



The comparison of short-term prognostic value of T1 mapping with feature tracking by cardiovascular magnetic resonance in patients with severe dilated cardiomyopathy

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Received: 16 April 2018 / Accepted: 17 August 2018 / Published online: 21 August 2018
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Abstract

To evaluate and compare the prognostic value of T1 mapping with feature tracking cardiovascular magnetic resonance (FT-CMR) imaging in patients with severe dilated cardiomyopathy (DCM) during short-term follow-up. A total of 46 patients with severe DCM (LVEF < 35%) underwent 3.0-T CMR with T1 mapping and FT-CMR analysis. The study end-point was defined as a combination of cardiac death, heart transplantation, and hospitalization due to cardiovascular events. The significance of the risk factors was mainly evaluated by univariate and multivariate Cox model analyses. During the median follow-up of 13 months (interquartile range 7–17 months), two patients died of heart failure, one received a heart transplantation, and six were hospitalized for heart failure. In the univariate analysis, extracellular volume fraction (ECV) showed significant predictive association with cardiovascular events (hazard ratio [HR] 1.35; 95% confidence interval [CI] 1.13–1.62; $P=0.001$). No strain parameters in FT-CMR differed significantly between patients with or without events (all $P>0.05$). In the multivariate analyses, ECV was the sole independent predictor of cardiovascular events (HR, 1.48; 95% CI 1.13–1.94; $P=0.005$). The area under the curve of the time-dependent receiver operating characteristic in leave-one-out cross-validation (all >0.70) further confirmed the predictive significance of ECV. In patients with severe DCM, ECV was not only a strong independent predictor of adverse cardiovascular events but also provided prognostic value prior to strain parameters of the FT-CMR in the short term.

Keywords Cardiovascular magnetic resonance · Short-term prognostic values · T1 mapping · Feature tracking · Severe dilated cardiomyopathy

Abbreviations

DCM	Dilated cardiomyopathy	STE	Speckle tracking echocardiography
CMR	Cardiovascular magnetic resonance	GLS	Global longitudinal strain
LVEF	Left ventricular ejection fraction	GCS	Global circumferential strain
LVEDVI	Left ventricular end-diastolic volume index	IQR	Interquartile range
LGE	Late gadolinium enhancement	ROI	Region of interest
ECV	Extracellular volume fraction	ROC	Receiver operating characteristic
FT-CMR	Feature tracking cardiovascular magnetic resonance	AUC	Area under the curve

Electronic supplementary material The online version of this article (<https://doi.org/10.1007/s10554-018-1444-8>) contains supplementary material, which is available to authorized users.

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Introduction

Dilated cardiomyopathy (DCM) is a common form of cardiomyopathy characterized by left ventricular (LV) cavity enlargement and impaired contractility [1]. DCM contributes to high 5-year cardiovascular morbidity and mortality rates (up to 20%) [2].

Clinically, the risk stratification of DCM patients is highly based on the assessment of left ventricular ejection fraction (LVEF). DCM patients with significant LV impairment (LVEF < 35%) are recommended for defibrillator implantation, resynchronization therapy, and heart transplantation in the current guidelines [3]. However, its predictive performance in patients with severe systolic dysfunction seems insufficient when it relies mainly on LVEF [4, 5]; therefore, the prediction of cardiovascular events in patients with severe systolic function impairment cannot rely mainly on LVEF [6]. The identification of additional predictors in CMR may be helpful for further risk stratification and therapy management in such high-risk patients.

Lately, feature tracking cardiovascular magnetic resonance (FT-CMR) imaging or speckle tracking echocardiography (STE) has been used to quantify myocardial strain [7]. A recent guideline reported that the strain parameters in STE were able to identify patients with heart failure and preserved EF [8]. Furthermore, a previous study of DCM patients demonstrated that the strain parameters obtained from FT-CMR can provide incremental prognostic information [9]. It was also reported that strain parameters are the predictor of LV reverse remodeling in patients with severe DCM [10].

