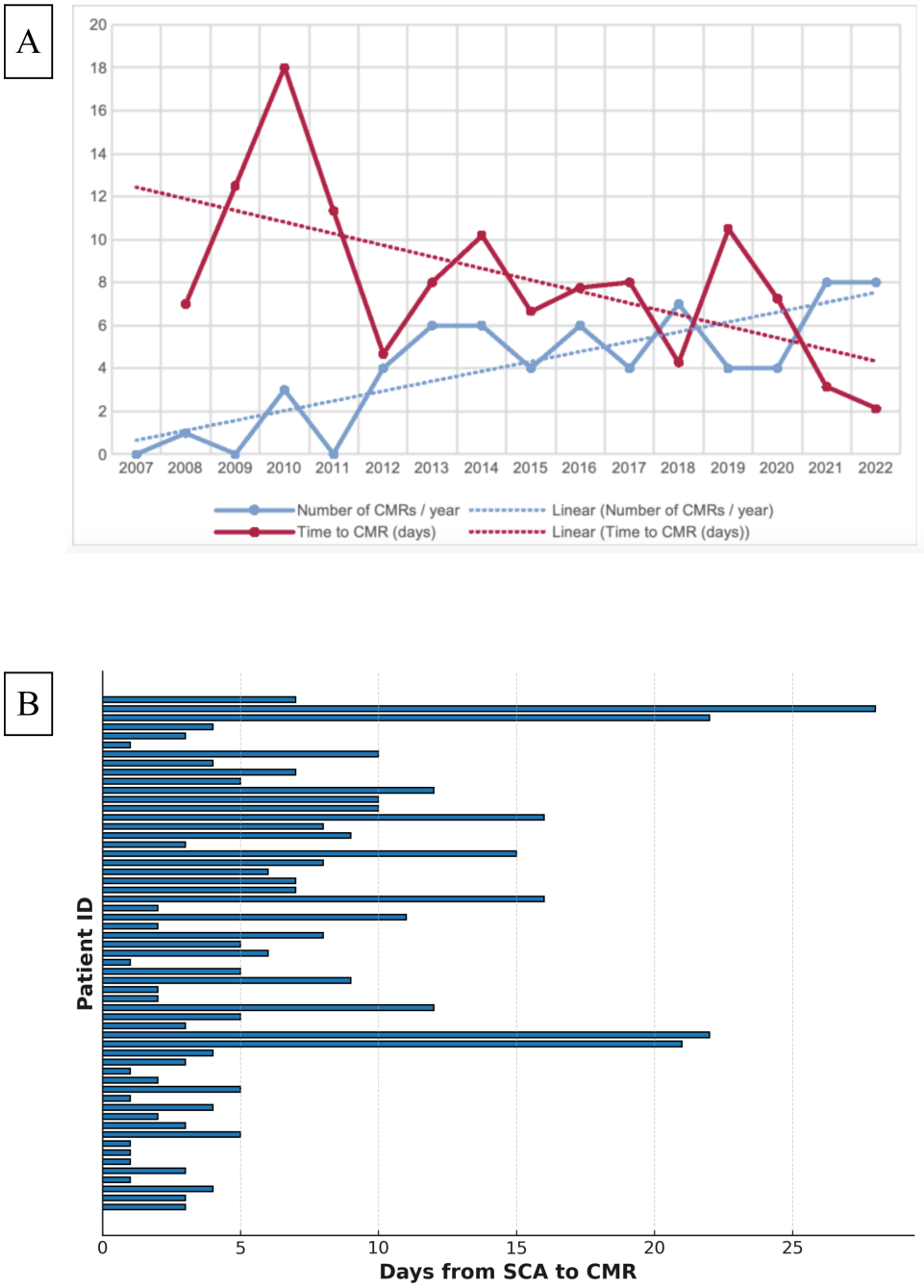

Figure 1 (A) Overview of numbers of cardiac MRI (CMRI) per year and time to CMRI in study cohort. (B) Exact constellation of patient ID and time to CMRI in study cohort. SCA, sudden cardiac arrest.

