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ORIGINAL RESEARCH ARTICLE

Characteristics and prognostic value of right ventricular (dys)function in patients with non-ischaemic dilated cardiomyopathy assessed with cardiac magnetic resonance imaging

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Abstract

Aims In non-ischaemic dilated cardiomyopathy (DCM), concomitant right ventricular (RV) dysfunction is frequently observed. This study sought to determine the correlation of RV dysfunction with several cardiac magnetic resonance (CMR) imaging characteristics in patients with DCM, and the prognostic value of RV dysfunction on all-cause mortality and ventricular arrhythmias (VA) was evaluated.

Methods and results Consecutive patients with DCM and left ventricular (LV) dysfunction (ejection fraction $<$ 50%) on CMR were included retrospectively. Left atrial (LA), LV, and RV volumes and function were quantified. RV systolic dysfunction was defined as RVEF$<$45%. The presence and pattern of late gadolinium enhancement (LGE) on CMR were assessed visually. Septal midwall LGE was defined as midmyocardial stripe-like or patchy hyperenhancement in the septal segments, and the extent was quantified using the full width at half maximum method. Primary endpoint was a composite of all-cause mortality and VA, including resuscitated cardiac arrest, sustained VA, and appropriate implantable cardioverter defibrillator therapy. Secondary endpoints were time to all-cause mortality alone and time to VA alone. A total of 216 DCM patients were included (42% female, age 58 ± 14 years). Mean RVEF was 46 ± 12%, and RV dysfunction was present in 38%. RVEF was moderately correlated with LA dilation (LA minimal volume $\rho = -0.38$, $P < 0.001$) and strongly correlated with LA and LV dysfunction (LA emptying fraction $r = 0.58$, $P < 0.001$ and LVEF $\rho = 0.52$, $P < 0.001$). Septal midwall LGE was more often observed in patients with RV dysfunction compared with patients with preserved RV function (respectively 40% vs. 26%, $P = 0.04$). No correlation was found between RVEF and the extent of septal midwall LGE ($\rho = -0.12$, $P = 0.34$). During a median follow-up of 2.2 years [IQR 1.6–2.8], 30 patients experienced the primary endpoint. RV dysfunction was significantly associated with shorter time to the composite primary endpoint (HR 3.19 [95% CI 1.49–6.84], $P < 0.01$) and to the secondary endpoint of VA alone (HR 6.48 [95% CI 1.83–22.98], $P < 0.01$). There was a trend towards increased mortality when RV dysfunction was present (HR 2.54 [95% CI 0.99–6.57], $P = 0.05$).

Conclusions Right ventricular dysfunction was predominantly observed in patients with DCM with advanced heart failure and pronounced myocardial remodelling, defined as increased LV and LA dilation and dysfunction and the presence of septal midwall LGE on CMR. During follow-up, RV dysfunction was associated with shorter time to all-cause mortality and ventricular arrhythmic events.

Keywords Non-ischaemic dilated cardiomyopathy; Cardiac magnetic resonance imaging; Late gadolinium enhancement; Survival; Arrhythmias

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Introduction

In patients with non-ischaemic dilated cardiomyopathy (DCM), diagnosis and prognosis are based on left ventricular (LV) dimension and function according to current guidelines,[1] and treatment is focused on LV function. However, a substantial number of patients with DCM demonstrate concomitant right ventricular (RV) dysfunction.[2–4] The underlying mechanisms causing RV function deterioration in DCM remain unclear. Probable causes of RV dysfunction include primary RV involvement in a biventricular cardiomyopathy causing intrinsic contractile dysfunction or secondary RV involvement resulting from alterations in RV loading conditions due to LV impairment.[4–6] A recent study demonstrated that severe RV dysfunction was most frequently seen in patients with left sided heart disease, up to 46%.[7] Echocardiographical RV analysis remains challenging due to the complex asymmetrical anatomy and extensive trabeculated myocardium.[8] Cardiac magnetic resonance (CMR) imaging is considered the gold standard for RV analysis and quantification providing high accuracy and reproducibility.[9,10] In addition, data on prognostic impact of RV dysfunction in patients with DCM for mortality and development of sustained ventricular arrhythmias (VA) is sparse.[2–4,11,12] Currently, indication for primary prevention implantable cardioverter-defibrillator (ICD) for the prevention of sudden cardiac death is based on the severity of LV dysfunction and the presence of symptomatic heart failure.[1,13] However, previous studies demonstrated no beneficial effect of primary prevention ICD in patients with DCM when indicated according to the current guidelines.[14] Therefore, enhanced risk stratification beyond left ventricular ejection fraction (LVEF) assessment is necessary to identify patients with DCM at risk of sudden cardiac death. The objective of this study was to describe determinants of RV dysfunction in patients with DCM using late gadolinium enhancement (LGE) CMR and assess the prognostic value of RV dysfunction on the occurrence of mortality and VA.

