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Research article

Cardiac magnetic resonance characteristics in young survivors of aborted sudden cardiac death

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ABSTRACT

Purpose: We aimed to identify and assess cardiac abnormalities by cardiovascular magnetic resonance (CMR) in a non-ischaemic aborted sudden cardiac death (SCD) population and to establish possible predictors of SCD.
Methods: Thirty-six consecutive SCD survivors [median age 37.6 years (IQR 24.1-43.2), 31% female] with no previous cardiac history or evidence of ischaemic heart disease underwent CMR on day 6 (IQR 4-10) after admission. Data on ventricular volumes and the extent of late gadolinium enhancement (LGE) were collected. Systolic strain analysis was performed using feature tracking software.
Results: Left ventricular (LV) and right ventricular (RV) indexed diastolic volumes were 92.9 ± 28.4 ml/m^2 and 94.1 ± 29 ml/m^2, respectively. LV ejection fraction (EF) and RV EF were $56.8 \pm 10.7\%$ and $53.7 \pm 10.7\%$, respectively. Global peak endocardial longitudinal, circumferential, and radial strain were $-17.9 \pm 4.28\%$, $-23.2 \pm 5.8\%$, and $32.8 \pm 10.6\%$, respectively. Compared to normal range, global longitudinal endocardial strain, longitudinal epicardial strain, circumferential endocardial strain, radial strain, and circumferential endocardial strain rate were impaired. Median volume of LGE was 0.25% (IQR 0.12-1.12) of the LV myocardium with highest prevalence in the inferolateral wall. Patients with cardiomyopathy diagnosis (n = 16) had lower LV strain rate compared to patients without cardiomyopathy (n = 20).
Conclusions: CMR findings in young patients with aborted SCD due to non-ischaemic heart disease seem to be minor. Although only present in small amounts, LGE appears to have a predilection towards the inferolateral wall in this population.

1. Introduction

Sudden cardiac death (SCD) caused by malignant ventricular arrhythmias accounts for an estimated 15–20% of all deaths [1]. Coronary heart disease (CHD) (75%) and valvular heart disease (1–5%) are the largest contributors of SCD in the Western countries in adults aged more than 35. Inherited cardiomyopathies (10–15%) and inherited arrhythmic syndromes (1–2%) are the primary non-ischaemic causes of SCD in the Western countries, typically affecting adults aged less than 35 [1].

The presumed pathophysiology of SCD in these patients is electrical instability precipitating ventricular arrhythmias and ultimately SCD. Post mortem data suggest, that in the absence of macroscopic changes, the arrhythmia might be attributed to microscopic substrates such as myocarditis or cardiomyopathy [2]. On the other hand, the post mortem prevalence of inherited arrhythmic syndromes seems to be similar both in patients with and without minor structural changes, e.g. in mild ventricular dilatation without fibrosis or inflammation [3]. Meanwhile, as far as survivors of non-ischaemic aborted SCD (aSCD) are concerned, a detailed description of functional and structural changes in this group previously has not been performed.

Cardiovascular magnetic resonance (CMR) is an imaging modality that yields accurate high-resolution images of the myocardium and both ventricles. It provides comprehensive information on cardiac morphology, function, deformation, and tissue characterization in a single examination. The aim of the present study was to assess CMR findings in a young non-ischaemic aSCD population and to identify possible predictors of SCD in this group.

Abbreviations: AHA, American Heart Association; aSCD, aborted sudden cardiac death; ATP, anti-tachycardia pacing; CMR, cardiovascular magnetic resonance scanning; CHD, coronary heart disease; CPVT, catecholaminergic polymorphic ventricular tachycardia; ECG, electrocardiogram; EDd, end-diastolic diameter; EF, ejection fraction; ICC, intraclass correlation coefficient; ICD, implantable cardioverter defibrillator; IQR, interquartile range; LGE, late gadolinium enhancement; LV, left ventricle; RV, right ventricle; SCD, sudden cardiac death; SSFP, steady-state free precession; SR, strain rate; TAPSE, tricuspidal annular plane systolic excursion; VF, ventricular fibrillation; VT, ventricular tachycardia

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2. Material and methods

2.1. Study population

Eligible patients for this single-centre, descriptive, retrospective, cross-sectional study were identified through the Danish Pacemaker and ICD Registry. The study population consisted of consecutive patients admitted to the Department of Cardiology, Aarhus University Hospital due to first episode of aSCD or sustained ventricular tachycardia (VT) during 2011–2014 who underwent CMR and received an implantable cardioverter-defibrillator (ICD). Only patients < 50 years of age were included. All patients admitted to the department were clinically evaluated with routine blood test, ECG, echocardiogram, coronary angiogram/coronary computed tomography to exclude reversible cause of aSCD. All medical records of patients < 50 years of age from the inclusion period were thoroughly read by author AKB. Baseline clinical characteristics and echocardiographic data were collected from patients records. Exclusion criteria were aSCD resulting from significant CHD (previous or current diagnosis of acute coronary syndrome, revascularization procedure, or presence of a flow-limiting coronary artery stenosis), myocarditis, previously documented structural heart disease (valvular heart disease, cardiomyopathy), ventricular fibrillation (VF) or sustained VT due to electrolyte abnormality or drug intoxication, or prior aSCD or sustained VT. Myocarditis patients were excluded based on Lake Louise criteria [4].