In addition, approximately 30% of DCM patients have a characteristic mid-wall pattern of focal fibrosis on the late gadolinium enhancement (LGE) technique in CMR, an indicator of poor outcomes independent of LVEF [5]. However, LGE is less suitable for quantifying the extent of extracellular matrix (ECM) expansion resulting from diffuse myocardial interstitial fibrosis. T1 mapping, a noninvasive technique, enables the quantification of diffuse fibrosis mainly by measuring native T1 and extracellular volume fraction (ECV) [11]. Native T1 and ECV reflect the histological degree of myocardial fibrosis [12] and provide prognostic value in patients with relatively mild DCM [13].

Though both techniques have shown important prognostic values from prior literatures, a comparison of their short-term prognostic values in patients with severe DCM were lacking. The study aimed to evaluate and compare the prognostic value of T1 mapping with FT-CMR techniques in patients with severe DCM during short-term follow-up.

Methods

Study population

CMR was performed in patients diagnosed with severe DCM (> 18 years old) between March 2015 and March 2017. The inclusion criteria were as follows: (a) increased left ventricular end-diastolic volume index (LVEDVI) compared with the reference range [14]; and

(b) LVEF < 35% in CMR. The exclusion criteria were as follows: (a) significant coronary artery disease (defined as $\geq 50\%$ luminal stenosis), previous coronary revascularization, or myocardial infarction; (b) valvular disease; (c) congenital heart disease; and (d) estimated glomerular filtration rate < 30 mL/min/1.73 m² or implanted devices. All patients were examined in a clinically stable condition (New York Heart Association functional class \leq III). Additionally, a total of 24 age- and sex-matched healthy controls with no history of cardiac disease, no diabetes mellitus and normal in CMR were selected.

Follow-up

The study end-point was defined as the occurrence of cardiac death, heart transplantation, and hospitalization due to cardiovascular events. Follow-up information was obtained from in-person or telephone interview at 3-month intervals. Event-free periods were tracked for > 6 months. Time to event was defined as the duration from the date of the CMR scan to an event.

CMR acquisition

CMR was performed on a 3T system (Ingenia, Philips Medical Systems, Best, the Netherlands). The body coil was used for radiofrequency transmission with 32 elements for reception. Along with long-axis planes (two-, three-, and four-chamber views), a stack of short-axis single-shot balanced standard steady-state in free-precession sequence images from apex to basal were collected. The imaging parameters were as follows: field of view, 230 mm \times 230 mm; voxels, 2 mm \times 2 mm \times 8 mm; repetition time, (3.0–3.2) ms; echo time, (1.5–1.6) ms; sense factor, 2; minimum inversion time, 105 ms; and flip angle, 45°.

T1 mapping was performed for all participants in a single mid-ventricular short-axis view. For the pre-contrast, MOLLI 5s(3s)3s scheme was performed. A total dose of 0.2 mmol/kg gadopentetate dimeglumine injection (Consun Pharmaceutical CO., LTD.) was administered. For post-contrast, a 4s(1s)3s(1s)2s scheme was performed (15–17) min after the ejection [1]. The imaging parameters were as follows: field of view, 230 mm \times 230 mm; voxels, 2 mm \times 2 mm \times 8 mm; sense factor, 2; minimum inversion time, 105 ms; and flip angle, 20°.

LGE images were performed along the long-axis and short-axis views using phase sensitive inversion recovery about (10–11) min after the ejection. The imaging parameters were as follows: field of view, 230 mm \times 230 mm; voxels, 2 mm \times 2 mm \times 8 mm; repetition time, (6.0–6.2) ms; echo time, (3.0–3.1) ms; and TI, measured at that time.

Image analysis

The FT-CMR analysis was performed in balanced steady-state-free-precession cine images in QMass 8.1 and QStrain 2.0 (Medis, Leiden, the Netherlands). The endocardial and epicardial borders of the LV were manually sketched in the end-diastolic and end-systolic phases, (Figs. 2 and 3 in the Supplementary materials) respectively, and trabeculations were all excluded from the endocardial borders. Global longitudinal strain (GLS) was obtained from two-, three-, and four-chamber views for LV. Global circumferential strain (GCS) was obtained from the basal, middle, and apical levels of the LV in the short-axis view.