Methods

This registry based single-centre observational cohort study retrospectively included consecutive patients with DCM and impaired LV function, defined as LVEF $<$50% on LGE-CMR performed between January 2016 to May 2018. Data regarding baseline characteristics and outcome during follow-up were obtained from electronic medical records, and survival status was independently assessed using the National Health and Social Care Information System. DCM was classified as LV dysfunction in the absence of obstructive coronary artery disease on imaging, severe arterial hypertension, or primary valvular heart disease, as per guidelines.[1] A history of myocardial infarction including previous coronary artery revascularization or ischaemic LGE pattern on CMR of sufficient severity to explain the degree of LV dysfunction was considered ischaemic heart disease and was excluded. Other exclusion criteria included pre-existing RV diseases, including arrhythmogenic RV cardiomyopathy, congenital heart disease with RV pressure or volume overload, and pulmonary hypertension. Furthermore, acute myocarditis, infiltrative heart disease, and hypertrophic cardiomyopathy were excluded. Scans without gadolinium administration with severe artefacts or incomplete scan protocols hampering CMR quantification were excluded.

Cardiac magnetic resonance imaging protocol

Indications for LGE-CMR included diagnosis of underlying aetiology in new onset heart failure, follow-up in known cardiomyopathy, or LV assessment prior to device implantation. All CMR acquisitions were performed on 1.5T MRI scanners (Siemens, Erlangen, Germany, and GE Healthcare, Chicago, IL, USA) with a dedicated phased array cardiac receiver coil. Myocardial function and anatomy were assessed using retrospective ECG-gated steady-state free precession cine imaging with breath-holding in standard three long-axis views and a stack short-axis slices, covering the ventricles from base to apex. Contrast images were acquired approximately 10–15 min after intravenous gadolinium administration using a T1-weighted inversion recovery-prepared gradient echo sequence with optimized inversion time.

Cardiac magnetic resonance analysis

All CMR analyses were performed using dedicated software packages from CVI[42] (Circle Cardiovascular Imaging Inc., Version 5.6, Calgary, Canada). Both LV and RV volumes were quantified by semi-automatic delineation of the endocardial border at end-diastole and end-systole from short-axis image stacks with manual correction in every patient. LV mass was quantified by additionally delineating LV epicardial contours. Papillary muscles and trabeculations were included in the blood pool. RV dysfunction was defined as RVEF $<$45%.[15] All measurements were indexed to body surface area. For assessment of reproducibility of RVEF quantification, 20 randomly selected CMRs were independently analysed by two observers (M. B. and M. W.). Left atrial (LA) volumes and areas were calculated using the area-length method, delineating the atrial border in the two-chamber and four-chamber long-axis view at multiple phases throughout the cardiac cycle.

The presence and pattern of LGE were assessed visually. LGE was considered present if hyperenhancement was

observed in two orthogonal views or in two serial slices. Septal midwall LGE was defined as midmyocardial stripe-like or patchy hyperenhancement in the interventricular septal segments including the RV insertion points. Quantification of septal midwall LGE extent was performed using the full width at half maximum method. Any LGE in segments other than the interventricular septum was not included in the quantification.

Follow-up

Primary endpoint was a composite of all-cause mortality, including heart transplantation, and VA including resuscitated cardiac arrest, sustained ventricular tachycardia, or ventricular fibrillation and appropriate ICD therapy. Appropriate ICD therapy was defined as antitachycardia pacing or shock for the occurrence of VA. Secondary endpoints were (i) all-cause mortality and (ii) VA. Follow-up data were obtained from medical records and patients were followed until October 2019. The investigation conforms with the principles outlined in the Declaration of Helsinki. The study protocol was approved by the local institutional ethics committee, and patients provided written informed consent for CMR analysis and data acquisition.

Statistical analysis

Continuous variables are presented as mean ± SD or as median [interquartile range], depending on whether they were normally distributed or not. Categorical data are summarized as frequencies and percentages. Between-group differences for continuous data were assessed using the independent samples *t*-test for normally distributed variables or otherwise Mann–Whitney *U*-test; χ^2 test was used for dichotomous data, while ordinal data were compared between groups using the Mann–Whitney *U*-test. A two-way random interclass correlation coefficient was assessed for interrater reproducibility of RV function. Pearson correlation or Spearman correlation was calculated to quantify the strength of association between continuous and ordinal variables. Time to primary and secondary endpoints was depicted graphically using Kaplan–Meier curves with between-group differences assessed by the log-rank test. Patients in which endpoint was not observed were censored at the end of follow-up. To test whether variables were associated with time to composite primary endpoint at follow-up, univariable Cox regression analyses were performed. Multivariable Cox regression analysis was performed using a backward selection model including LVEF and RVEF and clinical variables previously associated with worse outcome including age, sex, and New York Heart Association (NYHA) functional class. Collinearity was assessed using variance inflation factors. A two-sided significance level of 5% was used for all analyses. Statistical analyses were performed using SPSS (version 26, IBM SPSS Statistics, Chicago, IL, USA).