Flow chart for diagnostic work-up of patients with SCA

The initial assessment of patients post-SCA involves basic but essential diagnostic tools, starting with ECG and echocardiography. ECG is the first step, primarily aimed at identifying immediate life-threatening conditions such as acute coronary syndromes, significant arrhythmias or conduction abnormalities.[18] Concurrently, echocardiography is performed to evaluate cardiac function and structure, providing insights into ventricular size, wall motion abnormalities, ejection fraction and the presence of pericardial effusion.[19] These initial steps are critical for stabilising the patient and guiding subsequent diagnostic and therapeutic decisions. Following the initial imaging, a series of laboratory tests are conducted to provide additional insights. Cardiac biomarkers such as troponin levels are measured to assess the extent of myocardial injury.[20] A complete blood count, electrolytes, renal function tests and liver enzymes are also evaluated to give a comprehensive overview of the patient's systemic health and identify any underlying conditions that may have contributed to the cardiac arrest.[21 22] In cases where ECG or echocardiography suggests ischaemic heart disease or if the patient presents with symptoms indicative of an acute coronary syndrome, CAG is performed. CAG is crucial for visualising the coronary arteries, identifying stenoses and guiding revascularisation procedures if

Table 3 CMRI data of study cohort

CMRI	
Time to CMRI (days), median±IQR	5.0±7.0
Tesla, n (%)	
1.5	31/57 (54.4)
3.0	26/57 (45.6)
Heart rate (bpm), mean±SD	70.7±16.1
Left atrial depth (mm), mean±SD	60.5±10
Right atrial depth (mm), mean±SD	62.7±9.5
LVEF (%), mean±SD	50.5±15.4
LVSV (mL), median±IQR	90.3±30.2
LVCO (L/min), mean±SD	6.3±1.5
LVSI (mL/m^2), median±IQR	44.4±15.3
LVCI (L/min/m^2), mean±SD	3.1±0.8
LVEDV (mL), mean±SD	191.6±58.4
LVEDV/BSA (mL/m^2), mean±SD	96.2±27.6
LVESV (mL), median±IQR	71.1±64.4
LVESV/BSA (mL/m^2), median±IQR	46.7±78.1
LVEDD (mm), mean±SD	55.8±8.7
IVSD (mm), median±IQR	12.0±4.0
RVEF (%), mean±SD	59.2±9.2
RVSV (mL), mean±SD	97.4±24.6
RVCO (L/min), mean±SD	6.8±2.1
RVSI (mL/m^2), mean±SD	48.8±11.5
RVCI (L/min/m^2), mean±SD	3.4±1.0
RVEDV (mL), mean±SD	166.3±40.7
RVEDV/BSA (mL/m^2), mean±SD	83.1±18.0
RVESV (mL), mean±SD	68.9±27.8
RVESV/BSA (mL/m^2), median±IQR	30.0±12.2
RVEDD (mm), mean±SD	45.2±8.2
RVEDV/LVEDV, mean±SD	0.9±0.2
RVESV/LVESV, mean±SD	0.9±0.4
Radial strain peak (%), mean±SD	22.9±12.5
Radial strain TTP (ms), mean±SD	297.0±78.0
Circumferential strain peak (%), mean±SD	−10.5±3.9
Circumferential strain TTP (ms), mean±SD	301.0±81.0
Longitudinal strain peak (%), mean±SD	−10.4±4.5
Longitudinal strain TTP (ms), mean±SD	333.0±93.0
T1 mapping available, n (%)	25/57 (43.9)
T1 image quality diagnostic, n (%)	19/25 (76.0)
MF, n (%)	9/25 (36.0)
T2 mapping available, n (%)	36/57 (63.2)
T2 image quality diagnostic, n (%)	24/36 (66.7)
ME, n (%)	12/24 (50.0)
ECV, n (%)	10/57 (17.5)
ECV>30%, n (%)	8/10 (80.0)
Pericardial effusion, n (%)	17/57 (29.8)
LGE availability, n (%)	35/57 (61.4)
LGE pattern, n (%)	
Non-ischaemic	9/35 (25.7)
Ischaemic	25/35 (71.4)
Mixed	1/35 (2.9)
Wall motion disorders, n (%)	27/57 (47.4)
Perfusion deficit, n (%)	18/57 (31.6)

