Original Research

The Value of a New Speckle Tracking Index Including Left Ventricular Global Longitudinal Strain and Torsion in Patients with Dilated Cardiomyopathy

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Key words: **Dilated cardiomyopathy, left ventricular torsion, global longitudinal strain, speckle tracking echocardiography.**

Introduction: Torsional and longitudinal deformations are essential components of left ventricular (LV) performance. We believe that a precise assessment of LV function must take into account both LV torsion (LVtor) and global longitudinal strain (LVε). Therefore, we investigated with speckle tracking echocardiography the value of a new parameter, LVtor × LVε, for assessing LV function in dilated cardiomyopathy (DCM) and validated it against N-terminal pro-brain natriuretic peptide (NTproBNP).

Methods: Echocardiography was performed simultaneously with NTproBNP determination in 55 consecutive patients with DCM in sinus rhythm. The ratio of early diastolic transmitral velocity to early mitral annular diastolic velocity (E/E') was measured. LVtor was defined as the ratio between LV twist (LVtw) and LV end-diastolic longitudinal length. LVtw (net difference between rotation angles at base and apex) was obtained from parasternal apical and basal short-axis planes. LVε was obtained by averaging longitudinal peak systolic strain of all 17 LV-segments (from apical planes).

Results: Log-transformed NTproBNP correlated significantly with LVε ($r=0.56$, $p<0.001$), E/E' ($r=0.52$, $p<0.001$), LVtor ($r=-0.41$, $p=0.003$), LVtw ($r=-0.38$, $p=0.004$) and LV ejection fraction ($r=-0.37$, $p=0.005$). LVtor × LVε had the strongest correlation with log-NTproBNP ($r=0.71$, $p<0.001$). LVtor × LVε was a better predictor of NTproBNP levels >900 pg/ml (sensitivity 73%, specificity 82%) than LVε, E/E', LVtw, LVtor and LV ejection fraction (each $p<0.05$).

Conclusions: This study demonstrates that in patients with DCM in sinus rhythm, the evaluation of LV function can be accurately accomplished by using a new speckle tracking index, LVtor × LVε.

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Left ventricular (LV) function results from the contraction and relaxation of helically oriented myofibres. In the LV myocardial wall, myofibre geometry changes smoothly from a right-handed helix in the sub-endocardium to a left-handed helix in the sub-epicardium and the helix angle varies continuously from positive at the endocardium to negative at the epicardium.[1,2] Electrical activation and deformation propagate in apico-basal and endo-epicardial directions, synchronising the counter-directional layers into a single synergistically functioning system.[1] LV torsion (LVtor) is a critically important mechanism for the efficiency of LV function.[2-5] On the other hand, systolic performance expressed as global longitudinal strain (LVε) declines progressively in worsening heart failure.[6] In patients with dilated cardiomyopathy (DCM), altered LV geometry is associat-

ed with a reduction of systolic torsion and longitudinal strain.[7,8] Recently, speckle tracking echocardiography (STE) was successfully used for the measurement of myocardial deformation and rotation.[9-11] N-terminal pro-brain natriuretic peptide (NTproBNP) has been used for the non-invasive assessment of LV function[12,13] and has well established prognostic implications.[14]

We believe that a precise assessment of LV function must take into account both LVtor and LVε. Therefore, we investigated using STE the value of a new parameter, LVtor × LVε, for assessing LV function in DCM in comparison with NTproBNP. We supposed that the small subclinical changes in the deformation and rotation of myocardial layers are amplified by multiplying LV global longitudinal strain (controlled predominantly by sub-endocardial fibres) by LV torsion (related predominantly to the sub-epicardial fibres).[1-4]

Methods

Patients

We analysed 76 consecutive patients with DCM who were in sinus rhythm and were referred for aetiological evaluation. The diagnosis of DCM[15] was established when LV ejection fraction was <45% and LV end-diastolic dimension >112% of the predicted value, corrected for age and body surface area, in the absence of the following conditions: systemic arterial hypertension, coronary artery disease (obstruction >50% of the luminal diameter in a major branch), valvular heart diseases, and congenital heart diseases.