Results

A total of 216 consecutive patients with DCM were included. The majority was male (58%); mean age was 58 ± 14 years. *Table 1* depicts patient characteristics, stratified by the presence or absence of RV dysfunction. Median LVEF was 37% [25–44], and septal midwall LGE was present in 68 patients (32%) with a median extent of 7.7 g [5.0–13.1]. Mean RVEF was 46 ± 12% and RV dysfunction was found in 83 patients (38%). An interclass correlation coefficient of 0.88 (95% CI 0. 72–0.95) was found for the reproducibility of RVEF. Patients with RV dysfunction had higher NYHA functional class ($P < 0.001$) and consequently used more heart failure medication (*Table 1*). In patients with RV dysfunction, LV volumes were larger (indexed LV end diastolic volume (LVEDVi) 117 mL/m^2 vs. 105 mL/m^2, respectively, $P = 0.05$), LVEF was lower (23% [17–35] vs. 41% [34–46], $P < 0.001$), and septal midwall LGE was seen more often (40% vs. 26%, $P = 0.04$) compared with patients with a preserved RV function. A LVEF <35% was present in the majority of patients with RV dysfunction (73%). Mitral regurgitation was observed in 136 patients (63%), of whom 113 (83%) had only mild regurgitation secondary to annulus dilation. It was seen significantly more often in patients with RV dysfunction (75% vs. 56%, $P < 0.01$). Furthermore, LA volumes were enlarged in patients with RV dysfunction, and LA emptying fraction was lower (*Table 1*). RVEF demonstrated a moderate–good positive correlation to LVEF (Spearman $\rho = 0.52$, $P < 0.001$) and to LA emptying fraction (Pearson $r = 0.58$, $P < 0.001$). A weak–moderate negative correlation was found between RVEF and LA volumes (LAVi $r = -0.29$, $P < 0.001$ and LA minimal volume $\rho = -0.38$, $P < 0.001$). No correlation was found between RVEF and LGE extent ($\rho = -0.12$, $P = 0.34$) (*Figure 1*).

Forty-eight patients were lost to follow-up. During a median follow-up of 2.2 [1.6–2.8] years, 30 (18%) of the remaining 168 patients experienced the primary endpoint (i.e. 15 deaths and 15 VAs). VA included appropriate ICD therapy in 10 patients (three shocks and seven antitachycardia pacing (ATP) only), 3 survivors of sudden cardiac arrest due to VA, and 2 patients with sustained ventricular tachycardia. Three patients who experienced VA died later during follow-up. The survival curves of the primary and secondary endpoints stratified by RV dysfunction are demonstrated in *Figure 2* and *3*, respectively. Patients with RV dysfunction had a significantly shorter time to the primary composite endpoint (HR 3.19 [95% CI 1.49–6.48], $P < 0.01$, *Figure 2*) and to VA alone (HR 6.48 [95% CI 1.83–22.98], $P < 0.01$, *Figure 3A*).

