Role of cardiac magnetic resonance in heart failure of initially unknown etiology: A 10-year observational study

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ABSTRACT

Background: The heart failure (HF) population is estimated to be 64.3 million people worldwide and continues to grow. Identifying the underlying cause of HF is crucial for patient management and prognosis.

Aims: We sought to evaluate the role of cardiac magnetic resonance (CMR) imaging to identify the etiology of HF and to evaluate the impact of CMR on diagnosis and patient management.

Methods: We retrospectively reviewed the medical charts of 8630 consecutive patients referred for CMR in a large tertiary center between 2008 and 2017 (10 years). In this study, we only included patients referred for CMR due to HF of unknown etiology whose diagnostic workup had not revealed suspicion of any specific cardiac disease leading to HF. We also analyzed changes in patient management that were guided by the CMR findings, which were defined as changes in treatment and/or the necessity of further tests.

Results: The study sample included 243 patients: 173 (71.2%) patients were male, and the mean (SD) age was 44.0 (15.2) years. All patients underwent contrast-enhanced CMR. Late gadolinium enhancement (LGE) was detected in 74.9% of cases. In 94 patients (38.7%), CMR led to a new diagnosis. In 41 patients (16.9%), patient management was changed by CMR. The latter group comprised patients with coronary artery disease, amyloidosis, valvular disease, and cardiomyopathies other than dilated, namely hypertrophic, restrictive, and left ventricular noncompaction.

Conclusions: Our study strongly suggests that CMR imaging is a valuable tool for determining the etiology of HF and affects patient management.

Key words: cardiac magnetic resonance, heart failure, heart failure of unknown etiology, late gadolinium enhancement

INTRODUCTION

Heart failure (HF) is a clinical syndrome caused by structural or functional cardiac abnormalities. It is associated with significant morbidity and mortality [1] and affects 1%–2% of the adult population [2]. In 2020, the HF population was estimated to be 64.3 million people worldwide, and it continues to grow [2]. In Poland, 1.24 million people (3.2% of the population) live with HF [3]. In Europe (particularly in Eastern European countries, including Poland [2, 4]), coronary artery disease (CAD) is the most common cause of both chronic and acute HF.

The identification of the underlying cause of HF is valuable because the outcomes for HF patients vary greatly depending on the etiology [5, 6]. Cardiac magnetic resonance (CMR) imaging is a diagnostic tool used to determine the etiology of HF and is recommended by

WHAT'S NEW?

To our knowledge, this is the first study evaluating the impact of cardiac magnetic resonance (CMR) imaging in the management of patients with heart failure of unknown etiology. We retrospectively reviewed all consecutive patients referred for CMR imaging due to heart failure of unknown etiology in a large tertiary center over a period of 10 years. In a group of 243 patients, we compared pre-CMR diagnosis (diagnosis of exclusion) with post-CMR diagnosis and analyzed its impact on clinical management. In our cohort, CMR led to a new diagnosis in 38.7% of cases and impacted patient management in 16.9% of the cases.

the European Society of Cardiology (ESC) guidelines [1] for patients with a poor acoustic window and for those suspected to have myocardial tissue disease. Because of its unique ability to noninvasively evaluate the myocardium, CMR imaging provides deeper insight into the mechanism leading to cardiac dysfunction and/or dilatation. Consequently, it enables the initiation of the causal treatment of diseases leading to HF. However, studies that demonstrate the true impact of CMR on diagnosis and management in a group of patients with heart failure of unknown etiology are lacking. Thus, we aimed to assess the value of CMR in a cohort of patients in a tertiary reference center.

METHODS

Study design

We retrospectively reviewed all medical charts of 8630 consecutive patients referred for CMR in a large tertiary center between 2008 and 2017, selecting for CMR studies performed due to HF. Then, we analyzed all available medical data to select patients with HF of unknown etiology. Only patients with no specific pre-CMR initial diagnosis were included. Patients whose referring physician suspected myocarditis, cardiomyopathy, a present or a previous myocardial infarction or advanced stable coronary disease (based on clinical signs and symptoms, patient and family history, or all pre-CMR studies) or any specific disease leading to HF were omitted from our analysis. In addition, patients with a family history that indicated possible cardiomyopathy or patients with a clinical presentation suggestive of cardiac amyloidosis, sarcoidosis, or peripartum cardiomyopathy were also excluded. Thus, we included only patients with diagnostic workups that did not reveal suspicion of any specific cardiac disease leading to HF.