LGE extent was quantified using a semiautomatic detection method using full width at half-maximum and reported as a percentage of total LV mass in QMass 8.1 (Medis) and reported as a percentage of total LV mass. T1 mapping raw images were analyzed in QMapEC ver. 2.2.18 (Medis). Myocardial T1 times were measured carefully in a global region of interest (ROI) at whole the mid-ventricular wall; meanwhile, an ROI was drawn in blood pool to measure blood pool T1 times. ECV was calculated as follows:

$$ECV = (1 - \text{hematocrit}) \times \left[\frac{1}{\text{myo post T1}} - \frac{1}{\text{myo native T1}} \right] / \left[\frac{1}{\text{blood post T1}} - \frac{1}{\text{blood native T1}} \right] \quad (1)$$

The details were shown in Figs. 1, 2, 3 in Supplementary.

Every participant agreed to undergo the blood test for hematocrit within 24 h before the CMR scan.

Statistics

Normality of the distribution was tested using the Shapiro–Wilk test. Normally distributed continuous variables are described as mean \pm standard deviation, while skewed variables were reported as median and interquartile range (IQR). Binary variables were recorded as “0” or “1” and expressed as number (percentage). Student’s t-test was used to compare means of the normally distributed variables, while the Wilcoxon-rank test was used to compare the median of the skewed variables. Binary variables were compared using Fisher’s exact test. After testing for normality, correlation coefficients were described as Pearson r values or otherwise expressed as Spearman r values. All tests were two-sided, and P values < 0.05 were considered statistically significant.

Univariate analysis of the outcomes of patients with severe DCM was performed using the Cox proportional hazards model with the Breslow test. To determine the optimal cutoff value of the significant variable, the Youden test

was performed of the data computed from receiver operating characteristic curves (ROC). Survival curves for the significant markers were established by the Kaplan–Meier estimation method using the optimal cutoff value, while the event rates were compared using the log-rank test. The statistically significant variables in the univariate analysis were added to the multivariate Cox model analysis. However, for just two variables (ECV, LVEDVI) that satisfied the rule in univariate analysis, the extra variables (e.g. sex, systolic blood pressure, and creatine [1, 4]) from baseline and clinical data considered as risk factors in the clinical setting were manually added to the multivariate Cox model, which aimed to adjust the independent predictors. To further evaluate and confirm the predictive performance of the multivariate Cox regression models, we employed the time-dependent receiver ROC [15] and leave-one-out cross-validation.

All statistical analyses were performed with statistical software GraphPad Prism (version 6.0; GraphPad Software, San Diego, California, USA) and in R version 3.4.1 (R Foundation, Vienna, Austria). We used the “survival”, “survivalROC” and “forestplot” packages.

Results

Baseline

All baseline characteristics were depicted (Table 1). During a median follow-up of 13 months (IQR, 7–17 months), two patients died of heart failure, one underwent heart transplantation, and six were hospitalized with heart failure. The median time to the development of cardiovascular events was 6 months (IQR, 3–7 months), while patients without cardiovascular events survived for 14 months (IQR, 9–18 months). The MAGGIC Score (18.9 ± 4.3 vs. 18.1 ± 4.9 , $P = 0.676$) was similar between groups. With regard to sex, age, and medication, there was no significant difference between patients.

CMR

All CMR parameters are shown in Table 2. LVEF in patients with severe DCM was 20.0% (IQR, 15.0–27.5%), further GLS or GCS was $-8.45 \pm 3.8\%$ and $-6.25 \pm 2.9\%$, respectively. On tissue characterization, LGE was present in 37 (80%) patients and the median LGE extent was 8.68% (IQR, 1.84–12.7%). As for T1 mapping, native T1 was 1367 ± 79 ms and ECV was 30.4% (IQR, 26.9–36.2%).