Table 1 Patient characteristics

	Total *N* = 216	Preserved RV function *N* = 133 (62%)	RV dysfunction *N* = 83 (38%)	*P*-value
Male sex	126 (58%)	74 (56%)	52 (63%)	0.31
Age (years)	58 ± 14	58 ± 13	58 ± 15	0.83
BMI (kg/m^2)	26.3 ± 4.9	26.2 ± 5.0	26.3 ± 4.9	0.86
NYHA functional class	*N = 167*	*N = 102*	*N = 65*	<0.001[a]
Class I	74 (34%)	56 (55%)	18 (28%)	
Class II	56 (26%)	35 (34%)	21 (32%)	
Class III-IV	37 (17%)	11 (11%)	26 (40%)	
Unknown	49 (23%)			
CMR				
LVEDVi (mL/m^2)	108 [90–132]	105 [91–121]	117 [83–154]	0.05
LVESVi (mL/m^2)	66 [51–97]	61 [50–76]	87 [53–128]	<0.001
LVEF (%)	37 [25–44]	41 [34–46]	23 [17–35]	<0.001
LV mass (g/m^2)	75 ± 21	74 ± 17	78 ± 26	0.13
RVEDVi (mL/m^2)	81 ± 22	78 ± 19	86 ± 26	0.01
RVESVi (mL/m^2)	44 ± 20	36 ± 12	58 ± 23	<0.001
RVEF (%)	46 ± 12	54 ± 7	34 ± 9	<0.001
LA min volume (mL)	42 [28–68]	37 [26–53]	73 [34–109]	<0.001
LAVi (mL/m^2)	50 ± 19	46 ± 14	57 ± 23	<0.001
LA min area (cm^2)	17 [13–23]	15 [12–19]	22 [14–29]	<0.001
LA max area (cm^2)	28 ± 7	27 ± 6	30 ± 9	<0.01
LA emptying fraction (%)	48 ± 18	56 ± 12	36 ± 18	<0.001
Any LGE present	82 (38%)	42 (32%)	40 (48%)	0.01
Septal midwall LGE	68 (32%)	35 (26%)	33 (40%)	0.04
Extent septal midwall LGE (g)	7.7 [5.0–13.1]	7.8 [5.3–12.4]	7.2 [4.3–16.8]	0.98
Fraction septal midwall LGE (%)	6.0 [3.6–9.6]	6.1 [4.5–8.2]	5.3 [3.5–9.7]	0.70
Medical history				
Smoking (current/former)	51 (24%)	30 (26%)	21 (28%)	0.76
Hypertension	69 (32%)	46 (40%)	23 (31%)	0.21
Diabetes mellitus	24 (11%)	16 (14%)	8 (11%)	0.52
Chronic renal dysfunction	26 (12%)	11 (9%)	15 (20%)	0.02
Atrial fibrillation	43 (20%)	16 (12%)	27 (33%)	<0.001
Paroxysmal	21 (49%)	12 (75%)	9 (33%)	0.02
Persistent	22 (51%)	4 (25%)	18 (67%)	
Medication				
β-blocker	130 (64%)	70 (55%)	60 (80%)	<0.001
ACEi/ARB	144 (71%)	92 (72%)	52 (69%)	0.70
MRA	65 (32%)	29 (23%)	36 (48%)	<0.001
Diuretics	72 (36%)	30 (23%)	42 (56%)	<0.001
ICD	51 (24%)	21 (16%)	30 (36%)	0.001
CRT-D	22 (43%)	11 (52%)	11 (37%)	0.27
Primary prevention SCD	34 (67%)	11 (52%)	23 (77%)	0.07

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin-II receptor blocker; BMI, body mass index; CRT-D, cardiac resynchronization therapy ICD; ICD, implantable cardioverter defibrillator; LA, left atrium; LAVi, indexed left atrial volume; LGE, late gadolinium enhancement; LVEDVi, indexed left ventricular end diastolic volume; LVEF, left ventricular ejection fraction; LVESVi, indexed left ventricular end systolic volume; MRA, mineralocorticoid receptor antagonist; NYHA, New York Heart Association functional class; RV, right ventricle; RVEDVi, indexed right ventricular end diastolic volume; RVEF, right ventricular ejection fraction; RVESVi, indexed right ventricular end systolic volume; chronic renal dysfunction defined as eGFR <60 mL/min/1.73 m^2; SCD, sudden cardiac death.
[a]Patients with missing data were excluded from analysis.

There was a trend towards a difference in time to all-cause mortality between the groups (HR 2.54 [95% CI 0.99–6.57], *P* = 0.05, *Figure 3B*). *Figure 4* depicts the univariable Cox regression analysis for the primary composite endpoint. Multiple CMR characteristics were significantly associated with the composite endpoint, including LVEF (HR per 10% increase 0.75, *P* = 0.05), RV dysfunction (HR 3.19, *P* < 0.01), LA emptying fraction (HR 0.97, *P* < 0.01), and septal midwall LGE presence (HR 2.11, *P* = 0.04). Multivariable Cox regression demonstrated RVEF as significant predictor of the combined endpoint (HR per 10% increase 0.81, *P* = 0.02), independent of LVEF, NYHA functional class, sex, and age (*Table 2*).

Discussion

This study demonstrates that concomitant RV dysfunction in patients with impaired LV function due to DCM was present on CMR in 38% of patients. RV dysfunction was associated with increased NYHA class, larger LV volumes, and worse LV function, with decreased LA emptying fraction and more often septal midwall LGE present. In addition, during follow-up, the event-free survival was significantly lower in patients with RV dysfunction, for the primary composite endpoint and for the ventricular arrhythmic endpoint alone. There was a

Figure 1 Correlates of RVEF. RV dysfunction was primarily seen in patients with poor LV function (A). While there was a moderate–good positive correlation between RVEF and LA emptying fraction (B), there was no correlation between RVEF and septal midwall LGE extent (C). LA, left atrial; LGE, late gadolinium enhancement; LVEF, left ventricular ejection fraction; RVEF, right ventricular ejection fraction.

