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FULL PAPER

Prevalence and clinical relevance of the morphological substrate of ventricular arrhythmias in patients without known cardiac conditions detected by cardiovascular MR

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Objective: Cardiac MR (CMR) identifies the substrate of ventricular arrhythmia (VA) in cardiomyopathies and coronary heart disease. However, little is known about the value of CMR in patients with VA without previously known cardiac disorders.

Methods: 76 patients with VA (Lown ≥ 2) without known cardiac disease after regular diagnostic work-up were studied with CMR, and findings were correlated with electrocardiogram (ECG) and electrophysiological stimulation (EPS). Structural abnormalities matching the VA origin as defined by ECG and/or EPS, or a CMR-detected cardiac condition known to cause arrhythmia were defined as VA substrate. CMR findings were defined as clinically relevant, if resulting in a new diagnosis, change of treatment or additional diagnostic procedure.

Results: 44/76 patients demonstrated pathological CMR findings. In 24/76 patients, the pathology was detected

by CMR and not by echocardiography. CMR-based diagnoses of cardiac disease were established in 20/76 patients, and all were morphological substrates for VA. In seven patients, the location of the CMR finding (scar) directly matched the VA origin. CMR findings resulted in a change of treatment in 21 patients and/or additional diagnostics in 8 patients.

Conclusion: Undetected cardiac conditions are frequent causes of VA. This is the first study demonstrating the value of CMR for detection of morphological substrate and/or underlying cardiac disorders in VA patients without known cardiac disease.

Advances in knowledge: The high incidence of clinically relevant CMR findings which were not detected during initial diagnostic work-up strongly supports the use of CMR to screen VA patients for underlying heart disease.

Although the value of cardiac MR (CMR) for the diagnosis of cardiac diseases such as myocarditis is undisputed, CMR is also predictive of patients at high risk for ventricular arrhythmias (VAs) with conditions such as hypertrophic cardiomyopathy (HCM) and coronary heart disease (CHD).^{1–3} Recent studies have demonstrated the ability of CMR to identify the anatomical correlate of VA in those patients. This anatomical correlate has been characterized by CMR as a structural abnormality (e.g. fibrosis or peri-infarct region), which may go undetected using other non-invasive imaging modalities.^{4,5} A number of studies have been undertaken, or are ongoing, to further elucidate the added value of CMR in patients with known cardiac conditions, to improve risk stratification for VA and to optimize therapy.^{1,6–8} However, little is

known to date regarding the added value of CMR for detection of an arrhythmogenic substrate or underlying cardiac condition in patients who present with VAs without known cardiac disease.

Thus, the purpose of this study was to investigate the added value of CMR in patients with VAs for detection of underlying heart disease and an arrhythmogenic morphological substrate, and also to investigate the clinical relevance of CMR in those patients with positive findings.

METHODS

Patient population

The local institutional review board approved this retrospective study.

At the authors' institution (University of Bonn), it is common practice to schedule all patients with VAs for a CMR examination if the aetiology of arrhythmia is unclear.

A total of 168 patients with cardiac arrhythmias undergoing a CMR examination between February 2008 and September 2010 were screened for this study. In all but two patients, CMR was successfully completed (one case of scanner malfunction and one case of patient's claustrophobia). Only those patients with high-grade VA without a known cardiac condition at the time of the CMR investigation were included in the study, based on the patients' histories and clinical presentations. Patients with, for example, a clinical presentation of CHD or myocarditis were excluded. High-grade VAs were defined as premature ventricular beats (PVBs) Lown ≥ 2 , ventricular tachycardia (VT), ventricular flutter (VF) or ventricular fibrillation. The Lown classification categorizes PVBs according to their frequency and complexity. Thus, only patients with frequent PVBs (>30 per hour), polymorphic PVBs, repetitive PVBs or early R on T PVB (Lown ≥ 2), as well as VT, VF or ventricular fibrillation were included.

