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Sudden cardiac arrest: focus on cardiac magnetic resonance

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INTRODUCTION

Untreated sudden cardiac arrest (SCA) leads to sudden cardiac death (SCD), accounting for 15%–20% of all deaths [1]. Cardiovascular diseases, particularly coronary artery disease, have been reported to be the most common cause of SCD [2], except in individuals younger than 30 years in whom the predominant diagnosis is unexpected, idiopathic cardiac death [3].

Cardiac magnetic resonance (CMR) is suitable for the evaluation of cardiac function and structure, providing deeper insight than more widely available echocardiography. The aim of this study was to investigate the role of CMR in a group of survivors of unexplained SCA.

METHODS

We retrospectively reviewed the medical records (electrocardiography, echocardiography, coronary angiography/coronary artery computed tomography, electrophysiology study) of all patients who underwent a CMR study between 2008–2017 in our hospital. In this study, we enrolled SCA survivors with an initially (before the CMR exam) unexplained or unclear cause of SCA. We included patients with a normal or slightly decreased ejection fraction, as

worsening systolic function could be caused by SCA. The study was approved by the local ethics committee. All participants gave written informed consent for the CMR study.

Cardiac magnetic resonance study

Cardiac magnetic resonance studies were performed on a 1.5 Tesla scanner (Avanto or Avanto^{fit}, Siemens, Erlangen, Germany) using breath-hold cines in long-axis planes and sequential short-axis slices. All patients underwent a CMR study with a gadolinium-based intravenous contrast agent at standard dosage.

Each study was reported by a team of at least two skilled investigators. The final diagnoses were made using all available data.

Statistical analysis

Statistical analysis was performed using MedCalc software, version 10.03 (Ostend, Belgium). Student's t-test was used to compare normally distributed variances. P <0.05 was considered significant. Non-normally distributed data were tested using the Mann–Whitney test. P <0.05 was considered significant.

RESULTS AND DISCUSSION

From all consecutive patients referred for CMR in our hospital between 2008 and 2017 (n = 8630), 57 patients were referred due to sudden cardiac arrest. We included 31 patients (16 male) in whom the SCA cause was unclear before CMR. The mean age (standard deviation [SD]) was 36.7 (12.2) years, and the mean (SD) left ventricular ejection fraction was 59.8 (7.6%). Late gadolinium enhancement (LGE) was observed in 13 cases (41.9%), whereas in 1 case, right ventricular LGE was present.

Diagnoses

Cardiomyopathies were diagnosed in 3 patients: one patient was found to have dilated cardiomyopathy (DCM), one had left ventricular non-compaction (LVNC), and one had arrhythmogenic right ventricular cardiomyopathy. In patients with DCM and arrhythmogenic right ventricular cardiomyopathy, LGE was observed. All of them had SCA as the first symptom of heart disease, and echocardiography was inconclusive.

One patient with a normal CMR scan was diagnosed with anorexia and hypokalemia (serum potassium level <2 mmol/l) as the trigger causing SCA. In the remaining 27 (87.1%) patients, the CMR function was grossly normal, and those patients were rated as having idiopathic

ventricular fibrillation (IVF). In 11 of them, a small patch of LGE was seen (Figure 1A–D). Late gadolinium enhancement type, localization and extension in this group indicated no pattern (Figure 1E). Fibrosis was found in the basal, mid septal, inferior, lateral and inferior segments as well as in the apex. Three patients had LGE in the inferior right ventricular insertion point.

There were no statistically significant differences in patients with normal cardiac function with and without fibrosis comparing age (mean [SD]: accordingly 38.8 [12.2] years; 37.6 [13.0] years; $P = 0.81$), left ventricular ejection fraction (mean [SD]: accordingly 57.7 [6.8%]; 62.3 [7.0%]; $P = 0.12$), left ventricular end-diastolic volumes (median (interquartile range): accordingly 82.0 (68.7–97.9) ml/m^2; 82.5 (76.8–94.4) ml/m^2; $P = 0.54$) and left ventricular end-systolic volume (mean [SD]: accordingly 34.2 (10.3) ml/m^2; 33.7 (11.0) ml/m^2; $P = 0.90$).

DISCUSSION

SCA is a major medical problem associated with poor outcomes, with a survival rate of only 9.6% [4]. In Poland, the out-of-hospital cardiac arrest incidence is 69.7 per 100 000 inhibitants [5]. Many cardiovascular diseases are associated with an increased risk of SCA events.

Our cohort consisted of patients with normal or inconclusive echocardiographic findings, whereas in 3 (9.7%) of them cardiomyopathy was diagnosed by CMR. In the rest of them mechanical cardiac function was grossly normal. In a large cohort of patients with normal echocardiography and ventricular arrhythmias (including aborted sudden cardiac arrest) [6], structural heart disease was found in 25.5% of patients.

In patients with idiopathic DCM, approximately 30% of deaths are sudden [7] and strongly correlated with the presence of LGE [8]. LVNC is associated with a higher risk of SCD and ventricular arrhythmias than age-matched DCM [9]. Arrhythmias in patients with LVNC occur even if LGE is not present [10]. Accordingly, in patients with arrhythmogenic right ventricular cardiomyopathy, life-threatening arrhythmias can occur before histological and imaging features [11].

In 87.1% of patients with normal cardiac function, there were no other causes according to a previous (pre-CMR) investigation. The CMR study led to the finding of a potential unstable arrhythmic substrate (represented by LGE) that might cause VT/VF leading to SCA in 11 of 27 patients with IVF.

In multiple studies analyzing causes of sudden cardiac death in the US and in European countries (Italy, Denmark and the United Kingdom), the most common was unexplained SCD in patients with structurally normal hearts [12]. In the out-of-hospital cardiac arrest real-life

cohort registry, IVF was diagnosed in 6.8% of SCAs among all survivors of SCAs with cardiac etiologies [13].