All patients with risk factors for CAD had undergone previous invasive coronary angiography and/or computed tomography angiography to exclude CAD as the etiology of HF. Either no obstructive coronary disease was present in those patients or a single vessel disease was found that was not consistent with severe systolic function impairment.

All patients underwent echocardiography, and some patients were diagnosed with valvular disease. CMR imaging was performed in individuals with severe HF symptoms not correlated with the severity of valvular disease as determined by echocardiography.

The study was conducted in accordance with the Declaration of Helsinki. The project was approved by the Bioethics Committee of the National Institute of Cardiology (no. IK-NPIA-0021-16/1686/18). All participants gave written informed consent for the CMR study.

CMR protocol

All CMR studies were performed on a 1.5 Tesla scanner (Avanto or Avanto^{fit}, Siemens, Erlangen, Germany) using breath-hold cine in long-axis planes and sequential short-axis slices.

A gadolinium-based intravenous contrast agent was administrated to all patients, and the presence of late gadolinium enhancement (LGE) was evaluated. The LGE images were acquired 10–15 minutes after injection of the contrast agent. The imaging protocol encompassed all commercially available and clinically indicated sequences required for patients with HF due to an unknown etiology based on current guidelines and recommendations.

Left ventricular end-diastolic and end-systolic volumes were calculated using MASS software (MASS 6.2.1 or later, Medis, Leiden, The Netherlands). Endocardial and epicardial contours were delineated manually in the end-diastolic and end-systolic phases. The anatomy and function of the great vessels and valves were also assessed. The imaging protocol was left to the discretion of the physician supervising the study, as well as a team comprised of a cardiologist and a radiologist. The final CMR imaging-based diagnosis was obtained by a consensus between at least two skilled operators.

The diagnosis based on the CMR results was recorded. Subsequently, we analyzed changes in patient management guided by the CMR results.

Statistical analysis

The Kolmogorov-Smirnov test was used to test whether the data were normally distributed.

Continuous variables are expressed as the mean (standard deviation [SD]) or the median (interquartile range [IQR]). Left ventricular end-systolic (LVESV) and end-diastolic volumes (LVEDV) were normalized to the body surface area. The Student's t-test was performed to analyze continuous and normally distributed variables; otherwise, the Mann-Whitney test was used to test the differences between the groups. The Kruskal-Wallis test was used to make comparisons among the four groups with the most common diagnosis. The post hoc Conover test was applied for statistically significant

Table 1. Patient characteristics

	All patients	Men	Women	*P*-value (men vs. women)
N (%)	243	173 (71.2)	70 (28.8)	
Age, years, mean (SD)	43.9 (15.2)	44.0 (14.3)	46.2 (16.0)	0.29
LVEF, %, mean (SD)	28.3 (11.9)	25.9 (10.8)	33.3 (11.9)	<0.0001
LVEDV, ml/m^2, mean (SD)	162.4 (59.7)	172.9 (59.5)	135.0 (40.8)	<0.0001
LVESV, ml/m^2, mean (SD)	121.1 (59.3)	132.2 (59.1)	93.2 (41.0)	<0.0001
LGE, n (%)	183 (75.3)	131 (75.7)	51 (72.9)	0.86
Subendocardial	35 (14.4)	28 (16.2)	7 (10.0)	
Midwall	153 (63.0)	113 (64.7)	41 (58.6)	
Subepicardial	21 (8.6)	15 (8.7)	6 (8.6)	
Transmural	33 (13.6)	26 (15.0)	7 (10.0)	

Abbreviations: LGE, late gadolinium enhancement; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESV, left ventricular end-systolic volume