29 patients were excluded from the study because of previously diagnosed underlying CHD ($n = 26$) or other previously documented cardiac condition ($n = 3$). In another 45 cases, the patient's history was not available/incomplete, and thus underlying cardiac diseases (*e.g.* CHD and myocarditis) could not be excluded, or no echocardiography was available ($n = 21$). 18 patients were excluded because they did not reveal higher grade VA in the electrocardiogram (ECG) or Holter ECG documentation, but, for example, atrial fibrillation, low-grade ventricular premature beats (Lown < 2) or unclear syncope(s) without any documentation of any cardiac arrhythmia.

Thus, a total of 76 patients (mean age, 45 ± 17 years; 39 males and 37 females) were included in the retrospective study, fulfilling

the inclusion criteria of high-grade VA without known underlying cardiac conditions following routine work-up (Figure 1).

Diagnostic and imaging procedures

Cardiac MR imaging

Imaging was performed on a clinical 1.5-T MR scanner (Philips Healthcare, Best, Netherlands). CMR studies included an axial steady-state free precession (SSFP) sequence covering the entire patient's chest; functional SSFP cine sequences covering the entire cardiac short axis; as well as horizontal, vertical and long axis views. In all patients, T_2 weighted fat-suppressed turbo spin echo (short tau inversion-recovery) images were acquired in the short axis orientation and late gadolinium enhancement (LGE) imaging was performed in the short, horizontal and vertical long axis orientation 12–15 min after injection of an extracellular contrast agent (0.2 mmol kg^{-1} of Magnevist; Bayer Vital GmbH, Leverkusen, German) for myocardial tissue characterization. In patients with arrhythmia originating from the right ventricle, additional functional axial cine sequences were acquired, covering the entire right ventricle from the diaphragmatic wall to the right ventricular outflow tract.

Transthoracic echocardiography

For the initial diagnostic work-up, comprehensive M-mode and two-dimensional echocardiographic examinations were performed in all patients with a commercially available system (iE33; Philips Healthcare). According to the American Society of Echocardiography guidelines, the left and right ventricular dimensions at end-diastole and end-systole were measured in the apical four-chamber view to calculate the ejection fraction. The long and short parasternal axis as well as the apical axis in four-, three- and two-chamber views were recorded for the assessment of wall motion, wall thickness and valvular morphology and function. Colour Doppler imaging as well as pulsed-wave and continuous-wave Doppler analyses were used

Figure 1. An overview of the study. CD, cardiac disease; CHD, coronary heart disease; CMR, cardiac MR; VA, ventricular arrhythmia.

to further assess valve disease, pulmonary artery pressure and diastolic dysfunction. Subxiphoidal or subcostal views were recorded to detect pericardial effusion and assess the width of the vena cava and response to inspiration.

Electrocardiography

A conventional 12-lead ECG was performed in all patients at the time of hospital admission. In addition, standard 24-h Holter ECG monitoring was performed in patients when arrhythmias were not detected on the 12-lead ECG.

Electrophysiology

Electrophysiological stimulation (EPS) was performed in 49/76 patients (65%). All patients underwent electrophysiological testing by standard techniques. Before the electrophysiological study, antiarrhythmic medication was discontinued for at least five drug half-lives in all patients. Electrodes were positioned in the high right atrium, at the bundle of His and at the right ventricular apex. Routine atrial and ventricular stimulation studies were performed to assess atrioventricular (AV) conduction. Inducibility of VT was assessed with the use of programmed single, double and triple extrastimuli introduced in sinus rhythm and at paced cycle lengths of 600, 500 and 400 ms. Ventricular stimulation was applied to the right ventricular apex and the right ventricular out-flow tract if stimulation at the first site failed to initiate tachycardia. In the absence of inducible VA, the protocol was repeated with isoproterenol infusion ($200 \mu\text{g min}^{-1}$).

Definition of clinical relevance

For data analysis, the complete patient records and charts including all findings of diagnostic procedures and laboratory analysis were used, including nursing records (by AL and JWT). In the case of equivocal imaging findings or diagnostic records, the original data were reassessed by an experienced cardiologist (11 years' clinical cardiology experience) or a radiologist (10 years' clinical radiology experience including CMR).

