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Clinical paper

Out of hospital cardiac arrest survivors with inconclusive coronary angiogram: Impact of cardiovascular magnetic resonance on clinical management and decision-making☆

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ABSTRACT

Background: Non-traumatic out of hospital cardiac arrest (OHCA) is the leading cause of death worldwide, mainly due to acute coronary syndromes. Urgent coronary angiography with view to revascularisation is recommended in patients with suspected acute coronary syndrome. Diagnosis and management of patients with inconclusive coronary angiogram (unobstructed coronaries or unidentified culprit lesion) is challenging. We sought to assess the role of Cardiovascular Magnetic Resonance (CMR) in the diagnosis and management of OHCA survivors with an inconclusive coronary angiogram.
Methods and results: This is a retrospective multicentre CMR registry analysis of OHCA survivors with an inconclusive angiogram. Clinical, ECG and multi-modality imaging data were analysed. Clinical impact of CMR was defined as a change in diagnosis or management. Out of 174 OHCA survivors referred for CMR, 110 patients (63%, 84 male, median age 58) had an inconclusive angiogram. CMR identified a pathologic substrate in 76/110 patients (69%): ischemic heart disease was found in 45 (41%) and non-ischemic heart disease in 31 (28%). A structurally normal heart was found in 25 patients (23%) and non-specific findings in 9 (8%). As compared to trans-thoracic echocardiogram, CMR proved to be superior in identifying a pathologic substrate (69% vs 54%, p = 0.018). The CMR study carried a clinical impact in 70% of patients, determining a change in diagnosis in 25%, in management in 29% and a change in both in 16%.
Conclusions: CMR showed a promising role in the diagnostic work-up of OHCA survivors with inconclusive angiogram and its wider use should be considered.

Introduction

Non-traumatic of hospital cardiac arrest (OHCA) is the leading cause of death worldwide [1–3] with an estimated incidence of 0.5/1000 person year [4–8]. Acute coronary syndromes (ACS) account for more than 2/3 of cases [9–12]. According to AHA guidelines, urgent angiography with view to primary percutaneous coronary intervention is a class IB recommendation in patients with resuscitated cardiac arrest whose electrocardiogram (ECG) shows ST elevation (STE) myocardial infarction (MI) [13]. Given the high incidence of underlying coronary artery disease (CAD) in this group of patients, European guidelines extended the recommendation to incorporate patients without diagnostic STE, but with high suspicion of on-going infarction (class IIaB) [14]. However non-ischemic cardiomyopathy accounts for up to 15% of OHCA [15–19] and a structurally normal heart can be found in up to 10–20% of cases [20–23]. While evidence of culprit lesion on angiogram supports acute ischemia as the cause of OHCA, diagnosis and clinical management of OHCA survivors with inconclusive coronary angiogram (either non-identifiable culprit lesion or unobstructed coronary arteries) is challenging. Cardiovascular magnetic resonance (CMR) is a non-invasive imaging technique providing accurate diagnosis based on its superior spatial resolution and unique non-invasive tissue characterization.

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We sought to assess the additional role and clinical impact of CMR in the diagnosis and management of OHCA survivors with an inconclusive coronary angiogram.

Materials and methods

The CMR registries from two tertiary Cardiac centres (Bristol, South West of England and Padua, Veneto Region, Italy) were analysed to identify OHCA survivors who underwent urgent coronary angiogram followed by CMR (October 2009-November 2015). The study focused on the analysis of patients with an "inconclusive angiogram", defined as evidence of stable obstructive CAD (SCAD) with no culprit lesion or unobstructed coronaries (normal coronaries/non-obstructive CAD). Culprit lesion was defined as obstructive (≥70%) CAD with TIMI 0/1 flow with abrupt closure, or TIMI 2/3 flow with features suggestive of thrombus/ulcerated plaques, ST segment-T wave changes in the corresponding ECG location, and evidence of matching regional wall motion abnormality on left ventriculogram or echocardiogram [24].