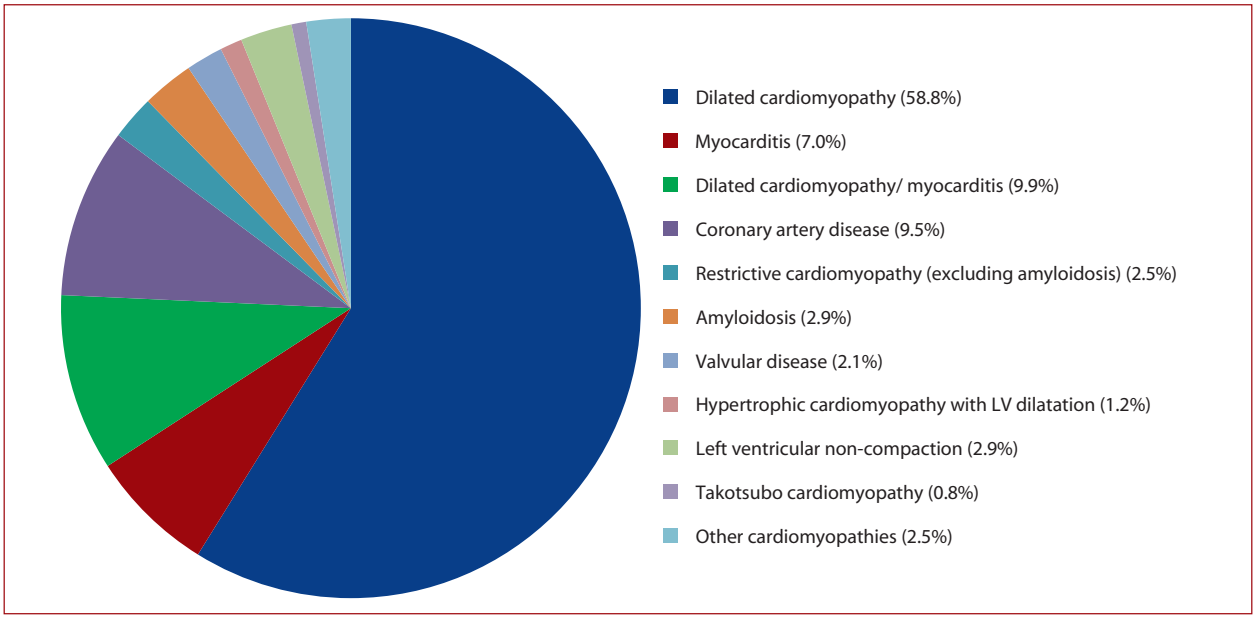

Figure 1. Final diagnoses

Abbreviations: LV, left ventricle

differences. Categorical variables were compared using the χ^2 test. A two-tailed *P*-value of less than 0.05 indicated statistical significance. Statistical analyses were carried out using MedCalc software (MedCalc 12.1.4.0, Ostend, Belgium).

RESULTS

Study cohort

Between January 2008 and December 2017, a total of 246 patients were referred for CMR study because of HF of unknown etiology. Of these, 1 patient was not included in the analyses due to poor-quality CMR images. Two other patients were excluded for the following reasons: 1 patient was excluded due to a significant improvement in cardiac function (normal left ventricular ejection fraction [LVEF] by CMR but poor left ventricular systolic function by the previous echocardiogram), and 1 patient was excluded due to a history of acute decompensated HF secondary to hyperthyroidism. In 5 cases (2.0%), the tests were shortened due to the critical condition of the patients.

The final cohort included 243 individuals, 71.2% (n = 173) of whom were male. The mean (SD) age of the patients was 43.9 (15.2) years. The mean (SD) LVEF was 28.3 (11.9)%. All patients underwent contrast-enhanced CMR. LGE was detected in 75.3% of the patients: 76.3% of men and 72.9% of women. A midwall pattern of LGE was detected in 63.0% of cases. The subendocardial, transmural, and subepicardial patterns of LGE were present in 14.4% and 8.6% of cases, respectively. Table 1 shows the baseline characteristics of the study cohort.

There were statistically significant differences in LVEDV, LVESV, and LVEF between men and women. There was no significant difference in age.

Final diagnoses

The final diagnosis distribution is shown in Figure 1. More than half of the patients (n=143; 58.8%) were diagnosed with dilated cardiomyopathy (DCM) (Figure 2A–C). In 17 patients (7.0%), CMR revealed a characteristic pattern of myocarditis, that met the Lake Louise criteria [7] (Figure 2D–F).