Twenty one patients were excluded: inadequate echocardiographic images in 11 patients, paced rhythm in 5, renal failure (defined as serum creatinine >2 mg/dL) in 4, and mitral prosthesis in one patient. The 55 remaining patients with DCM were included in the analysis and represented the study population.

All study participants underwent clinical examination, 12-lead electrocardiogram, transthoracic echocardiogram, NTproBNP measurements and coronary angiography. All patients were on appropriate medical therapy, including beta-blockers, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, and diuretics.

The study complies with the Declaration of Helsinki and was approved by the local research ethics committee. Informed written consent was obtained from all patients.

Echocardiography

Conventional echocardiography and tissue Doppler imaging were performed simultaneously with NT-proBNP measurements. M-mode, two-dimensional and Doppler echocardiographic examinations were performed with an ultrasonographic system (Vivid 7, General Electric, Milwaukee WI, USA) equipped with a multi-frequency transducer (M3S 1.5-4.0 MHz), in accordance with guidelines.[16,17] All images were stored digitally and analysed off-line with EchoPac PC Dimension software (GE Medical). LV ejection fraction was calculated from apical two- and four-chamber views using LV volumes by the modified biplane Simpson rule, in accordance with guidelines.[16] LV end-diastolic volume and LV end-systolic volume were indexed to body surface area. Peak early (E) and late (A) filling velocities, E/A ratio, and E-velocity deceleration time were measured from the LV-inflow pattern at the tips of the mitral valve at end expiration. Measurement of systolic pulmonary artery pressure was performed using the maximal regurgitant velocity at the tricuspid valve by continuous Doppler.[17]

Peak systolic (S') and peak early diastolic velocities (E') were obtained at the septal and lateral sites of the mitral annulus by pulse wave tissue Doppler imaging from apical four-chamber views. All velocities were recorded for three consecutive cardiac cycles during end-expiratory apnoea, and the results were averaged. The E/E' ratio was then calculated using the average of the septal and lateral E'.[18,19]

Cardiac rotation was computed using STE. Grey-scale digital cine loops triggered to QRS complexes were acquired from two LV short-axis planes: at LV basal level with the cross-section as circular as possible (identified by the mitral valve), and at apical level (distally to the papillary muscles with an optimised transducer position to ensure a proper, circular short-axis cut with no papillary muscles present).[20,21] In each plane, three cardiac consecutive cycles were recorded during breath-hold at a frame rate of 70-100 frames/s and stored on hard disk for subsequent off-line analysis. Counter-clockwise rotation was marked as a positive value and clockwise rotation as a negative value when viewed from the apex. The LV twist curve was generated by calculating the difference between apical and basal rotations at each corresponding time point. The software used did not allow calculation of LV twist by this method if there was a significant difference in heart rate between cycles. The

peak difference between rotation angles at the apex and base was used in our study (LVtw). Peak LVtor was derived from LVtw divided by LV diastolic longitudinal length, as described previously.[9,21]

STE was also used for myocardial deformation measurements. Longitudinal peak systolic strain was determined from apical planes (four-, three- and two-chamber views) in a 17-LV-segments model. The value of LVε was obtained by averaging all 17 LV segments.[6,22] The LVtor × LVε index was then calculated. Off-line analysis was performed by two observers blinded to the clinical data.

NTproBNP measurement

NTproBNP levels were measured in blood samples collected by venipuncture into EDTA tubes, within 30 minutes before or after echocardiography. The automated electrochemiluminescence immunoassay (Elecsys proBNP, Roche Diagnostics, Germany) was used. We used a cut-off of 900 pg/ml for NTproBNP, as recommended in the PRIDE study (NTproBNP Investigation of Dyspnoea in the Emergency Department).[13]

Statistical analysis

Numeric variables are presented as mean value ± standard deviation (SD) and were compared using Student's t-test or analysis of variance, as appropriate. Categorical variables as absolute values and frequency percentages were compared using the χ^2 test. NTproBNP was log-transformed because its distribution was skew. Pearson's correlation was used to investigate relations between variables. We performed multivariate logistic regression analysis to assess the influence on NTproBNP of variables reaching statistical significance on univariate analysis ($p<0.05$). Receiver operating characteristic (ROC) curves were constructed to determine the optimal sensitivity and specificity. All statistical analyses used the software package SPSS version 11.5 (SPSS Inc, Chicago IL, USA). A p value <0.05 was accepted as statistically significant.