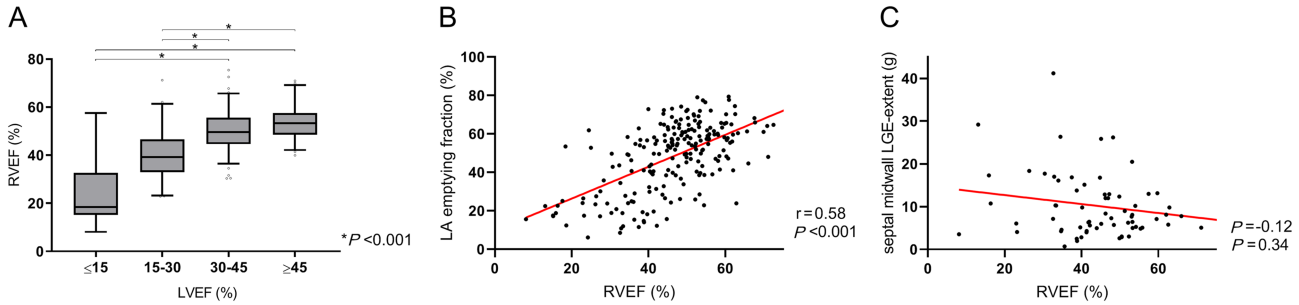
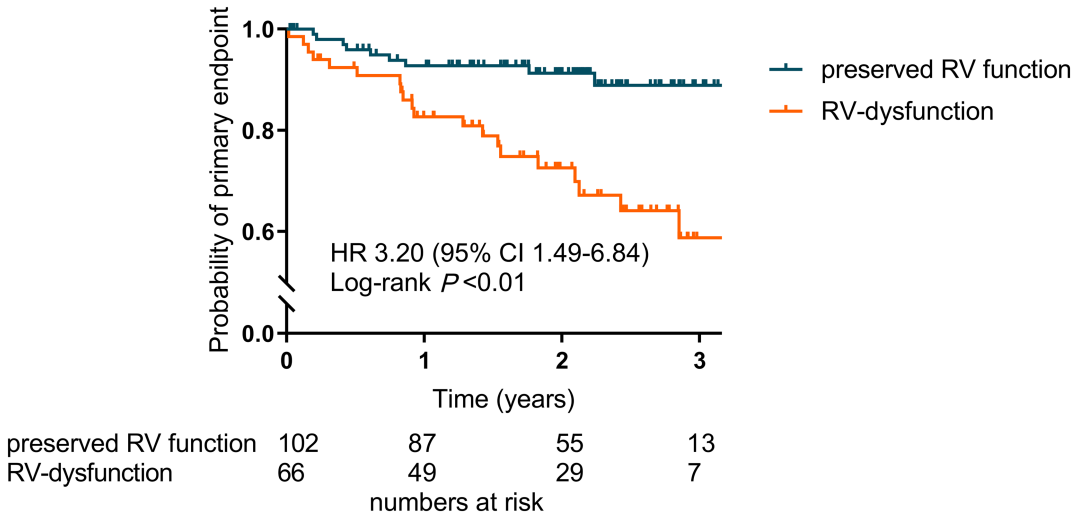


Figure 2 Survival curves of the composite primary endpoint. Kaplan–Meier survival curves stratified by RV dysfunction of the combined primary endpoint including all-cause mortality and VAs, demonstrating a significantly shorter time-to-event in patients with RV dysfunction compared with patients with preserved RV function. CI, confidence interval; HR, hazard ratio; RV, right ventricular.