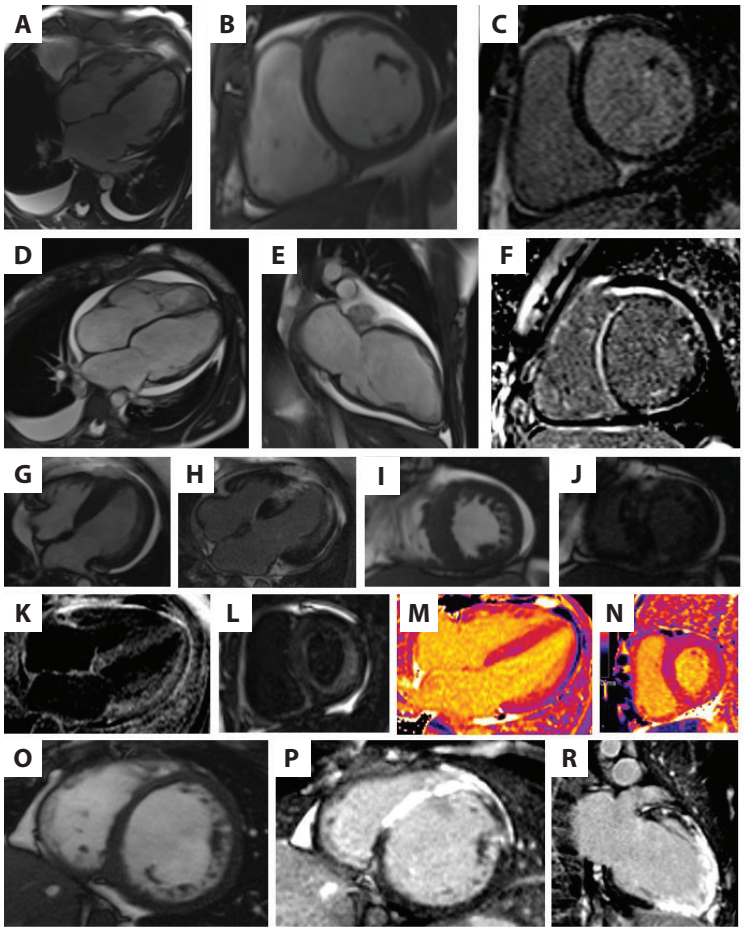

Figure 2. Examples of various new diagnoses and late gadolinium enhancement (LGE) patterns. **A–C.** Dilated cardiomyopathy. Biventricular dilatation (**A, B**) and midwall LGE in the interventricular septum and both right ventricular insertion points (**C**). **D–F.** Myocarditis. Biventricular dilatation (**D, E**) and subepicardial LGE in the interventricular septum, and in the anterolateral and posterior walls (**F**). **G–J.** Hypertrophic cardiomyopathy with left ventricular dilatation. Midwall to transmural LGE in hypertrophied segments (**H, J**). **K–N.** Amyloidosis. Typical global diffuse LGE patt ern (**K, L**). T1 mapping with increased T1 time (**M, N**). **O–R**. Coronary artery disease. Left ventricle dilatation (**O**). Transmural LGE in the septum, anterior wall, posterior wall and apex (**P–R**)

In 24 cases (9.9%), CMR-based diagnosis was unclear, and it was not known if these patients had myocarditis or DCM.

Other cardiomyopathies (qualified under the American Heart Association classification [8]) were less frequent: restrictive cardiomyopathy was found in 13 cases (5.3%), left ventricular noncompaction was found in 7 cases (2.9%), end-stage hypertrophic cardiomyopathy with left ventricular dilatation was found in 3 cases (1.2%) (Figure 2G–J) and stress cardiomyopathy (Takotsubo cardiomyopathy) was found in 2 cases (0.8%). Other cardiomyopathies were diagnosed in 6 cases (2.5%): hypokinetic non-dilated cardiomyopathy (HNDC) [9] was diagnosed in 4 patients, tachyarrhythmic cardiomyopathy was diagnosed in 1 patient, and storage cardiomyopathy was diagnosed in 1 patient.

In 53.8% of the restrictive cardiomyopathy (RCM) cases (7 patients, 2.9%), CMR showed a typical amyloid LGE pattern (Figure 2K–N).

In 23 cases (9.5%), the pattern of subendocardial to transmural enhancement and wall motion abnormalities indicated the presence of a previous undetected infarction (Figure 2O–R).

Five patients (2.1%) were diagnosed with valvular diseases responsible for HF. Three of these patients were found to have aortic valve disease: aortic stenosis in two cases and combined aortic stenosis and insufficiency in one case. One patient was diagnosed with right ventricular HF due to right-sided pathology caused by tricuspid insufficiency. In one case, mitral valve prolapse was previously missed by echocardiography, which led to HF.

Late gadolinium enhancement

There were no statistically significant differences between patients with LGE diagnosed by CMR and patients without LGE on CMR imaging when comparing age (median [IQR], accordingly 45.0 [43.0–56.0] years; 43.0 [34.8–54.3] years; $P = 0.72$) and volumes: LVEF (mean [SD], accordingly 27.4 (11.5)%; 29.8 (11.7)%; $P = 0.52$), LVEDV (median [IQR], accordingly 156.0 [110.0–194.0] ml/m^2; 140.5 [116.0–190.0] ml/m^2; $P = 0.39$), LVESV (median [IQR], accordingly 114.0 [79.9–160.8] ml/m^2; 97.0 [72.0–146.0] ml/m^2; $P = 0.25$).