Results

The study included 55 consecutive patients (mean age: 62 ± 12 years; 35 men) with DCM, in sinus rhythm. The aetiology of DCM was as follows: toxic in 26 patients, idiopathic in 19 patients, post-infective (in 7 patients), peripartum (in 2 patients), and immune (in 1 patient). The characteristics of the study population are presented in Table 1.

Simple regression analysis demonstrated the highest coefficient of linear correlation between log-NTproBNP and LVtor × LVε ($r=0.71$, $p<0.001$) (Figure 1a). Significant correlations were also found between log-NTproBNP and LVε ($r=0.56$, $p<0.001$) (Figure 1b), LVtor ($r=-0.41$, $p=0.003$) (Figure 1c), LVtw ($r=-0.38$, $p=0.004$), E/E' ($r=0.52$, $p<0.001$) (Figure 1d), systolic pulmonary artery pressure ($r=0.46$, $p<0.001$), S' ($r=-0.45$, $p<0.001$), LV ejection fraction ($r=-0.37$, $p=0.005$). No significant correlation was found between log-NTproBNP and E-velocity deceleration time, E wave, E/A ratio, LV end-diastolic volume or LV end-systolic volume.

The area under the receiver-operating characteristic curve (AUC) for predicting NTproBNP levels >900 pg/ml was greatest for LVtor × LVε (AUC=0.80, $p<0.001$) (Figure 2), followed by LVε (AUC=0.74, $p<0.001$), E/E' (AUC=0.72, $p<0.001$),

Table 1. Baseline characteristics of the study population. Data are presented as mean ± SD or absolute values (%).

Variable	Value
Mean age (years)	62 ± 12
Female/male gender	20 (36.4%) / 35 (65.6%)
Body surface area (m^2)	1.8 ± 0.4
Heart rate (beats/min)	83 ± 14
New York Heart Association:	
class II	16 (29%)
class III	25 (45.5%)
class IV	14 (25.5%)
Mean arterial pressure (mmHg)	98.5 ± 14.2
Aetiology of DCM:	
Toxic	26 (47.4%)
Idiopathic	19 (34.5%)
Post-infective	7 (12.7%)
Peripartum	2 (3.6%)
Immune	1 (1.8%)
LV end-diastolic diameter index (cm/m^2)	4.1 ± 0.9
LV end-systolic diameter index (cm/m^2)	3.5 ± 1.2
LV end-diastolic volume index (ml/m^2)	137 ± 51
LV end-systolic volume index (ml/m^2)	98 ± 42
LV ejection fraction (%)	29 ± 10
E/E' ratio	17 ± 9
LV strain (%)	-5.6 ± 2.5
LV twist (°)	6.2 ± 2.7
LV torsion (°/cm)	0.96 ± 0.42
LV torsion × LV strain (% × °/cm)	-5.5 ± 2.9
Pulmonary artery systolic pressure (mmHg)	46 ± 18
NTproBNP (pg/ml)	4047 ± 3953
Serum creatinine (mg/dl)	1.21 ± 0.5
Haemoglobin (g/dl)	12.6 ± 1.7