LVEF had a strong and significant correlation with GLS ($r = -0.899$, $P < 0.001$) and GCS ($r = -0.859$, $P < 0.001$), respectively. However, T1 values had a weak correlation

Table 1 Patients' and controls' characteristics

	All patients n=46	Control n=24	Event n=9	No event n=37	P value
Age (years)	46.7 ± 12.9	45.7 ± 13.6	40.9 ± 9.01	48.1 ± 13.4	0.134
Gender (male, %)	33 (72%)	17 (71%)	7 (78%)	26 (70%)	> 0.999
Body mass index (kg/m ²)	23.8 (21.7, 27.0)	23.5 (21.0, 25.3)	22.8 (20.9, 25.1)	24.5 (22.3, 27.1)	0.570
Systolic blood pressure (mmHg)	114.4 ± 13.0*	119.8 ± 7.2*	108.8 ± 14.9	115.8 ± 12.3	0.149
Diastolic blood pressure (mmHg)	72.8 ± 9.5	74.4 ± 7.1	71.1 ± 11.8	73.3 ± 9.1	0.548
Heart rate (beats/min)	80.0 ± 14.6*	69.0 ± 11.1*	77.0 ± 14.4	80.8 ± 14.8	0.493
Hematocrit (%)	43.9 ± 5.9*	41.2 ± 1.7*	42.8 ± 5.4	44.2 ± 5.3	0.474
New York functional class = III (n, %)	20 (43%)		6 (67%)	16 (38%)	0.278
NT-proBNP (Pg/mL)	2211 (816, 4260)		3199 (2746, 4016)	1727 (751, 4179)	0.168
CREA (μmol/L)	80.9 (66.3, 92.7)		80.9 (75.0, 91.8)	80.9 (63.7, 102.6)	0.814
Hypertension (n, %)	8 (17%)		1 (13%)	7 (19%)	> 0.999
Atrial fibrillation (n, %)	9 (20%)		1 (11%)	8 (22%)	0.664
Diabetes mellitus (n, %)	7 (15%)		2 (22%)	5 (14%)	0.609
Hyperlipidemia (n, %)	14 (30%)		3 (33%)	11 (30%)	> 0.999
Smoke (n, %)	12 (32%)		3 (33%)	9 (24%)	0.678
Alcohol (n, %)	6 (13%)		1 (13%)	5 (14%)	> 0.999
Family history of cardiomyopathy	3 (7%)		1 (11%)	2 (5%)	0.488
MAGGIC SCORE	18.3 ± 4.8		18.9 ± 4.3	18.1 ± 4.9	0.676
Medicine					
Beta-blocker (n, %)	45 (98%)		9 (100%)	36 (97%)	> 0.999
ACE-inhibitors (n, %)	39 (85%)		7 (77.8%)	32 (86.5%)	0.609
Angiotensin II receptor blockers (n, %)	11 (24%)		0 (0%)	11 (32.4%)	0.089
Spironolactone (n, %)	41 (89%)		7 (88.9%)	34 (91.9%)	> 0.999
Diuretics (n, %)	45 (98%)		9 (100%)	36 (97.3%)	> 0.999
Digoxin (n, %)	25 (54%)		5 (55.6%)	20 (54.1%)	> 0.999
Function and structure					
LVEF (%)	20.0 (15.0, 27.5)*	60.0 (57.0, 64.0)*	25.0 (15.0, 26.0)	20.0 (15.0, 28.0)	0.835
LVEDVI (mL/m ²)	201.7 ± 50.8*	83.24 ± 9.4*	230.9 ± 66.2	194.6 ± 44.5	0.053
LVESVI (mL/m ²)	159.4 (117.3, 187.5)*	33.01 ± 5.4*	178.2 (129.9, 211.4)	154.1 (114.0, 185.7)	0.293
RVEF (%)	30.2 ± 13.6*	54.50 ± 6.9*	23.2 ± 8.5	31.8 ± 14.2	0.089
RVEDVI (mL/m ²)	108.2 ± 37.7*	76.41 ± 13.9*	130.4 ± 32.8	102.8 ± 37.2	0.048
RVESVI (mL/m ²)	68.0 (45.5, 105.5)*	34.9 ± 9.3*	101.0 (76.0, 111.0)	60.0 (42.0, 93.0)	0.022
Tissue characterization					
LGE present (n, %)	37 (80%)	0 (0%)	7 (78%)	30 (81%)	> 0.999
LGE extent (%)	8.68 (1.84, 12.7)		10.4 (9.93, 14.8)	7.14 (1.74, 12.5)	0.290
Strain					
GLS (%)	-8.45 ± 3.8*	-22.83 ± 3.0*	-8.62 ± 4.2	-8.41 ± 3.7	0.879
GCS (%)	-6.25 ± 2.9*	-20.56 ± 2.9*	-6.35 ± 3.5	-6.22 ± 2.8	0.909
T1 mapping					
ECV (%)	30.4 (26.9, 36.2)*	25.7 (24.2, 28.0)*	37.6 (37.1, 38.8)	29.9 (26.6, 32.7)	0.003
T1-native (ms)	1367 ± 79*	1253 ± 25*	1376 ± 93	1367 ± 77	0.749
T1-post (ms)	532 ± 63*	575 ± 61*	545 ± 74	528 ± 61	0.494