Diagnosis of cardiomyopathy by CMR and interpretation of LGE images followed standard clinical practice.^{9,10} CMR findings were correlated with ECG findings and, if available, EPS findings. A morphological substrate was defined as a pathological CMR finding, known to cause associated arrhythmia (e.g. cardiomyopathies). If the location of a structural abnormality or scar tissue, as defined by LGE, directly matched the origin of the VA, as defined by ECG and/or EPS, the finding was judged to be an electroanatomical correlate.

To assess the clinical relevance of the CMR study, all positive CMR studies were assessed for their influence on patient treatment, as previously described by others.¹¹

A positive CMR finding was defined as clinically relevant if (1) the finding resulted in a new diagnosis, (2) it had an impact on patient treatment (change of medical or invasive therapy) or (3) it resulted in additional diagnostic procedures.

Statistical analysis

Data were analysed using a commercially available software program (SPSS® v. 18; SPSS Inc., Chicago, IL). Data are expressed as

absolute frequencies or relative percentages for categorical variables.

RESULTS

The demographic patient characteristics and distribution of types of arrhythmia are given in Table 1.

Imaging findings

Pathological or abnormal CMR findings were found in a total of 44 (58%) patients. The findings were wall motion abnormalities [right ventricle (RV): $n = 6$; left ventricle (LV): $n = 18$; total = 24, 31.6%], concentric ($n = 7$) or eccentric ($n = 3$) ventricular hypertrophy ($n = 10$, 13.2%), dilatation [LV: $n = 12$; RV: $n = 10$; left atrium (LA), $n = 6$; total = 28, 36.8%], wall thinning ($n = 3$, 3.9%), pericardial and/or pleural effusion ($n = 7$, 9.2%), LGE

Table 1. Characteristics of patients with ventricular arrhythmias

Characteristic	No. of patients (%)
Demographic patient data	
Patients (n)	76
Gender	
Female (n)	37 (48.7%)
Male (n)	39 (51.3%)
Mean age (years)	45 ± 16.7
Range (age; years)	15–79
Type of ventricular arrhythmia	
PVBs ($\geq$ Lown 2)	30 (39.5%)
Monomorphic PVB (Holter registration)	3 (3.9%)
Monomorphic PVB, LBBB configuration	18 (23.4%)
Monomorphic PVB, RBBB configuration	8 (10.5%)
Polymorphic PVB	1 (1.3%)
VT	38 (50.0%)
Non-sustained	19 (25.0%)
Sustained	19 (25.0%)
VT (Holter registration)	10 (13.2%)
Monomorphic VT (LBBB configuration)	9 (11.8%)
Monomorphic VT (RBBB configuration)	8 (10.5%)
Polymorphic VT (with or without QT interval prolongation)	8 (10.5%)
Irregular broad complexes tachycardia	3 (3.9%)
Ventricular flutter	1 (1.3%)
Ventricular fibrillation	7 (9.2%)

LBBB, left bundle branch block; PVB, premature ventricular beat; RBBB, right bundle branch block; VT, ventricular tachycardia.

consistent with myocardial inflammation or scar or structural abnormalities ($n = 15$, 19.7%), increased trabeculation ($n = 2$, 2.6%) and myocardial clefts ($n = 2$, 2.6%). Additional extracardiac findings were detected in three more patients (liver cyst and haemangioma, pneumonia).

Compared with echocardiography, pathological findings were exclusively detected by CMR in 24 patients (31.6%). These pathological findings were, in detail: scar ($n = 9$), dilatation (RV: $n = 6$; LV: $n = 3$), wall motion abnormalities ($n = 6$), myocardial inflammation/oedema/relative delayed enhancement ($n = 5$), structural abnormality ($n = 2$) and increased trabeculation ($n = 1$).

Based on CMR findings, a diagnosis of heart disease was established in a total of 20 patients (20/76, 26.3%). The range of diagnoses is presented in Table 2, with inflammatory findings discovered most frequently. In all but the three patients with dilated cardiomyopathy (DCM) and one patient with findings consistent with arrhythmogenic right ventricular cardiomyopathy (ARVC) (16/20, 80%), the final diagnosis could only be determined from CMR (16/76, 21% of all patients) and not from echocardiography. There was no instance where a diagnosis based on echo findings was missed by CMR.