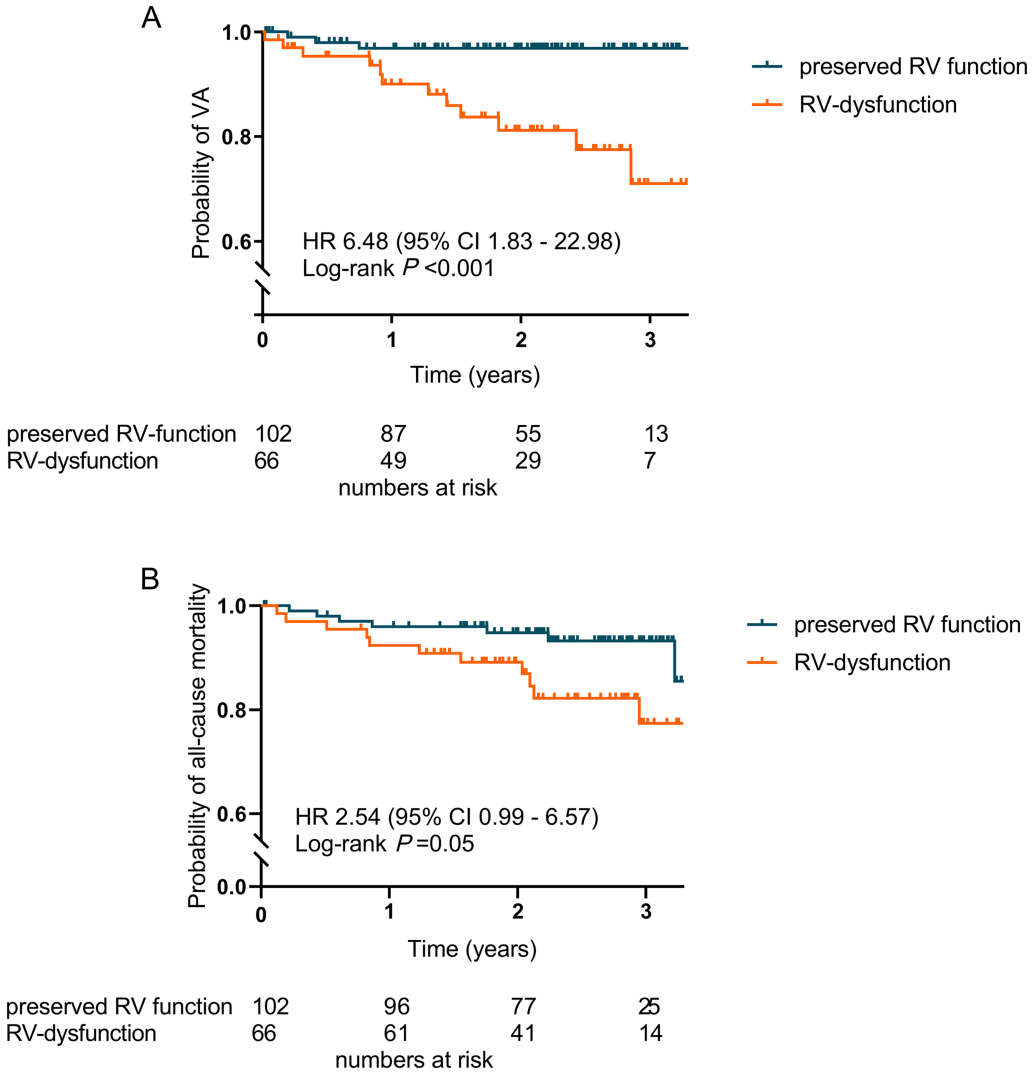


trend towards higher mortality rate in patients with RV dysfunction.

The presence of RV dysfunction in DCM was previously demonstrated with an incidence varying between 30%[3] up to 70%.[16] Although the RV differs anatomically and functionally from the LV, they are closely related by interventricular interdependence, resulting in LV function as an important contributor of RV systolic function.[6] This close relation is reflected by the positive correlation between LV and RV functions found in the present study, in line with previous publications.[4,12,17] Furthermore, RV dysfunction was primarily seen in patients with LVEF <35%, which is the current cut-off value for ICD implantation for primary prevention of sudden cardiac death.[1] Other characteristics in patients with RV dysfunction in this study were larger LA volume and lower LA emptying fraction. Increased LA volumes and in particular decreased LA emptying fraction are established markers for LV diastolic dysfunction[18,19] and are known predictors of adverse events and mortality.[20,21] Prolonged increase in LV afterload results in increased LA pressure, which causes remodelling with subsequent LA dilation and dysfunction.[18,20,22] The resultant increase in RV afterload initiates RV dysfunction.[4–6] Over time, RV dysfunction may recover in some patients with DCM,[11] suggesting that treatment of congestive heart failure and reduction of LV filling pressures, subesequently reduces RV afterload and improves RV function. These findings suggest that RV dysfunction is secondary to left-sided heart failure.