Normally distributed continuous variables are shown as mean ± SD; non-normally distributed continuous variables are shown as median (interquartile range); categorical variables are shown as n (percentage). An intergroup comparison was made. P values compare events and no events (P < 0.05 considered significant). *Indicated the significant difference between patients and controls. Values in bold indicate significance

CMR cardiovascular magnetic resonance, LVEF left ventricular ejection fraction, LVEDVI left ventricular end diastolic volume index, LVESVI left ventricular end systolic volume index, RVEF right ventricular ejection fraction, RVEDVI right ventricular end-diastolic volume index, RVESVI right ventricular end-systolic volume index, LGE late gadolinium enhancement, GLS global longitudinal strain, GCS global circumferential strain, ECV extracellular volume fraction

Table 2 Results of multivariable analyses of cardiovascular events

	Model		
	Hazard ratio	95% CI	P value
Sex (male)	0.66	0.09, 5.00	0.700
Systolic blood pressure (mmHg)	1.04	0.96, 1.14	0.322
Creatine (μmol/L)	1.02	0.98, 1.05	0.440
LVEDVI (mL/m ² × 10)	1.11	0.93, 1.34	0.225
LGE extent (%)	0.96	0.85, 1.09	0.517
ECV (%)	1.48	1.13, 1.94	0.005
Log rank, P < 0.01			

Multivariate analysis results are presented as hazard ratios and 95% confidence intervals (CI). Values in bold indicate significance