Figures 2–4 are an example of a patient with a positive CMR finding and without previously known cardiac disease. The otherwise healthy patient (a 41-year-old male) presented at the emergency room with a non-sustained VT episode after physical activity, which was haemodynamically tolerated. Figure 2 demonstrates the recorded ECG and VT upon admission with a right bundle branch morphology of inferior/septal origin (negative axis in leads II, III + aVF, positive axis in leads I + aVL). There was no cardiac enzyme abnormality at the time of admission. Echocardiography revealed normal global myocardial function and minimal hypokinesis in the apical anterior wall. A CMR study was performed revealing scarring throughout the entire left ventricle, predominantly located in the midventricular subepicardial inferoseptal and anteroseptal walls (Figure 3). Ejection fraction was in the (low) normal range (60%) with a normal end-diastolic volume. No oedema or pericardial effusion was seen. A diagnosis of diffuse post-inflammatory scarring was made, matching the patient's history of a reported severe influenza-like illness with prolonged

convalescence 1 year earlier. The patient underwent EPS (Figure 4), which demonstrated a pleomorphic-onset VT followed by VT originating from the inferoseptal axis and pleomorphic salvages. The results of the CMR study, supported by EPS, led to the diagnosis of previous myocarditis, and a cardioverter defibrillator (ICD) device was subsequently implanted.

Figure 5 shows additional exemplary cases. The patient in Figure 5a had repetitive tachycardia. CHD had been excluded by coronary angiography. CMR detected extensive perimyocarditis scarring with mild wall motion abnormality. Figure 5b shows CMR images from a patient with VT following heavy physical stress. Echo revealed good LV function and significant CHD was invasively excluded. The CMR study revealed post-myocardial infarction scar in the inferior basal wall correlating with the origin of the documented VT (EPS).

Morphological substrate

All CMR-based diagnoses ($n = 20$) were classified as morphological substrates based on the predefined criteria (Table 3). In seven patients, the location of the relevant CMR finding (myocardial scar/structural abnormality) directly matched the origin of the VA as defined by ECG ($n = 2$) or EPS ($n = 5$). In none of these patients was the relevant finding detected by echocardiography. A substrate for the underlying arrhythmia was detected in 3/30 patients with isolated PVBs (10%), in 5/19 patients with non-sustained VTs (26%), in 7/19 patients with sustained or incessant VTs (37%) and in 5/8 patients with VF or fibrillation (63%).

Clinical relevance

The CMR findings were judged to be clinically relevant in a total of 26/76 patients (34.2%; Table 4). In 23/76 patients, these findings were only detected by CMR but not by echocardiography (30%).

A new diagnosis was found in a total of 20 patients (26.3%) (Table 2). A new diagnosis or pathological MRI finding had an impact on treatment in 21 patients (27.6%) and/or resulted in an additional diagnostic procedure in 8 patients (10.5%) (Table 4).

DISCUSSION

Previous studies have demonstrated the value of CMR for risk stratification for VAs in patients with known cardiac conditions. However, this is the first study revealing the utility of CMR in detecting an underlying cardiac condition and morphological substrate in patients with lower and high-grade VAs without previously known cardiac disease.

Cardiac conditions and morphological substrate of arrhythmia

The cardiac conditions that were detected by CMR in this study are all known to be causative for VAs. The most frequent cardiac condition was the presence of acute myocarditis and evidence of post-inflammatory scar. Although patients with acute myocarditis usually present with a typical clinical history and symptoms, the disease may well present with an arrhythmic episode (e.g. VT and heart block) as the heralding event.^{12–15} In a study investigating aborted sudden cardiac death (SCD) and high-grade

Table 2. Diagnoses obtained by cardiac MR (CMR)

Diagnosis	No. of patients (%)
Dilated cardiomyopathy	3 (3.9%)
Hypertrophic cardiomyopathy	1 (1.3%)
CMR criteria for ARVC	3 (3.9%)
Post-myocarditis scar	5 (6.6%)
Post-myocardial infarction scar	2 (2.6%)
Myocarditis	6 (7.9%)
Total	20 (26.3%)

ARVC, arrhythmogenic right ventricular cardiomyopathy.