The association of RV dysfunction with lower LVEF, larger LV volumes, and a higher incidence of septal midwall LGE on CMR suggests an advanced stage of myocardial remodelling, including deposition of interstitial fibrosis.[23] This suggest that RV dysfunction is a sign of advanced disease, which would explain the worse symptomatic heart failure,

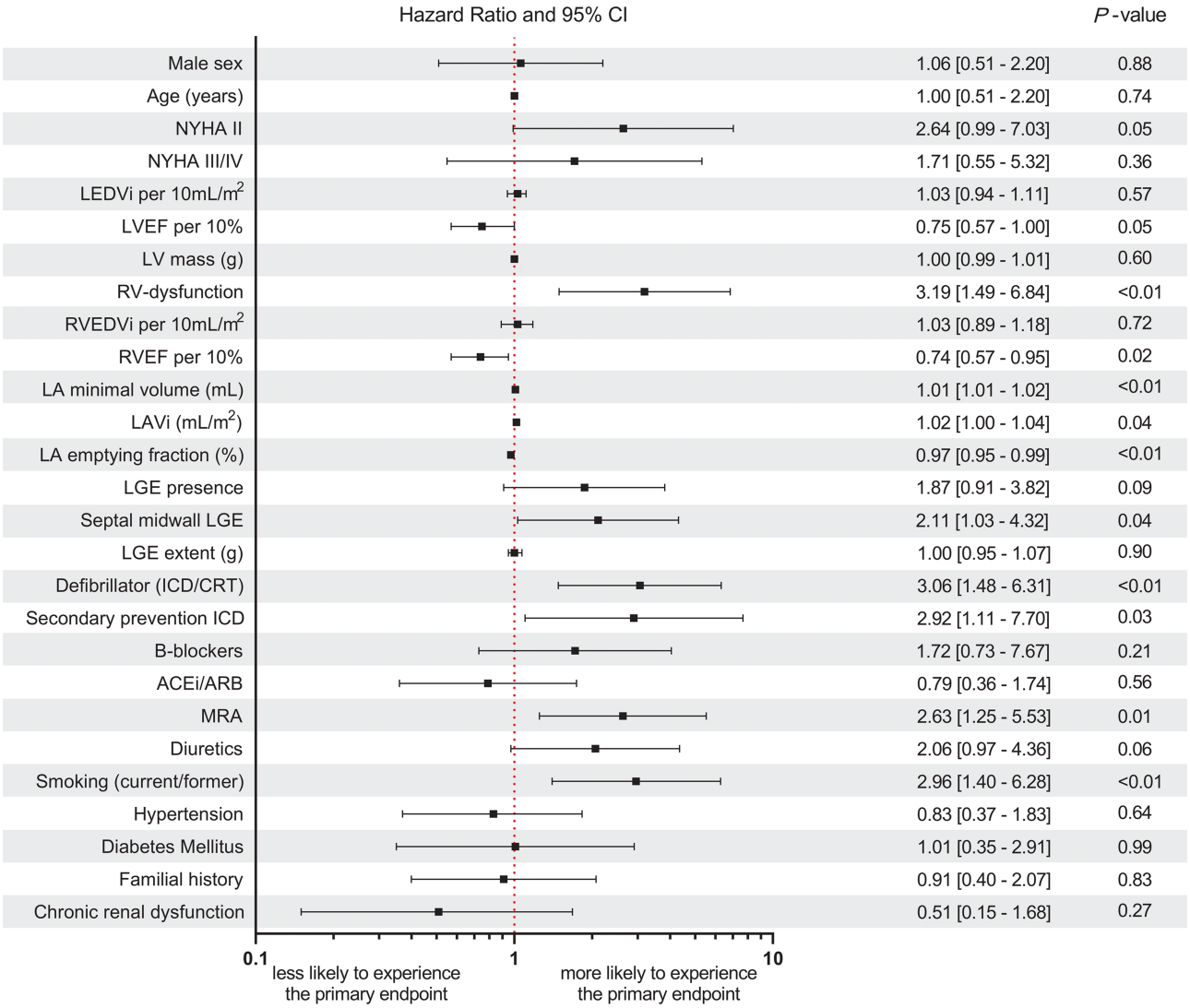
Figure 3 Survival curves of the secondary endpoints. Kaplan–Meier curves stratified by RV dysfunction of the secondary endpoint VAs (A) with a significantly shorter time-to-event in the presence of RV dysfunction. For the secondary endpoint of all-cause mortality (B), there was a trend towards shorter time-to-event with RV dysfunction present. RV, right ventricular.



expressed as higher NYHA class. With RV dysfunction as a sign of advanced disease, worse prognosis seems legitimate. The unfavourable prognostic value of the presence of RV dysfunction on survival and the occurrence of VA in the present study is in line with earlier work.[3,4,24] In addition, RV dysfunction was previously found to significantly add to the prediction of adverse events on top off the currently used risk factor LVEF.[12] Patients with both poor LVEF and poor RVEF had a twofold increase in mortality, compared with patients with poor LVEF alone and preserved RVEF.[12]