The presence, type, localization, and extension of LGE were crucial in determining the final diagnosis (as presented in Figure 2).

Heart failure with reduced, mildly reduced, or preserved ejection fraction

Based on the ESC guidelines [1], all patients were stratified by their LVEF as having HF with reduced (HFrEF), mildly reduced (HFmrEF), or preserved (HFpEF) ejection fraction (as shown in Table 2). The heart failure with reduced ejection fraction (HfrEF) group was the largest and included 81.5%

Table 2. Patients subdivided into subgroups with HFrEF/HFmrEF/HFpEF

	HFrEF (n = 198)	HFmrEF (n = 36)	HFpEF (n = 5)	LVEF unknown (n = 4)
Dilated cardiomyopathy	130 (65.7%)	12 (33.3%)	0	1 (25.0%)
Myocarditis	13 (6.7%)	4 (11.1%)	0	0
Dilated cardiomyopathy/ myocarditis	17 (8.7%)	7 (19.4%)	0	0
Restrictive cardiomyopathy (excluding amyloidosis)	2 (1.0%)	2 (5.6%)	1 (20.0%)	1 (25.0%)
Hypertrophic cardiomyopathy with LV dilatation	3 (1.5%)	0	0	0
Left ventricular noncompaction	7 (3.5%)	0	0	0
Takotsubo cardiomyopathy	0	1 (2.8%)	1 (20.0%)	0
Coronary artery disease	16 (8.1%)	5 (13.9%)	0	2 (50.0%)
Valvular disease	3 (1.5%)	1 (2.8%)	1 (20.0%)	0
Other cardiomyopathies	3 (1.5%)	3 (8.3%)	0	0
Amyloidosis	4 (2.0%)	1 (2.8%)	2 (40.0%)	0

Abbreviations: HFmrEF, heart failure with mildly reduced ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; LV, left ventricle; other — see Table 1

Table 3. Most frequent diagnoses

	Dilated cardiomyopathy	Myocarditis	Dilated cardiomyopathy/myocarditis	Coronary artery disease	*P*-value
LVEDV, ml/m^2, median (IQR)	159.0 (127.8–191.8)	151.0 (111.3–245.5)	145.0 (121.5–203.8)	162.0 (121.0–211.0)	0.93
LVESV, ml/m^2, median (IQR)	118.0 (86.8–155.5)	111.0 (79.8–199.0)	85.0 (75.3–175.3)	119.0 (79.8–167.0)	0.84
LVEF, %, median (IQR)	26.9 (18.8–33.1)	25.8 (18.1–40.0)	30.9 (16.8–41.2)	29.5 (20.2–36.2)	0.65
LVSV, ml/m^2, median (IQR)	40.0 (33.0–47.0)	41.0 (29.0–45.8)	44.0 (30.5–49.0)	39.0 (37.8–48.5)	0.80
Age, years, median (IQR)	43.0 (32.3–54.8)	35.0 (22.5–44.8)	35.0 (26.0–50.0)	51.0 (41.0–56.0)	0.005

Abbreviations: LVSV, left ventricular stroke volume; other — see Table 1

(n = 198) of the patients. Thirty-six patients (14.8%) were diagnosed with mildly reduced LVEF.

The heart failure with preserved ejection fraction (HfpEF) group involved 5 patients. Three of these patients were diagnosed with restrictive cardiomyopathy, with 2 cases caused probably by amyloidosis. One patient in the HFpEF group was found to have Takotsubo cardiomyopathy, and the fifth patient had mitral valve prolapse. In these 5 cases, the left ventricular end-diastolic and end-systolic volumes were not calculated due to poor-quality CMR images.

Most frequent diagnoses

The majority of patients were diagnosed with DCM, CAD, myocarditis, or had an ambiguous diagnosis: DCM/myocarditis. We performed statistical analyses among these groups (Table 3), and the volumes and LVEF exhibited no differences (Figure 3). There was a significant age difference, and the myocarditis and DCM/myocarditis groups were younger than the patients with DCM or CAD (Figure 4).

Change in the diagnosis and the impact on patient management

The diagnoses of exclusion in the HFrEF and HF with mid-range ejection fraction (HFmrEF) groups were DCM and HNDC, whereas, in patients with HFpEF, the diagnosis of exclusion was diastolic dysfunction. We compared the final and pre-CMR diagnoses (Figure 5). Changes in the diagnoses were reported in 94 cases (38.7%).