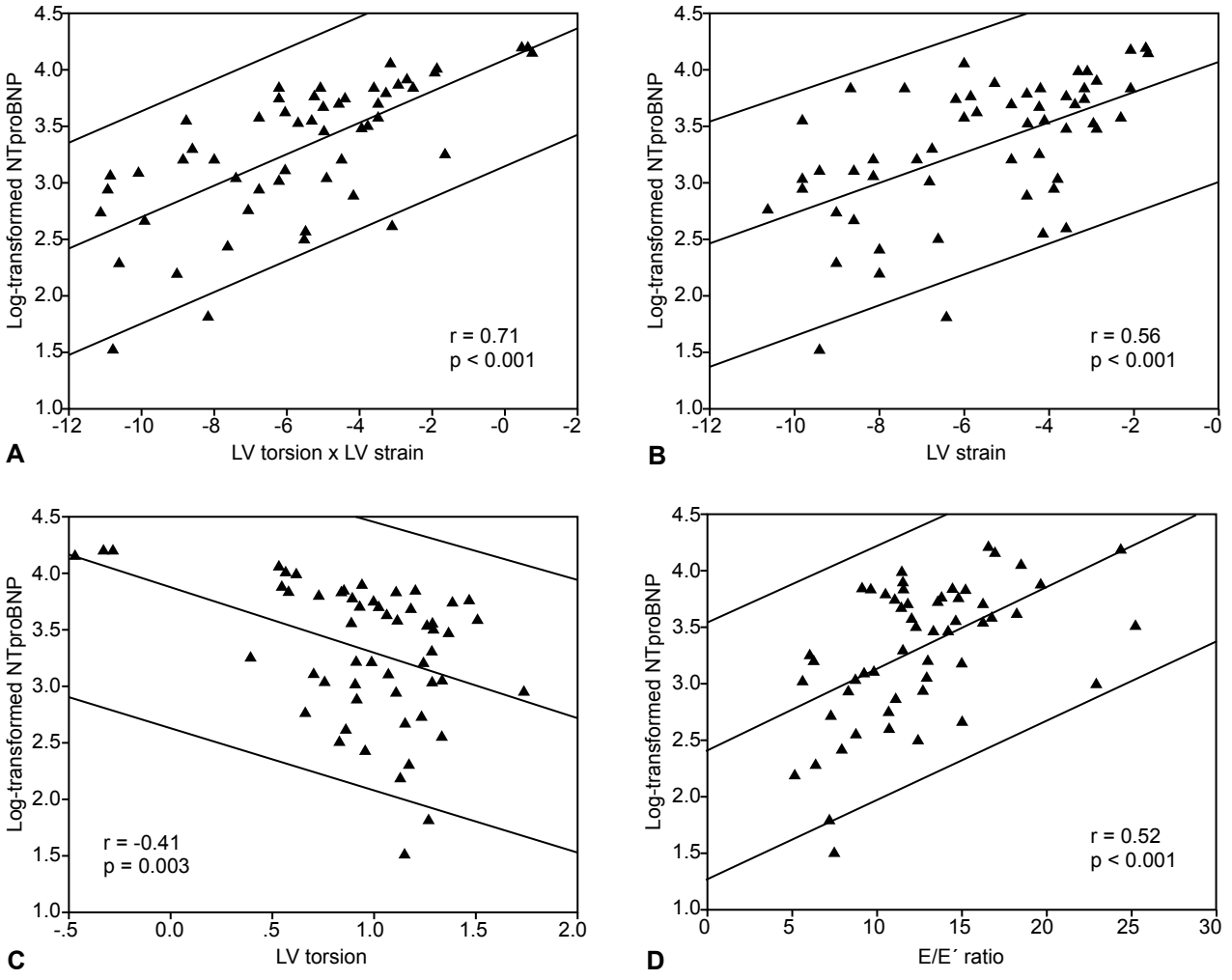

Figure 1. Scatter plot of the relationship between LV torsion × LV strain (panel A), LV strain (panel B), LV torsion (panel C), E/E' ratio (panel D) and log-transformed N-terminal pro-brain natriuretic peptide (NTproBNP) in the 55 patients with dilated cardiomyopathy. E – maximal early diastolic transmitral velocity; E' – maximal early mitral annular diastolic velocity using the average of the medial and lateral site of mitral annulus; LV – left ventricle.

LVtw (AUC=0.62, p<0.001) and LVtor (AUC=0.59, p<0.001). LVtor × LVε was more accurate than LVε, E/E', LVtw and LVtor (each p<0.05) for predicting NTproBNP levels >900 pg/ml. The optimal LVtor × LVε cut-off for predicting NTproBNP levels >900 pg/ml was -6.8 % × °/cm, with a sensitivity of 73% and a specificity of 82%.

On multivariable analysis (Table 2), including LVtor × LVε, LVtw, LVtor, LVε, E/E' ratio, LV ejection fraction, pulmonary artery systolic pressure and S' wave as candidate variables, LVtor × LVε (β=0.96, p=0.003) emerged as the best independent predictor of NTproBNP levels (r^2=0.68; t=7.9; p<0.001).