In the present study, the prognostic value of RV dysfunction was mainly associated with shorter time to VA. However, the mechanism of the substrate for VA in DCM remains debatable. During myocardial remodelling in LV dysfunction, initially an adaptive mechanism including myocyte hypertrophy and increased collagen synthesis, accumulation of interstitial fibrosis may result in irreversible myocardial scarring.[25] This scarring may function as substrate for VA,[26] because in patients with DCM, the majority of VA are scar-related re-entry tachyarrhythmia.[27] On the other hand, as a result of myocardial remodelling and LV dilation, wall stress increases. Higher wall stress is known to have direct proarrhythmic effects on a cellular level, which increases susceptibility of VA.[28,29] Moreover, in a previous report, impaired LA emptying fraction was thought to reflect increased LV wall stress.[30] The association between LA emptying fraction and the primary outcome found in the present study is in line with a previous ICD cohort study, in which impaired LA emptying fraction was a strong independent predictor of appropriate ICD therapy for VA.[31]

Figure 4 Variables associated with the composited primary endpoint. Forest plot visualizing the hazard ratios and 95% CI of variables included at univariable Cox regression analysis for the association with the primary endpoint. The presented *P*-values were not corrected for multiple testing. ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin-II receptor blocker; CI, confidence interval; CRT, cardiac resynchronization therapy; HR, hazard ratio; ICD, implantable cardioverter defibrillator; LA, left atrial; LAVi; indexed maximum left atrial volume; LGE, late gadolinium enhancement; LV, left ventricular; LVEDVi, indexed left ventricular end diastolic volume; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; NYHA, New York Heart Association functional class; RV, right ventricular; RVEDVi, indexed right ventricular end diastolic volume; RVEF, right ventricular ejection fraction.



Several previous studies have demonstrated the prognostic value of RV dysfunction in DCM, with relatively small cohorts and often heterogeneous patient populations. RV function was often assessed using echocardiography, which is of limited value in the anatomical complex RV, and different cut-offs and definitions for RV dysfunction were used. The additional value of the present study includes the use of CMR, currently considered the gold standard for non-invasive assessment of ventricular function. In addition, the assessment of atrial volume and function was found to be more accurate using CMR compared with echocardiography.[32] Furthermore, because all CMRs included LGE imaging, we were able to assess the presence and extent of septal midwall LGE, another established predictor of mortality and VA in DCM. This enabled us to compare and correlate the various identified predictors in the same patient cohort. This stretches the prognostic value of routine CMR performance in the risk assessment, in particular for ventricular events and appropriate ICD-therapy.

Table 2 Multivariable Cox regression analysis

Model I	HR [95% CI]	*P*-value
LVEF per 10%	…	ns
RVEF per 10%	0.81 [0.68–0.97]	0.02
NYHA functional class	…	ns
Age	1.02 [1.00–1.04]	0.05
Male sex	…	ns

"…" indicates a parameter that was included as candidate predictor in multivariable analysis but removed in backward selection procedure and therefore does not appear in the final model (i.e. nonsignificant).
CI, confidence interval; HR, hazard ratio; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association functional class; RVEF, right ventricular ejection fraction.

Limitations

We must acknowledge several limitations of this study. This was a single centre, retrospective, observational study, a design associated with potential selection bias. Furthermore, the retrospective nature of this study precluded a structured patient follow-up, and available data were extracted from electronic medical records. Serial assessment of LV and RV function determining their course over time was not possible due to the retrospective study design. During follow-up, there was a considerable amount of patients lost, decreasing the sample size. In addition, the rather large number of variables assessed in univariable Cox regression may potentially result in false-positive outcomes. Moreover, various CMR variables associated with the primary endpoint were correlated limiting multivariable Cox regression analysis with several univariably significant CMR variables. However, low event rates are common in patients with DCM, compared with patients with ischaemic heart disease.[33] Another limitation is the lack of data on invasive cardiac catheterization, the gold standard for measurement of ventricular pressure, and assessment of diastolic function. However, in routine clinical practice, invasive assessment is impractical, whereas CMR is frequently performed and widely available, underlining the importance of identification and correlation of various CMR parameters in patients with DCM.

Conclusions

Right ventricular dysfunction was primarily seen in patients with DCM with more severe symptomatic heart failure and advanced myocardial remodelling, including worse LV dysfunction, presence of septal midwall LGE, and more LA dilatation and dysfunction. This suggests that RV dysfunction is secondary to LV impairment. During follow-up, RV dysfunction was associated with shorter time to the combined endpoint of all-cause mortality and VA, driven by the significantly higher VA event rate in patients with RV dysfunction.

Conflict of interest

None declared.

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