Figure 2. Electrocardiogram (ECG) recording of ventricular tachycardia (VT). ECG recording on admission: VT with right bundle branch morphology of inferior/septal origin (negative axis in leads II, III + aVF; positive axis in leads I + aVL).

VAs, the most frequent finding in those patients without a previously known cardiac condition was clinically unsuspected myocarditis, as revealed by biopsy.¹² The aforementioned studies relied on invasive myocardial biopsies to screen for underlying causes of arrhythmia, for example myocarditis. However, CMR

has now emerged as a non-invasive reliable tool for diagnosis of acute and chronic myocardial inflammation^{16–18} and, in this study, did reveal its value for detection of myocarditis in patients without previously known cardiac disease. The presence of myocardial inflammation and respective necrosis or scar is not

Figure 3. Cardiac MR (CMR) of scattered post-myocarditis scar. There is scattered scarring throughout the entire left ventricle involving the subepicardial and the midmyocardial layers (arrows). Note, slow flow/gadolinium pooling is present along the right ventricle wall and may mimic pathological contrast enhancement (arrowhead). The subendocardium is spared, a typical finding consistent with post-myocarditis scarring. The most prominent scar was present in the midventricular inferoseptal/inferior segment. Although most likely attributable to viral myocarditis, this form of scarring may also be found in non-infectious myocarditis (*i.e.* cardiac sarcoidosis) and warrants further work-up if the clinical history is inconclusive.

Figure 4. Recording of electrophysiological stimulation. After application of programmed extrastimuli, a pleomorphic-onset ventricular tachycardia followed by ventricular tachycardia originating from the inferoseptal axis was induced.

only known to be associated with high-grade arrhythmias but, in our study, directly matched the origin of arrhythmia as defined by ECG or EPS in nearly half of the patients, directly linking myocarditis-related findings to arrhythmia.

Comparably, a recent CMR study in a mixed (with and without previous cardiac disease) patient population with SCD and sustained monomorphic VT (SMVT) found causative inflammatory changes to be the finding that was most often missed by other non-invasive imaging.¹⁹

Other diagnoses related to arrhythmia in this study were cardiomyopathies. Among them were three cases of DCM, where ischaemia-related changes or inflammation could be excluded by CMR.²⁰ Interestingly, in our patients with DCM, no

regional scarring—known to be associated with an adverse outcome and an increased rate of arrhythmogenic events—was detected by CMR.^{7,8} Future CMR studies are needed to demonstrate whether not only the absence or presence of focal LGE but also the presence of diffuse myocardial fibrosis (T_1 mapping) may identify those DCM patients with more adverse outcomes and increased risk of arrhythmia.²¹

A wide spectrum of other cardiac diseases has been identified in the past to be associated with VAs. Among them, the most common is previous myocardial infarction. Large areas of myocardial scarring constitute an area of conduction block as a prerequisite for re-entry. Accordingly, CMR could demonstrate that patients with a large scar are at increased risk for VA.²² Contrary to previous CMR studies showing a high incidence of

Figure 5. Typical findings detected by cardiac MR (CMR). (a) A patient with recurrent sustained ventricular tachycardia (VT). During electrophysiological stimulation (EPS), two fast VTs with right bundle branch block (RBBB) morphology, inferior axis, were inducible. Matching perimyocarditis scar is predominantly located in the subepicardial myocardial wall, involving the subepicardial fat, extending to the pericardium (arrows). (b) A patient presenting with unclear tachycardia revealed in the electrocardiogram. During EPS, a VT with the same morphology was inducible in the left ventricle (RBBB, superior axis and marked left-axis deviation). A CMR-detected post-myocardial infarction scar in the inferior basal wall. The scar was subendocardial and in part transmural (arrows). Because of the small size of the scar, wall motion was not significantly impaired and relevant thinning of the wall had not occurred. Note, myocardial clefts are occasionally present in the same location and may mimic ischaemic scars. These clefts can easily be differentiated from scars by consulting the corresponding cine images, which demonstrate the corresponding muscular cleft.