As reversed apical rotation identifies a subgroup of patients with more advanced disease,[3] this group was analysed separately. In our patients, four different patterns of LV rotation were noticed: rotation of apex and base in normal directions (26 patients), "paradoxical" (reversed) rotation of apex (clockwise) and base (counter-clockwise) (3 patients), concordant clockwise rotation of apex and base (12 patients) and concordant counter-clockwise rotation of apex and base (14 patients). Thus, 15 patients had reversed (clockwise) apical rotation (group 1) and 40 patients had normally directed (counter-clockwise) apical rotation (group 2). LVtor × LVε, LVtor and LVtw were lower in group 1 than in group 2 (-3.6 ± 2.1 vs. -6.3 ± 2.6 % × °/cm, p=0.04; 0.69 ± 0.66 °/cm vs. 1.06 ± 0.22 °/cm, p<0.01; and 4.0 ± 4.07° vs. 7.01 ± 1.53°, p<0.01, respectively). NTproBNP levels and LV end-diastolic volume index were higher in group 1 than in group 2 (6848 ± 5514 pg/ml vs. 2996 ± 2564 pg/ml, p<0.001;

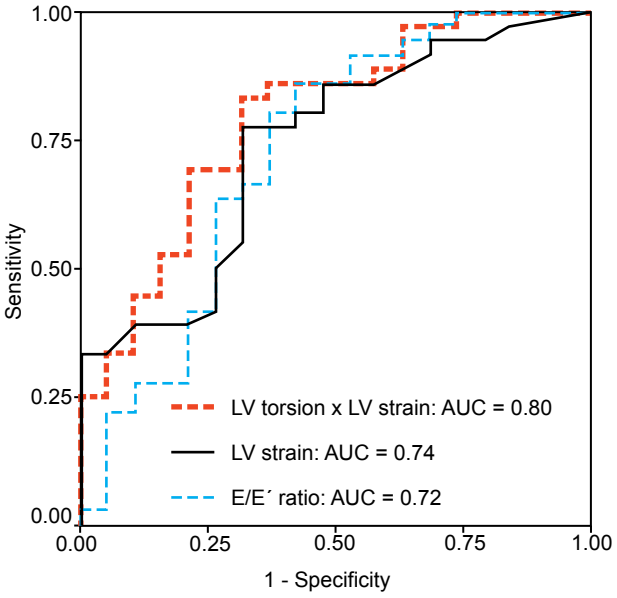

Figure 2. Receiver operating characteristic (ROC) curves for LV torsion × LV strain, LV strain and E/E' ratio for predicting N-terminal pro-brain natriuretic peptide (NTproBNP) levels >900 pg/ml in the 55 patients with dilated cardiomyopathy. Area under the ROC curve (AUC) = 0.80 (95% confidence interval [CI] 0.66-0.94) for LV torsion × LV strain, AUC = 0.74 (95% CI 0.60-0.88) for LV strain, AUC = 0.72 (95% CI 0.58-0.86) for E/E' ratio (p<0.001 for all). Abbreviations as in Figure 1.

Table 2. Results of multivariate analysis in the study population. Data are presented as mean ± SD or absolute values (%).

Determinant of NTproBNP	β	p
LV torsion × LV strain	0.96	0.005
LV twist	0.92	0.008
LV torsion	-0.63	0.063
LV strain	-0.22	0.352
E/E' ratio	0.25	0.009
LV ejection fraction	0.04	0.907
Pulmonary artery systolic pressure	0.21	0.026
S' wave	-0.04	0.748

DCM – dilated cardiomyopathy; E – maximal early diastolic transmitral velocity; E' – maximal early mitral annular diastolic velocity using the average of the medial and lateral site of mitral annulus; LV – left ventricle; NTproBNP – N-terminal pro-brain natriuretic peptide.

143 ± 61 vs. 112 ± 27 ml/m^2, p<0.01). For the body surface area, NYHA class, LV ejection fraction, LVε, S' wave, E/E' and pulmonary artery systolic pressure the difference did not reach statistical significance.