BSA, body surface area; CMRI, cardiac MRI; ECV, extracellular volume; IVSD, interventricular septum thickness in diastole; LGE, late gadolinium enhancement; LVCI, left ventricular cardiac index; LVCO, left ventricular cardiac output; LVEDD, left ventricular end-diastolic diameter; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESV, left ventricular end-systolic volume; LVSI, left ventricular stroke index; LVSV, left ventricular stroke volume; ME, myocardial oedema; MF, myocardial fibrosis; RVCI, right ventricular cardiac index; RVCO, right ventricular cardiac output; RVEDD, right ventricular end-diastolic diameter; RVEDV, right ventricular end-diastolic volume; RVEF, right ventricular ejection fraction; RVESV, right ventricular end-systolic volume; RVSI, right ventricular stroke index; RVSV, right ventricular stroke volume; TTP, time to peak.

necessary.[23 24] This step is vital for patients with suspected CAD as it directly influences the treatment strategy, including percutaneous coronary intervention or coronary artery bypass grafting. Additionally, CT is employed in specific scenarios. For instance, CT CAG can be used when CAG is not immediately available or contraindicated. Moreover, CT can assess pulmonary embolism, aortic dissection or other thoracic pathologies that might mimic or contribute to cardiac symptoms.[25 26]

If initial assessments and angiographic findings do not fully explain the patient's condition or if there is suspicion of underlying structural heart disease, CMRI is conducted. CMRI provides detailed information on myocardial structure, function and tissue characterisation. It is particularly useful for diagnosing ischaemic cardiomyopathy, differentiating between acute and chronic ischaemic cardiomyopathies, assessing infarct extent and identifying pre-existing myocardial scarring, which can be the structural basis for malignant ventricular arrhythmias.[3 27 28] In selected cases where the diagnosis remains unclear, or there is suspicion of myocarditis, infiltrative diseases or specific cardiomyopathies, an EMB may be performed.[29] This invasive procedure allows for direct tissue analysis, providing histopathological confirmation of the underlying disease process. Electrophysiological studies (EPS) are also integral to the work-up, especially in patients with unexplained arrhythmias or syncope.[30] EPS help identify arrhythmic foci, assess conduction pathways and guide the implantation of devices such as pacemakers or defibrillators. The comprehensive diagnostic work-up involving ECG, echocardiography, laboratory tests, CAG, CT, CMRI, EMB and EPS enables a thorough evaluation of patients post-SCA. The integration of these findings is critical for formulating an accurate diagnosis

Table 4 Final diagnosis, medication and outcome data of study cohort

Final diagnosis	
Barlow's disease, n (%)	2/57 (3.5)
DCM, n (%)*	10/57 (17.5)
HCM, n (%)†	6/57 (10.5)
ICM, n (%)	16/57 (28.1)
LQT, n (%)‡	4/57 (7.0)
Myocarditis, n (%)	3/57 (5.3)
Sarcoidosis, n (%)	1/57 (1.8)
Structural normal heart, n (%)	8/57 (14.0)
Takotsubo syndrome, n (%)	1/57 (1.8)
Unclear, n (%)	6/57 (10.5)
Medication after SCA	
OAC, n (%)	15/57 (26.3)
Aspirin, n (%)	25/57 (43.9)
P2Y12 inhibitor, n (%)	12/57 (21.1)
ACEI/ARB, n (%)	28/57 (49.1)
BB, n (%)	48/57 (84.2)
MRA, n (%)	16/57 (28.1)
SGLT-2 inhibitor, n (%)	1/57 (1.8)
Class Ic antiarrhythmics, n (%)	2/57 (3.5)
Class III antiarrhythmics, n (%)	7/57 (12.3)
EMB, ICD, MACE and death	
EMB, n (%)	3/57 (5.3)
ICD implantation, n (%)	39/57 (68.4)
CRT implantation, n (%)	0/57 (0.0)
MACE, n (%)	
3 months	4/57 (7.0)
6 months	4/57 (7.0)
1 year	8/57 (14.0)
Death, n (%)	
3 months	2/57 (3.5)
6 months	2/57 (3.5)
1 year	3/57 (5.3)

*3/10 genetically tested and verified.
†2/6 genetically tested and verified.
‡2/4 genetically tested and verified.
ACEI, ACE inhibitor; ARB, angiotensin II receptor blocker; BB, beta blocker; CRT, cardiac resynchronisation therapy; DCM, dilated cardiomyopathy; EMB, endomyocardial biopsy; HCM, hypertrophic cardiomyopathy; ICD, implantable cardioverter-defibrillator; ICM, ischaemic cardiomyopathy; LQT, long QT; MACE, major adverse cardiac event; MRA, mineralocorticoid receptor antagonist; OAC, oral anticoagulant; SCA, sudden cardiac attack; SGLT-2, sodium-glucose cotransporter-2.

and guiding appropriate treatment strategies. Notably, CMRI plays a pivotal role by providing detailed myocardial characterisation that can significantly influence treatment decisions.