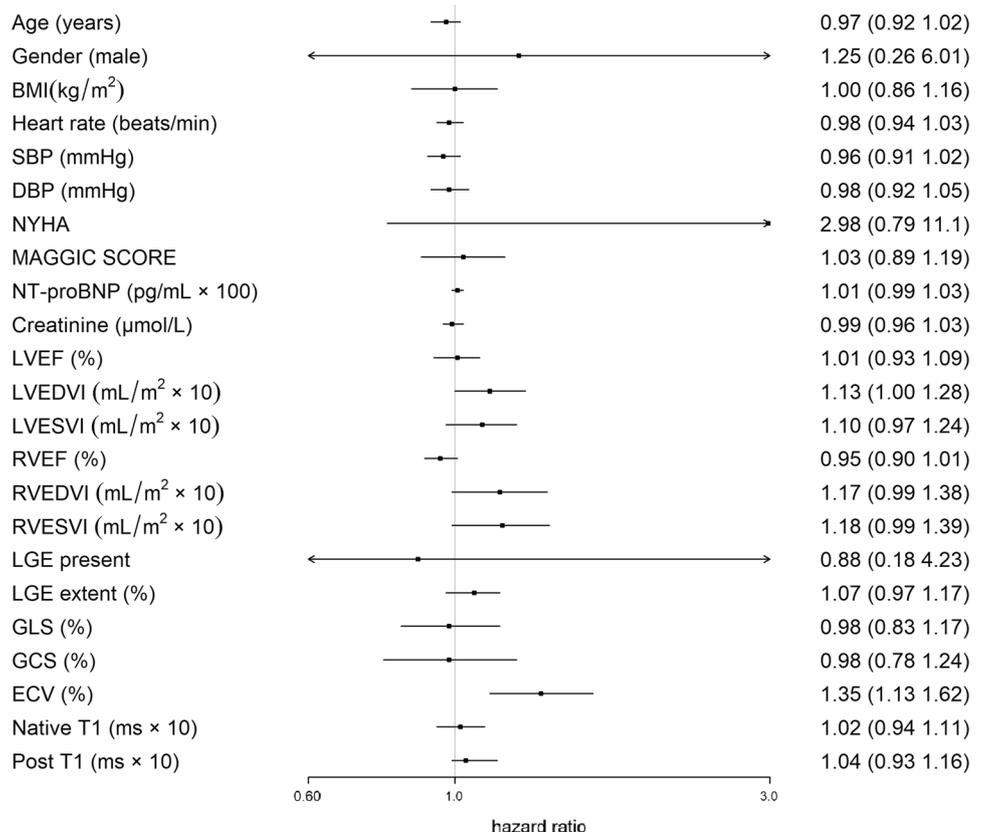
LVEDVI left ventricular end-diastolic volume index, LGE late gadolinium enhancement, ECV extracellular volume fraction

with LVEF and strain parameters (Figs. 9, 10, 11 in Supplementary materials).

Predictors of cardiovascular events

In univariate analysis, ECV showed significant predictive associations with cardiovascular events (P = 0.001), (hazard ratio [HR] 1.35; 95% confidence interval [CI] 1.13–1.62)

Fig. 1 Hazard ratios on univariate Cox regression model. Univariable analyses results are presented as unadjusted hazard ratios and 95% confidence intervals. BMI body mass index, SBP systolic blood pressure, DBP diastolic blood pressure, NYHA New York Heart Association functional classification, LVEF left ventricular ejection fraction, LVEDVI left ventricular end-diastolic volume index, LVESVI left ventricular end-systolic volume index, RVEF right ventricular ejection fraction, RVEDVI right ventricular end-diastolic volume index, RVESVI right ventricular end-systolic volume index, GLS global longitudinal strain, GCS global circumferential strain, ECV extracellular volume fraction



(Fig. 1). LVEDVI was mildly related with cardiovascular events (P < 0.05), (HR 1.13 and 95% CI 1.00–1.28).

Furthermore, after adjustment of the multivariate Cox proportional-hazards analysis, the results indicated that ECV (P < 0.001) was the significantly independent predictor of cardiovascular events. In the leave-one-out cross-validation of the predicting models, the predictive performances were all satisfactory (all AUC > 0.70, the figures showing time-dependent ROC analysis were shown in Supplementary materials). Kaplan–Meier curves (Fig. 2) were shown using optimal cut-off value (ECV = 35.5%, P < 0.001). In detail, eight of 13 patients in the group with higher ECV suffered from adverse cardiovascular events versus one of 33 patients in the other group.

Discussion

In this study, the prognostic value of combined FT-CMR with T1 mapping was explored in patients with severe DCM in short-term follow-up. Our results found that ECV was a significantly strong predictor of adverse cardiovascular events over that of strain parameters from FT-CMR.