CMR

CMR was performed on a 1.5T scanner (Avanto, Siemens Healthcare, Germany) with a protocol including long and short axis cine sequences and post-contrast imaging, performed ten minutes after intravenous administration of 0.1 mmol/Kg of Gadobutrol (Gadovist 1.0 mmol/ml, Bayer-Schering, Berlin, Germany) in identical planes to cine images. Additional sequences for the assessment of myocardial oedema (T2-short tau inversion recovery, T2-STIR) or myocardial ischemia (stress perfusion with 140–210 ug/Kg/min adenosine) were performed when indicated, based on clinical and angiographic findings. Ventricular function was assessed with dedicated software (Circle Cardiovascular Imaging, Calgary, Canada), by tracing endo- and epicardial borders on each short axis cine slice in end-diastole and end-systole. All volumes were indexed to body surface area. The localization, extent and distribution pattern of late gadolinium enhancement (LGE) were assessed by using short- and long-axis views and confirmed only if detectable in two orthogonal planes. The pattern of LGE distribution was defined as ischemic, subendocardial or transmural, if involving <50% or ≥50% of wall thickness, respectively, and as mid-wall/epicardial if patchy/spotty intra-mural or sub-epicardial enhancement was detected. The presence of LGE at the right ventricle/left ventricle insertion points, in the absence of other distribution patterns, was defined as non-specific findings, as its diagnostic and prognostic meaning is still unclear.

All the analyses were carried out in accordance with the recommendation of the Society for Cardiovascular Magnetic Resonance [25]. The study was reviewed by the local Institutional Research and Innovation Department and in view of the retrospective design, formal ethical approval was waived off. All patients gave written informed consent.

Clinical impact

Clinical, ECG and echocardiographic data were collected and independently analysed by two clinicians blinded to CMR findings. A diagnosis was made based on clinical and imaging data available prior to CMR. According to previously used definitions [26], "clinical impact" of CMR was defined as change in diagnosis, compared to the composite pre-CMR diagnosis, or change in management. A change in management was defined as CMR findings either leading to change in medication, to an invasive procedure (i.e. repeat angiogram, myocardial revascularization, ICD implantation) or to the avoidance of such invasive procedures. Patients with a change both in diagnosis and management were only counted once.

Statistical analysis

Continuous and categorical variables are expressed as mean ± SD or median (IQR), and n (%), respectively. Categorical variables were compared by using the chi-square or Fisher exact test, as appropriate. Continuous data were compared by using the 2-tailed unpaired *t*-test (for normally distributed data sets) or by using the Mann-Whitney *U* test. Inter-rater agreement for categorical variables was assessed by Cohen's kappa coefficient. A p-value of <0.05 was considered statistically significant. Data were analysed with SPSS® version 23 (IBM®).

Results

Clinical characteristics

Out of 174 consecutive OHCA survivors referred to CMR after coronary angiogram (performed on same day of admission, IQR 0-2 days), 110 patients (63%, 84 male, age 58 years, IQR 46–68) had an inconclusive angiogram and were enrolled in the study: 37 patients (34%) had evidence of SCAD with no culprit lesion and 73 patients (66%) showed unobstructed coronaries. The first registered rhythm was ventricular tachycardia (VT)/ventricular fibrillation (VF) in 104 patients (95%) and pulseless electrical activity (PEA) in 6 patients (5%). The first ECG was available in 86 patients (78%): non-ST eleva-

Table 1
Baseline patient characteristics based on angiogram findings.

	Total Cohortn = 110	SCAD withNo Culprit Lesionn = 37	Unobstructed Coronariesn = 73	p-value
Male	84 (76)	34 (92)	50 (68)	0.006
Age, years	58 (46–68)	65 (58–75)	52 (40–63)	<0.001
Hypertension	36/100 (36)	20/35 (57)	16/65 (25)	0.001
Diabetes	13/100 (13)	7/35 (20)	6/65 (9)	0.127
Active smoking	25/100 (25)	11/35 (31)	14/65 (22)	0.276
Hyperlipidaemia	20/100 (20)	10/35 (29)	10/65 (15)	0.116
Family history CAD	9/100 (9)	4/35 (11)	5/65 (8)	0.533
Family history SCD	2/100 (2)	0/35 (0)	2/65 (3)	0.295
Previous CAD	24/100 (24)	17/35 (49)	7/65 (11)	<0.001
Previous NIHD	5/100 (5)	1/35 (3)	4/65 (6)	0.471
First rhythm, VT/VF	104/110 (95)	34/37 (92)	70/73 (96)	0.383
First rhythm, PEA	6/110 (5)	3/37 (8)	3/73 (4)	0.382
First ECG post ROSC, STE	18/86 (21)	11/27 (41)	7/59 (12)	0.002
First ECG post ROSC, non-STE	68/86 (79)	16/27 (59)	52/59 (88)	0.002

Values are n (%) or median (interquartile range).
CAD, coronary artery disease; SCD, sudden cardiac death; NIHD, non-ischemic heart disease; VF, ventricular fibrillation; VT, ventricular tachycardia; PEA, pulseless electrical activity; ROSC, return of spontaneous circulation; SCAD, stable coronary artery disease; STE, ST segment elevation; NSTE, non-ST segment elevation.