SCA survivors with CMR-based diagnosis have been found to have a worse prognosis than patients with a normal scan or minor nonspecific changes [14]. Moreover, the presence and extent of LGE correlates with a worse prognosis [15].

Limitation and strength

To our knowledge, this is the first study evaluating the CMR value in survivors of SCA of unknown aetiology in a Polish population. This study was conducted within the context of a limited cohort and retrospective analyses. Patients were referred for CMR study by a primary physician. We retrospectively recruited patients after SCA with good outcomes. Consequently, we examined only SCA survivors who did not represent the whole population with SCA. This could explain why our cohort consisted mostly of young people without comorbidities and why IVF was so frequent.

CONCLUSION

In a cohort of unexplained SCA survivors, the CMR study led to the finding underlying cardiomyopathy in 3 patients (9.7%) and to find potential arrhythmic substrates in 11 (35.5%) patients with idiopathic ventricular fibrillation.

In 16 (51.6%) patients with IVF CMR scan was normal.

The role of CMR in SCA survivors needs further investigation.

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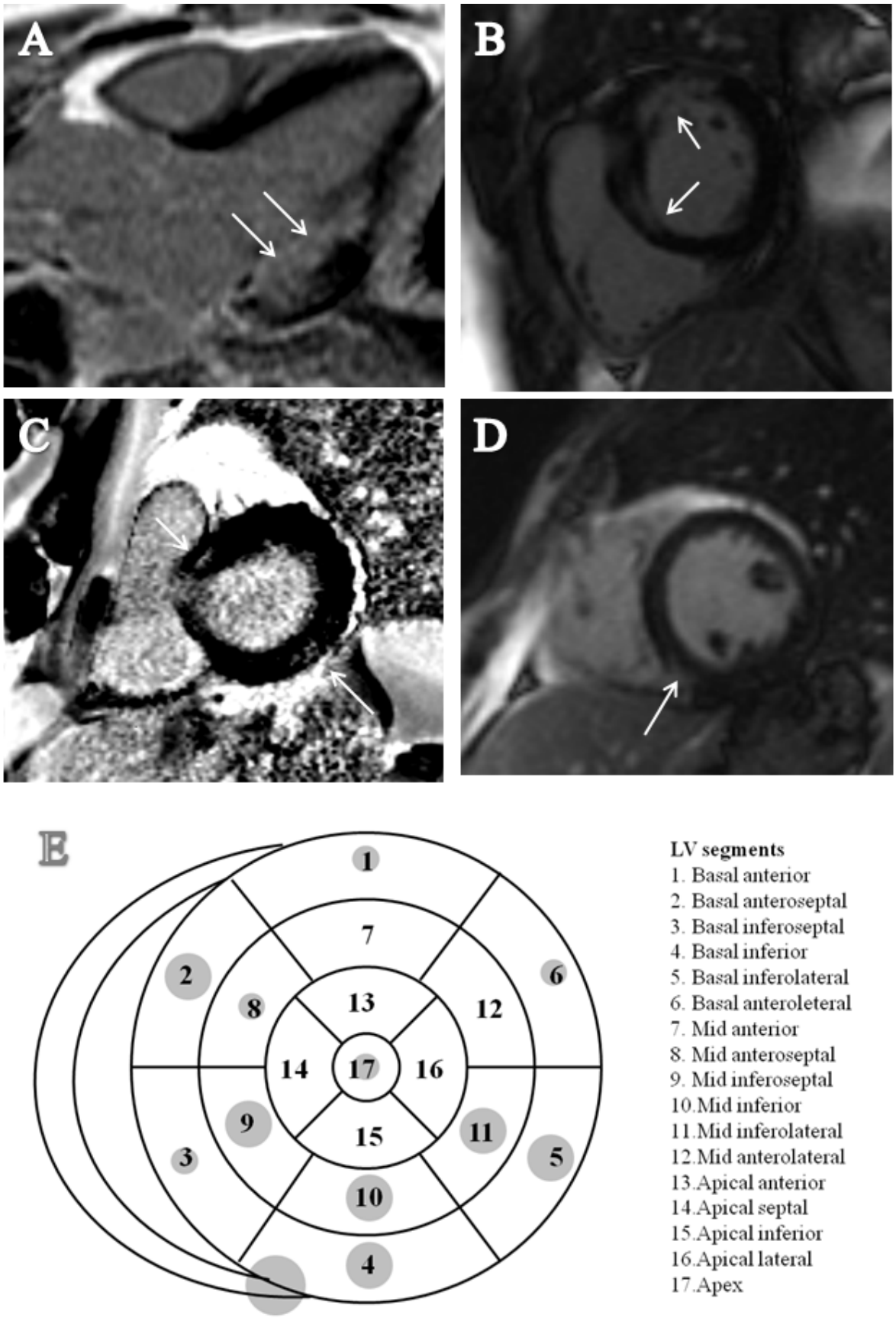

Figure 1. A–D. Examples of LGE found in SCA survivors with normal cardiac function (arrows). **A.** Three-chamber view of the subendocardial pattern of LGE in a 35-year-old female

patient. **B.** Midventricular short axis image of midwall and subendocardial LGE in a 31-year-old male patient. **C.** Short axis view of the basal midwall and subepicardial LGE in a 49-year-old male patient. **D.** Short axis view of midventricular midwall LGE in a 27-year-old male patient. **E.** LGE pattern in patients with IVF. Gray dots show localization of fibrosis and their size corresponds to the number of patients with fibrosis found in specific segments

Abbreviations: IVF, idiopathic ventricular fibrillation; LGE, late gadolinium enhancement; LV, left ventricular; SCA, sudden cardiac arrest