We also analyzed changes in patient management as guided by the CMR results, which were defined as changes in treatment and/or the necessity of further tests. Changes in the pre-CMR diagnosis were judged crucial and led to therapeutic consequences in 41 patients (16.9%) with CAD, amyloidosis, valvular disease, and other cardiomyopathies.

DISCUSSION

CMR is a non-invasive method used to assess cardiac function, volumes, and the great vessels, and provides deep insight into the myocardial structure (in terms of focal or diffuse fibrosis). Cine CMR has become the gold standard for the quantification of ventricular volumes and ejection fractions [10].

Echocardiography is the first-line imaging technique in patients with HF [10], whereas according to the ESC guidelines on acute and chronic HF [1], CMR is recommended for patients with technically limited echocardiographic images, and when there is a suspicion of the presence of disease affecting the cardiac muscle.

According to the American College of Cardiology Foundation (ACCF), American College of Radiology (ACR), American Heart Association (AHA), North American Society for Cardiovascular Imaging (NASCI), and Society for

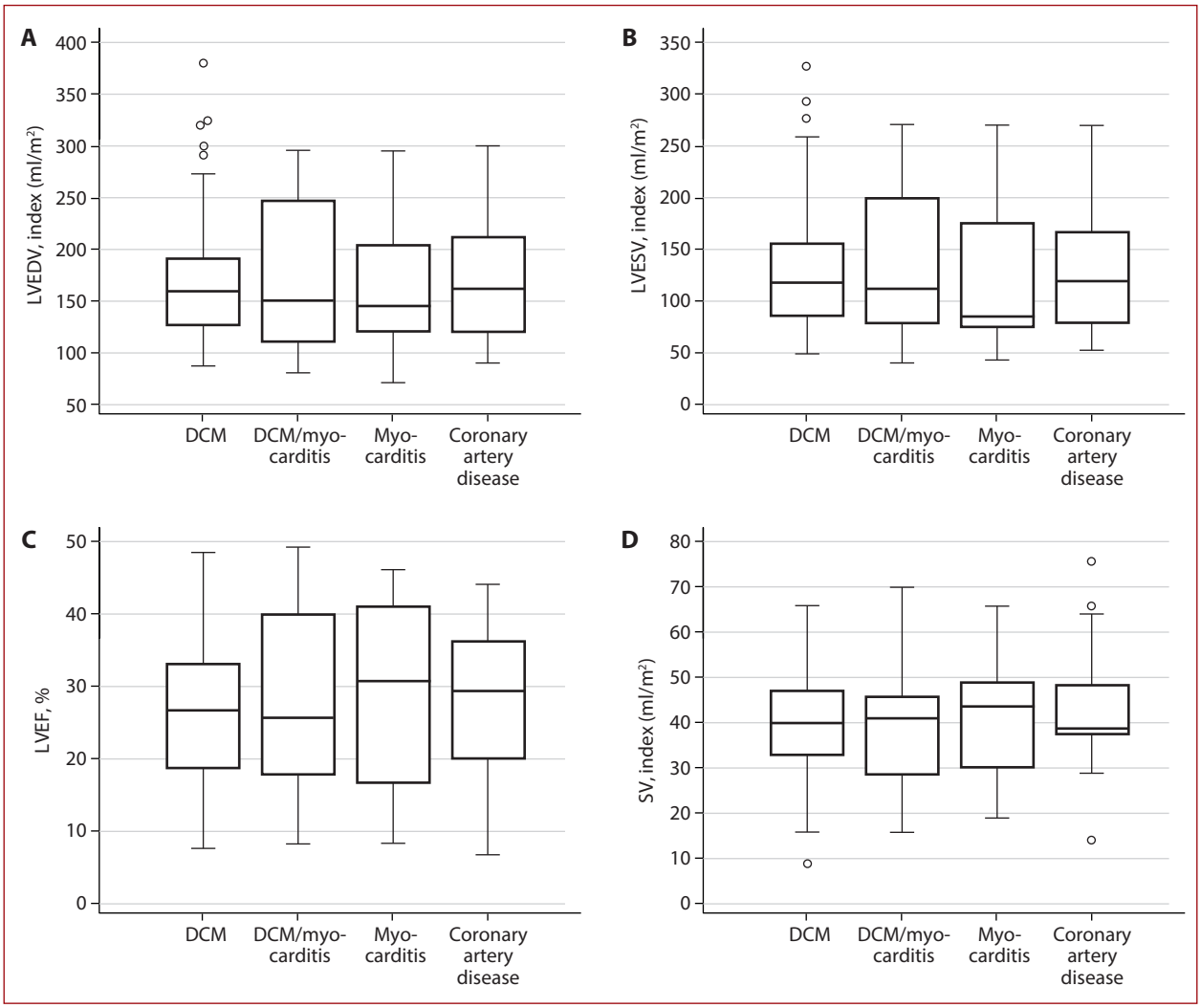

Figure 3. Left ventricular end-diastolic, left ventricular end-systolic volumes, left ventricular ejection fraction, and stroke volume of patients with the most common diagnoses. Box plot with median and IQR, whiskers are the minimum and the maximum, dots represent outliers