2.2. Clinical follow-up

Follow-up was defined as time from ICD implantation until 31 st of December 2016 or end of repeated clinical evaluation. Primary endpoint was time to first event [anti-tachycardia pacing (ATP), appropriate ICD therapy, hospital admission with heart failure (HF) or death]. ATP was included to accommodate for different programming of the ICDs. Hospital admission with HF was included, as a proportion of the study population were diagnosed with cardiomyopathy.

2.3. Image acquisition

CMR scans were performed with a 1.5 T Philips Achieva dStream scanner (Philips Medical Systems, Best, The Netherlands) (n = 24), 3 T Siemens Skyra scanner (Siemens Healthcare, Erlangen, Germany) (n = 10), or 1.5 T GE Signa scanner (GE Medical Systems, Milwaukee, Wisconsin) (n = 2). Patients were scanned 6 days [interquartile range (IQR) 4–10] after conversion to sinus rhythm. A typical imaging protocol consisted of a survey scan followed by ECG-triggered steady-state free precession (SSFP) cine images obtained during breath hold. The following images were acquired: vertical long axis corresponding to a 2-chamber view, horizontal long axis, short-axis images covering the entire LV, 3-chamber, and 4-chamber views. Slice thickness and slice gap were 8 mm and 0 mm, respectively. Late gadolinium enhancement (LGE) imaging was started ten minutes after IV administration of 0.1 mmol/kg gadobutrol (Gadovist, Bayer HealthCare, Berlin, Germany). A 3D phase-sensitive inversion recovery (PSIR) pulse sequence with navigator-based respiratory gating and ECG-triggering was used (Philips MRI scanner). On Siemens and GE scanners, ECG-triggered 2D inversion recovery gradient echo sequence during breath hold was used. Short-axis, 2-chamber, and 4-chamber views were acquired.

2.4. Image analysis

LV and right ventricular (RV) volume data were derived from short-axis cine images using semiautomatic tracing of the endo- and epicardium in end-diastole and end-systole using dedicated software Segment v1.9 (Medviso AB, Lund, Sweden). LV myocardial volume was calculated as the difference between epicardial and endocardial LV volumes in diastole. Papillary muscles were included in the blood volume. LV mass was calculated as a product of LV myocardial volume multiplied by density of myocardium (1.05 g/ml). LGE was quantified by automatic algorithm available in the Segment software. Upon visual agreement between the investigators (WYK, TZ), a threshold of 3.5 standard deviations from remote was used to identify the hyper-enhanced myocardium. Manual corrections were afterwards applied if deemed necessary. Prevalence of LGE was calculated on a segment basis, using a 17-segment model as proposed by the American Heart Association (AHA) [5]. However, due to inconsistent availability of strain data for the apical (17) segment, this segment was excluded from the study. LV myocardial deformation analysis was performed on SSFP cine images using CMR feature tracking software 2D CPA MR (Tomtec Imaging Systems, Munich, Germany) [6,7]. Endocardial and epicardial LV borders were drawn manually in the end-diastolic frame and then propagated automatically to the rest of the cardiac cycle. Manual adjustments of the borders were performed in the end-diastolic frame in case of inadequate initial automatic tracking. Short-axis cines at basal, midventricular and apical levels were used. Midventricular slice with a clear presence of both papillary muscles was chosen first. Afterwards, two equidistal slices were selected, with myocardium visible as a circumference at basal and at apical levels both in diastole and in systole. Long-axis images at 2-chamber, 3-chamber, and 4-chamber projections were used. In case one of the long-axis images was missing, only the two remaining images were included. Longitudinal, radial, and circumferential strains and strain rates were then calculated. Circumferential and longitudinal values were calculated both for endocardium and epicardium. Short-axis images were used for circumferential and for radial measurements, while long-axis images were used for the assessment of longitudinal strain and strain rate (SR). Peak values were recorded for every segment according to the AHA 17-segment model. To minimise the subjectivity in the selection of the systolic peaks, any presystolic or post-systolic shortening was included in the assessment [8]. Global LV radial, circumferential and longitudinal strains and strain rates were calculated as averages of the segmental peak values [9]. All volumetric, LGE and deformation image analyses were performed by an experienced observer (TZ) blinded for all clinical data of the patients.

2.5. Statistics

Continuous variables are reported as median and IQR or mean ± SD. Categorical variables are reported as absolute numbers and percentages. Continuous variables were compared by the two-sample *t*-test or by the Mann-Whitney U test. Chi-square test was used to evaluate categorical variables. One-sample Z-test was used to compare the findings with previously published normal reference materials [10,11]. Two-sided tests with $p < 0.05$ were considered as statistically significant. The analyses were performed using R version 3.2.4 (The R Foundation for Statistical Computing, Vienna, Austria).

Intra-observer agreement for global strain and SR was assessed after performing feature tracking analysis 30 days later by the same operator in 8 randomly selected patients. Intraclass correlation coefficients (ICCs) and 95% confidence intervals (CIs) were calculated.

3. Results

Baseline clinical characteristics and echocardiographic data are provided in Table 1. The majority of the patients were males (69%), and the most common primary arrhythmia was VF (56%) followed by VT (44%). Cardiomyopathy patients had the following diagnoses: arrhythmogenic right ventricular cardiomyopathy (ARVC) in 6 (17%), dilated cardiomyopathy (DCM) in 7 (19%), and hypertrophic cardiomyopathy (HCM) in 3 (8%) cases. In arrhythmia patients without cardiomyopathy diagnosis, the majority were diagnosed with idiopathic VT/VF in 14 (39%) patients, while the remaining patients had catecholaminergic polymorphic ventricular tachycardia (CPVT) in 2 (6%), early repolarisation syndrome in 2 (6%), long QT syndrome in 1 (3%),

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