In patients with reversed apical rotation (group 1), simple regression analysis demonstrated the highest coefficient of linear correlation between log-NTproBNP and LVtor × LVε (r=0.80, p<0.001). Significant correlations were also found between log-NTproBNP and LVε (r=0.73, p=0.002), LVtor (r=-0.59, p=0.02), LVtw (r=-0.57, p=0.02), S' (r=-0.71, p=0.003) and E/E'(r=0.67, p=0.006). For LV ejection fraction, E wave, E/A ratio, E-velocity deceleration time, LV end-diastolic volume and LV end-systolic volume, no correlation was found. The area under the ROC curve for predicting NTproBNP levels >900 pg/ml was greatest for LVtor × LVε (AUC=0.91, p<0.001) followed by LVε (AUC=0.85, p<0.001) and LVtor (AUC=0.73, p<0.001). LVtor × LVε was more accurate than LVε and LVtor (each p<0.05) for predicting NTproBNP levels >900 pg/ml.

In group 2, LVtor × LVε was also found to be correlated with log-NTproBNP (r=0.63, p<0.001). The correlation was weaker for the E/E' ratio (r=0.49, p=0.001), LV ejection fraction (r=-0.48, p=0.002), LVε (r=0.46, p=0.003), S' (r=-0.46, p=0.003) and pulmonary artery systolic pressure (r=0.40, p=0.01). For LVtor, LVtw, E wave, E/A ratio, E-velocity deceleration time, LV end-diastolic volume and LV end-systolic volume no correlation was found. The area under the ROC curve for predicting NTproBNP levels >900 pg/ml was greatest for LVtor × LVε (AUC=0.77, p<0.001) followed by LVε (AUC=0.70, p<0.001) and LV ejection fraction (AUC=0.68, p<0.001). LVtor × LVε was more accurate than LVε and LV ejection fraction (each p<0.05) for predicting NTproBNP levels >900 pg/ml.

Because in routine clinical practice LV ejection fraction is the most common estimate of cardiac function, we separately analysed the relationship between LV ejection fraction and STE parameters. Simple regression analysis demonstrated a statistically significant linear correlation between LV ejection fraction and LVtor × LVε (r=-0.56, p<0.001), LVε (r=-0.46, p=0.01), LVtw (r=0.31, p=0.01) and LVtor (r=0.30, p=0.01). The area under the ROC curve (AUC) for predicting LV ejection fraction ≤25% was greatest for LVtor × LVε (AUC=0.79, p<0.001) followed by LVε (AUC=0.71, p<0.001), LVtw (AUC=0.70, p<0.001) and LVtor (AUC=0.65, p<0.01). LVtor × LVε was more accurate than LVε, LVtw and LVtor for the prediction of an LV ejection fraction ≤25% (each p<0.05).

Discussion

In the present study we investigated the additive value of torsion to global longitudinal strain as a new

speckle tracking index of LV function in DCM. Compared with LV ejection fraction, tissue-Doppler derived indices (E/E', S'), and STE parameters that explore LV deformation and rotation (LVtw, LVtor, LVε), the combined index (LVtor × LVε) predicted plasma NTproBNP levels with excellent accuracy.

The ventricles consist of a single myofibre band, starting at the right ventricle just below the pulmonary valve and forming a double helix extending to the LV where it attaches to the aorta.[23] The relationship between LV longitudinal and torsional deformation provides information about the transmural heterogeneity in ventricular contractile function.[2] In the LV wall, myofibre geometry changes smoothly from a right-handed helix in the sub-endocardium to a left-handed helix in the sub-epicardium and the helix angle varies from positive at the endocardium to negative at the epicardium.[1,2,20] LV torsion is an essential component of systolic function and contributes to an efficient ejection, necessary to produce physiological stroke volumes.[24] STE allows accurate measurement of the magnitude, timing, and dynamics of LV torsion and shows excellent correlation with magnetic resonance imaging data.[10] A significant correlation between LV torsion and systolic dysfunction in children with DCM was previously reported by Jin et al.[25]

LV longitudinal strain reflects LV long axis function, controlled predominantly by sub-endocardial fibres.[2,26] LVε shows a progressive decline in parallel with worsening of heart failure.[6] In a study by Yoneyama et al,[27] longitudinal LV strain was closely related to natriuretic peptide levels in patients with congestive heart failure.