Abbreviations: DCM, dilated cardiomyopathy; IQR, interquartile range

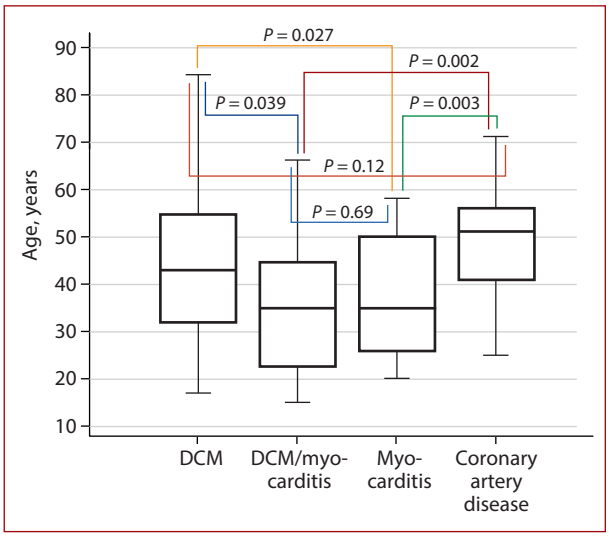

Figure 4. Ages of patients with the most common diagnoses. Box plot with median and IQR whiskers are the minimum and the maximum

Abbreviations: see Figure 3

Cardiovascular Magnetic Resonance (SCMR) consensus document [11], CMR imaging may be used to assess the biventricular size, function, and morphology and to evaluate the myocardium to determine the etiology of HF. CMR allows us to distinguish between ischemic and non-ischemic etiology in patients with HF [11, 12] and can be used to identify the underlying cause of non-ischemic cardiomyopathies [13].

The Heart Failure Association of the ESC recommends CMR imaging in etiological workups in patients with HFpEF [14]. Although echocardiography is the primary imaging technique of choice in the HFpEF group [15], CMR imaging also provides information on diastolic function [16].

Our study aimed to evaluate the role of CMR in identifying the underlying cause of HF due to an initially unknown etiology as well as its impact on patient management.

The final diagnosis was different from the pre-CMR diagnosis in 38.7% of patients and led to serious therapeutic consequences in 16.9% of cases. Consequently, the findings from the CMR study also impacted patient prognosis.