Table 3. Presumed morphological substrate of arrhythmia as detected by cardiac MR

Cardiac disease	Main morphological finding and location (if applicable)	Arrhythmogenic correlate (ECG and/or EPS ^a)	MRI is additional information to echocardiography
Dilated cardiomyopathy (3/76, 3.9%)	1–3. Global LV dilation, reduced ejection fraction	1. Sustained polymorphic VT	1. No
		2. Irregular VT with RBBB type pattern	2. No
		3. VT (Holter ECG)	3. No
HCM (1/76, 1.3%)	Septal hypertrophy and structural abnormality	Ventricular fibrillation	Yes
MR criteria for ARVC (2/76, 2.6%)	1. Apical RV hypokinesia	1. Sustained VT with LBBB configuration ^a	1. No
	2. RV dilatation	2. Monomorphic PVB with LBBB configuration	2. Yes
Post-myocarditis (6/76, 7.9%)	Scar location (segments)		
	1. Basal, anterolateral	1. Sustained VT with RBBB configuration, inferior axis ^{a,b}	1. Yes
	2. Basal/midventricular lateral	2. High-frequency pleomorphic VT ^{a,b}	2. Yes
	3. Diffuse, scattered	3. Incessant polymorphic VT, RBBB configuration ^{a,b}	3. Yes
	4. Apical, anterior	4. Ventricular fibrillation	4. Yes
	5. Midventricular, inferior	5. VT with RBBB ^b	5. Yes
	6. Anterior, lateral and inferior	6. Incessant VT (ECG), PVB from LV ^a	6. Yes
Post-myocardial infarction (2/76, 2.6%)	Scar location (segments)		
	1. Basal, inferior	1. Sustained VT with RBBB configuration superior axis ^{a,b}	1. Yes
	2. Midventricular inferoseptal	2. Monomorphic PVB, RBBB configuration ^b	2. Yes
Acute myocarditis (6/76, 7.9%)	Inflammatory changes (segments)		
	1. Anterior and lateral	1. Sustained VT and PVB with RBBB configuration ^{a,b}	1. Yes
	2. Diffuse	2. Ventricular fibrillation	2. Yes
	3. Basal, inferoseptal	3. PVB (bigeminy)	3. Yes
	4. Inferior	4. Ventricular fibrillation	4. Yes
	5. Apical, septal	5. Ventricular fibrillation	5. Yes
	6. Diffuse	6. VT (Holter ECG)	6. Yes

ARVC, arrhythmogenic right ventricular cardiomyopathy; ECG, electrocardiogram; EPS, electrophysiological stimulation; HCM, hypertrophic cardiomyopathy; LBBB, left bundle branch block; LV, left ventricle; PVB, premature ventricular beat; RBBB, right bundle branch block; RV, right ventricle; VT, ventricular tachycardia.

Only the main morphological findings are listed. An electroanatomical correlate (^b) was defined, if the location of a structural abnormality or scar tissue, as defined by late gadolinium enhancement, directly matched the origin of the ventricular arrhythmia as defined by ECG and/or EPS (^a).

unrecognized myocardial infarction in high-risk groups^{2,23} or patients with SCD and SVT,¹⁹ the incidence of myocardial infarction and respective scar in this study was very low. A younger patient population with even gender distribution and systematic exclusion of patients with known cardiac conditions or clinical

presentation of CHD may explain this finding. However, in those patients presenting with typical ischaemic scar, the scar was a direct electroanatomical correlate (*i.e.* the location of the scar matched the origin of the arrhythmia), underscoring the role of ischaemic scar as a trigger of arrhythmia.

Table 4. Clinical relevance of cardiac MR (CMR) findings

Finding	No. of patients (%)
Clinically relevant CMR findings	26/76 (34.2%)
New diagnosis	20 (26.3%)
and/or impact on treatment	21 (27.6%)
Recommendation for/implantation of ICD	9
Change in medication	12
Additional diagnostic procedure	8 (10.5%)
Event recorder	1
Myocardial biopsy	1
Myocardial biopsy + CT	2
CT	2
Hepatic ultrasound	1
Repeated echocardiography	1

ICD, implanted cardioverter defibrillator.