In DCM, altered LV ventricular geometry resulting from cardiac remodelling (dilatation, wall thinning, and reduction in fibre angles) is associated with reduction of the systolic torsion and longitudinal strain.[7,8] With LV dilation, the oblique fibre orientation changes to a more transverse direction, leading to decreased twisting, thickening and longitudinal shortening.[25] The present study demonstrates for the first time that the association of two essential components of LV function (LVε and LVtor) provides a close prediction of NTproBNP levels. This cardiac hormone has been used for the non-invasive assessment of LV function[12,28] and has prognostic implications.[14] Our novel parameter, LVtor × LVε, associates the value of peak LV torsion with the value of global LV longitudinal strain and therefore may provide supplementary information compared to each component alone.

The mechanisms responsible for changes in torsion dynamics are unclear, but might relate to LV dilatation, remodelling of cardiac myocytes and connective tissue matrix, and increased electrical dyssynchrony.[25] The loss of LV torsion is mainly due to a decrease in counter-clockwise apical rotation. Apical rotation represents the dominant contribution to LV twist.[29] Popescu et al, in a recent study of patients with DCM, demonstrated that reversed systolic apical rotation is the expression of more advanced disease, with more severe LV remodelling, dyssynchrony, and systolic dysfunction compared to DCM patients with normally directed apical rotation.[3] In our patients, four different patterns of LV rotation were noticed: rotation of apex and base in normal directions, "paradoxical" (reversed) rotation of apex and base, concordant clockwise rotation of apex and base, and concordant counter-clockwise rotation of apex and base. Compared to patients with normal apical rotation, the group with reversed systolic apical rotation presented lower LVtor × LVε, LVtor and LVtw, greater LV end-diastolic volume index, and higher levels of plasma NTproBNP. The combined index showed excellent performance even in this group with advanced disease. LVtor × LVε is a promising parameter that deserves research to establish its clinical meaning and prognostic value.

LVtor × LVε and LVε were moderately correlated with LV ejection fraction, which is the classic parameter of systolic function in routine practice. Our results are in agreement with previous studies. Moderate correlations between LVε and LV ejection measured by Simpson's biplane rule were reported in patients who had a previous myocardial infarction,[30] acute ST-segment elevation myocardial infarction[31] or advanced ischaemic heart failure.[31] Significant correlations between LVtw and LV ejection fraction were noted by Takeuchi et al[32] in patients who had an anterior myocardial infarction, and by Goffinet et al[33] in patients with various heart diseases. It is not surprising that in our study LVtor × LVε presented a higher value of correlation coefficient compared to each component alone. Preserved LVtw appears to contribute to the normal EF in patients with diastolic heart failure,[6] whereas impaired torsional and longitudinal deformations alter LV ejection fraction in patients with DCM.

Limitations

Our results should be considered in the context of sev-

eral limitations. The number of patients in this study was relatively small; however, we were able to reach several significant observations. The success of LVtw, LVtor and LVε measurements by STE was dependent on the quality of 2D echocardiographic images. The feasibility of STE-derived LV parameters was 82%. This value is comparable to figures previously reported.[3,25,34] The torsion measurement is a time-consuming method and LV longitudinal length used for the determination of LVtor might not completely correspond to the true LV long axis length between basal and apical planes measured by echocardiography. This issue would probably be resolved with the use of 3D STE. The 3D STE approach should also simplify the calculation of our combined index. Patients were receiving their usual treatment at the time of this study, so confounding effects of drug treatment cannot be excluded. All patients analysed were on appropriate medical therapy; therefore, our results must be viewed with caution in patients with DCM before drug treatment. Our preliminary data should be confirmed in larger studies with carefully matched control groups.

Conclusions

This study demonstrates that, in patients with DCM in sinus rhythm, the evaluation of LV function can be accurately accomplished by combining LVtor with LVε. This novel STE combined parameter (LVtor × LVε) correlates strongly with plasma NTproBNP. Moreover, the combined index is reliable, particularly in patients with reversed systolic apical rotation, and is linearly related to biplane LV ejection fraction.

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