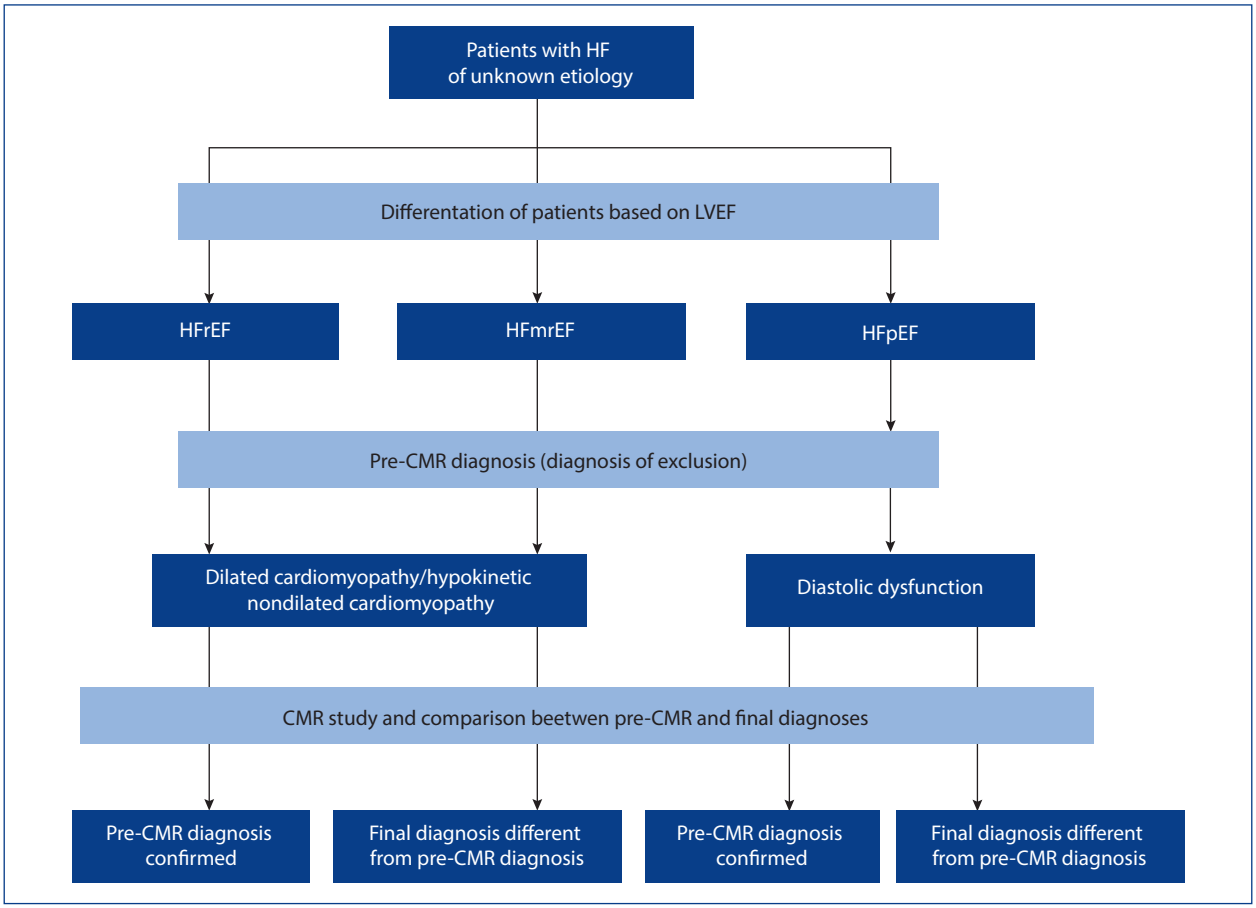

Figure 5. Definition of a change in the pre-CMR diagnosis

Abbreviations: CMR, cardiac magnetic resonance; HF, heart failure; other — Figure 1 and Table 2

In the EuroCMR registry, in more than 27 000 consecutive patients, CMR-based diagnosis differed from the pre-CMR diagnosis in 8.7% of patients and impacted patient management in 61.8% of cases [17]. Abassi et al. [18] reviewed CMR studies of 150 consecutive patients with LVEF ≤50% and studied the clinical impact of CMR defined as a new diagnosis or change in management. In their study, CMR impacted 65% of patients, led to a new diagnosis in 30% of cases, and impacted management in 52% of patients. Kangala et el. evaluated the impact of CMR on the diagnosis and prognosis in a group of patients with HFpEF [19]. In 27% of patients, CMR led to the identification of a previously undetected pathology that correlated with a worse prognosis.

Lin et al. [20] reported that non-ischemic cardiomyopathy was significantly less likely to be misdiagnosed in patients who underwent CMR before cardiac transplantation than in patients who did not undergo CMR.

To our knowledge, this is the first study to evaluate the impact of CMR in patients with an initially unknown etiology. Our study strongly confirmed the notion that CMR imaging is a valuable tool for determining the etiology of HF. Having studied a large real-life population, we provided evidence that CMR should be considered in all patients with unexpected and newly diagnosed HF, particularly if other tests cannot determine the etiology.

Study limitation

First, this study was conducted as a retrospective study. Patients were referred for CMR study by primary-care physicians. We analyzed only the data that was available in the medical records. Second, the patient cohort consisted only of Caucasians, and 99.2% of the patients were of Polish origin. Third, we have no follow-up data. Moreover, the HFpEF group included only 5 patients (2.1%), while the HFpEF group was estimated to include approximately 50% of all HF patients [16].

CONCLUSIONS

Our study highlights the usefulness of magnetic resonance imaging in determining the etiology of HF in patients in whom a diagnostic workup did not reveal the cause of impaired cardiac function. The study also suggests that CMR imaging significantly affects patient management.

Article information

Conflict of interest: None declared.

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