Findings and implications

Our study showed that CMRI was instrumental in diagnosing ischaemic cardiomyopathy (28.1% of cases) and dilated cardiomyopathy (17.5%), and identifying structurally normal hearts (14%). These findings illustrate the diverse aetiologies of SCA and the necessity of a comprehensive diagnostic approach. CMRI's ability to detect ME through ECV measurements and T2 mapping was also notable. In our cohort, ECV was elevated in 80% of cases after resuscitation, suggesting it may be more sensitive than T2 mapping (elevated in only 50% of cases) for detecting extracellular oedema. Another explanation is, of course, an elevated ECV in the context of fibrotic changes. This has significant implications for the SCA evaluation, as it highlights the potential for ECV to serve as a more reliable marker of myocardial injury.[27 31]

In our cohort, the most common diagnosis by CMRI was ischaemic cardiomyopathy. CMRI was essential in differentiating between acute and chronic ischaemic cardiomyopathy and in determining the extent of infarction or identifying pre-existing scarring as the structural basis for malignant ventricular arrhythmia. These distinctions are critical for guiding therapeutic decisions and for risk stratification in SCA survivors. Nevertheless, ischaemic cardiomyopathy is under-represented due to the selective exclusion of patients with acute myocardial infarction as the primary cause of SCA, as these patients typically receive a diagnosis via CAG rather than CMRI.

The ability of CMRI to detect ME and scarring has practical implications for patient management. The elevated ECV in postresuscitation patients suggests that this measure might be more sensitive than T2 mapping for detecting extracellular oedema/ME,[32 33] an important factor in the management and prognosis of these patients.

The higher and normal LVEF observed in CMRI compared with echocardiography likely reflects reversible myocardial dysfunction as part of the postcardiac arrest syndrome. This phenomenon, well-documented in previous studies such as Grand *et al*,[34] may be explained by the fact that CMRI was performed at a median of 5.0±7.0 days post-CPR, allowing for recovery of transient myocardial stunning. Additionally, the improved LVEF is consistent with the nature of this patient cohort, many of whom experienced acute arrhythmic events rather than severe myocardial infarctions, leading to a quicker recovery. This selective patient population and the rapid treatment of rhythm disturbances are key factors that must be emphasised to contextualise the results accurately.

The potential of CMRI to influence treatment planning is significant; future studies should explore how often CMRI findings lead to changes in clinical management, such as the decision between a wearable cardioverter-defibrillator and an ICD.

Limitations

Our study is retrospective, limited by a small sample size (n=57), and the patient cohort is highly selective, which may impact the generalisability of our findings. A major