We found that patients with elevated ECV had higher risks of cardiovascular events in Kaplan–Meier curves. Our results supported previous studies that demonstrated that

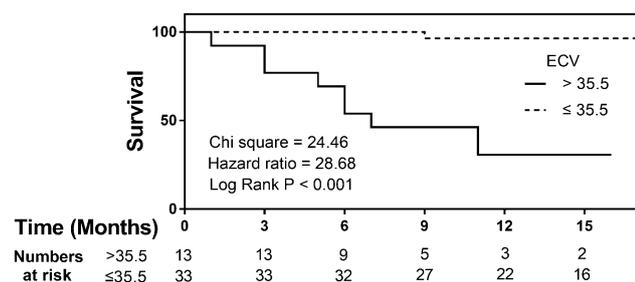


Fig. 2 Kaplan–Meier curves of extracellular volume fraction (ECV), optimized cutoff values using Youden test, and P values

ECV could provide strong prognostic value in T1 mapping [13, 16]. The histological basis of ECV reflected the degree of myocardial fibrosis, an intermediate marker of disease severity and major cause of death in DCM [12, 17]. Miller et al. showed a similar correlation between ECV and collagen volume fraction in histological whole-heart studies of explanted DCM hearts after transplantation [18].

In our study, native T1 in T1 mapping, a controversial indicator, lacked the ability to predict cardiovascular events. Although Puntmann et al. showed that native T1 could play a central role in risk stratification [13], Hong et al. demonstrated that it failed to identify controls and DCM patients [19]. Native T1 is specific to the field strength, vendors, cardiac phase, schemes, and operators [20]; consequently, its reproducibility is limited.

LVEF is currently recognized as the major determining factor of risk stratification, but it failed to identify patients with or without events in our study. It was reported that when LVEF was < 25%, the relationship between LVEF and survival probabilities was reportedly weaker [4]. In this situation, patients with severe systolic impairment accepted medical therapy with improved LV functional index [21]. Therefore, it is difficult to predict the adverse outcome using LVEF alone, especially in patients with severe systolic impairment; the novel predictors like ECV require exploration.

Our results demonstrated that GLS and GCS were not the significant predictors of adverse cardiovascular events, which differed from many studies in STE or FT-CMR in the cohorts of patients with varying severity of LVEF ranging from mild to severe DCM [9, 22]. The main reason for the differences across these studies has been attributed to the differences in patient cohorts; in our study, LVEF failed to predict adverse events. Evidence from previous studies showed that myocardial strain served as an indicator of reversible cardiac abnormalities prone to the development of structural cardiac damage and dysfunction in early-stage DCM [8, 23]. However, in the advanced stage of DCM, the deformation ability of the myocardium was significantly impaired in all directions, strongly in parallel to LV systolic dysfunction severity [24]. In our study, strain parameters

were strongly associated with LVEF; consequently, the relationship between strain parameters and survival probabilities also became weaker.

Our findings demonstrated that ECV was an independent predictor with prognostic value and that neither systolic dysfunction nor deformation ability was associated with ECV in our results. Despite DCM patients with elevated ECV exhibited more severe systolic impairment in a previous study [19], Siepen et al. reported a weak correlation between systolic function and ECV [12], which supported our findings. Furthermore, Wong et al. shared a similar conclusion that ECV was an independent indicator of poor outcomes [16].

Limitations

This study had several limitations. First, it was a single-center study with a single CMR protocol and interpretation. The validation of our conclusion in a multi-center study is required. Second, the number of patients was insufficient, which limited its the strength of evidence.

Conclusion

In patients with severe DCM, ECV was a significantly strong predictor of adverse cardiovascular events over strain parameters in FT-CMR in the short term. Therefore, considering ECV a priority could lead to more efficient risk stratification and management decisions in patients with severe DCM.

Acknowledgements The study received funding from the National Natural Science Foundation of China (No. 81771799), Guangdong Provincial Science and Technology Planning Project (No. 2014A020212676), and Science and Technology Program of Guangzhou, China (No. 201707010306).

Author contributions RC drafted the manuscript; JW, JX and WW acquired the data; YHJ, CWSC, HF, LL, JM, SW, CL revised the manuscript; RC, ZD, YZ and ZY provided the analysis method. HL provided the conception and design of the study.

Compliance with ethical standards

Conflict of interest The authors declare no conflicts of interest.

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