A high incidence of scar of up to 50% in patients with non-ischaemic cardiomyopathy referred for ablation of VT and premature ventricular complexes has previously been reported.^{4,24} Also, it was found that if a critical component of arrhythmia could be identified, it was always confined to the scar tissue. This finding is in good agreement with this study, where the location of scar tissue could directly be linked to the location of VA in the majority of our patients.

Interestingly, the incidence of a morphological substrate or electroanatomical correlate did strongly correlate with the severity of arrhythmia in this study. It was rather low in patients with just frequent PVBs. By contrast, Bogun *et al*⁴ could directly link premature ventricular complexes (without VTs) to scar tissue in almost half of the patients with scar tissue. Thus, the relationship between severity of arrhythmia and the presence of a morphological substrate warrants further investigation in the future.

Clinical relevance

CMR revealed clinically relevant information, missed by the initial diagnostic work-up, including echocardiography, in approximately one-third of the patients with arrhythmia. The relevant information was mostly the detection of an underlying cardiac condition based upon findings of myocardial inflammation or scar as well as other morphological changes, which subsequently led to initiation of an adequate therapy. An underlying cardiac condition was detected in 26% of the patients not known to have a cardiac pathology, which compares well to a recent study performed in SCD and SMVT patients, revealing clinically relevant myocardial disease in 24%.¹⁹ Although single-photon emission CT and positron emission tomography imaging may represent a viable alternative for assessment of myocardial viability and identification of

arrhythmogenic scar, it is of limited use for detection of small myocardial scar or inflammation.^{25–27} In addition, it is associated with ionizing radiation.

Although a diagnosis of inflammation, scar or structural abnormality may also be obtained by histology, myocardial biopsy is invasive, may often result in false-negative findings and, therefore, is not considered part of the routine work-up of arrhythmic patients. Also, the non-invasive detection of, for example, acute myocarditis may obviate the need for further invasive EPS testing and more advanced therapies, such as ICD implantation, since myocarditis is a self-limiting disease in many cases and spontaneous resolution does often occur.²⁸ In addition, the detection of scar and its location by CMR may help with EPS mapping and planning of the ablation strategy, if necessary.⁴ Among the causes for myocarditis, a viral infection (viral myocarditis) is the most frequent. However, other non-infectious forms of myocarditis, for example sarcoidosis, may present with a very similar clinical history and CMR morphology and should be suspected in patients with chronic inflammatory or worsening inflammatory cardiac CMR findings. If a myocardial biopsy is performed, CMR may help in guiding biopsy.^{29,30}

In comparison with the EuroCMR study, the overall impact on patient treatment was lower in this study, while a new diagnosis made by CMR was more frequent. According to the pilot phase of the EuroCMR study³¹ and the multinational follow-up data CMR had a significant impact on patient treatment in >60%, leading to a new diagnosis in approximately 8.7%.⁶ However, this may be explained by the fact that, in the EuroCMR study, patients with known cardiac conditions received a dedicated CMR study for given indications, for example stress CMR for detection of myocardial ischaemia, whereas the patients in this study were screened for cardiac disease.

Limitations

Our study has several limitations. First, the study design is retrospective. Therefore, the number of patients with arrhythmia not studied by CMR because of contraindications, refusal of the study, or because they were not referred cannot be determined. Second, the overall patient number is somewhat small; however, even in this limited number of patients, important findings—undetected by echocardiography—were discovered. And third, our data represent a single-centre experience. Regional variation in the incidence of the detected underlying cardiac conditions, for example ARVC or HCM, may therefore influence the outcome at other centres.

CONCLUSIONS

In summary, this study raises the question as to whether CMR should routinely be performed in those patients presenting with a higher grade VA (Lown ≥ 2). Not only may CMR detect a morphological substrate but also CMR findings may have a direct impact on patient management. In those patients undergoing subsequent EPS, a knowledge of the location and distribution of scar may help with mapping and the isolation of VT foci or the decision to abstain from ablation.

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