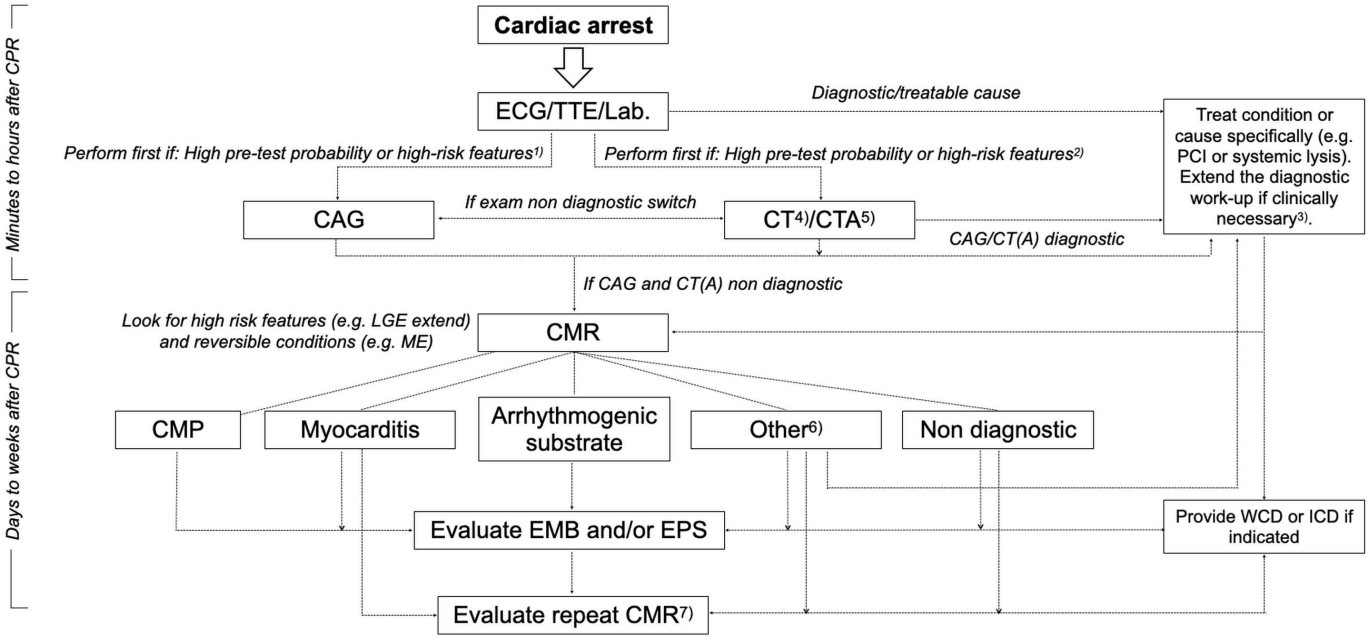

Figure 2 Diagnostic work-up after sudden cardiac arrest (SCA). [1]For example, ST-elevation myocardial infarction, haemodynamic instability, etc. [2]For example, right heart strain/'D-sign', pericardial effusion, etc. [3]For example, genetic testing, exercise stress testing, further imaging modalities, etc. [4]To rule out intracranial bleeding/stroke. [5]To rule out pulmonary embolism/aortic dissection. [6]For example, cardiac sarcoidosis, athlete's heart, etc. [7]For example, bad image quality, reversible conditions. CAG, coronary angiography; CMP, cardiomyopathy; CMRI, cardiac MRI; CPR, cardiopulmonary resuscitation; CTA, CT angiography; EMB, endomyocardial biopsy; EPS, electrophysiological study; ICD, implantable cardioverter-defibrillator; Lab, laboratory work-up with focus on electrolytes; LGE, late gadolinium enhancement; ME, myocardial oedema; PCI, percutaneous coronary intervention; TTE, transthoracic echocardiography; WCD, wearable cardioverter-defibrillator.

limitation of this study is the heterogeneity introduced by the long observational period (2007–2022), during which several technological changes occurred. Scanner hardware, contrast agents and contrast agent protocols, and mapping sequences evolved over time. Although we attempted to minimise bias through centralised reanalysis of imaging data and the use of consistent reference standards, we acknowledge that this variability may still influence mapping-derived parameters and comparisons across the dataset. The time between the SCA and CMRI varied across patients, with a median of 5 days. This timing heterogeneity may influence mapping results, strain analysis and the detection of reversible oedema or transient dysfunction, thereby limiting comparability across patients. Additionally, the study's observational design does not allow for causal inferences between identified cardiac pathologies and outcomes.

CONCLUSION

Our study underscores the critical role of CMRI in the postresuscitation care of SCA survivors. The detailed structural and functional information provided by CMRI enables the identification of a wide array of underlying cardiac conditions, facilitating tailored therapeutic interventions. Our diagnostic work-up and flow chart serve as a guide for integrating CMRI into clinical practice, ensuring a comprehensive evaluation of patients who had SCA. Future research with larger, prospective cohorts is needed to validate our findings and further refine the role of CMRI in this clinical context. The evolving use of CMRI, as indicated by the increasing number of scans and decreasing time to scan post-CPR, reflects its growing recognition and adoption in the management of patients who had post-SCA. By enhancing our understanding of the underlying pathologies, CMRI contributes significantly to the personalised care and improved outcomes for survivors of SCA.

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