CLINICAL STUDY

Relationship Between Myocardial Perfusion and Myocardial Function in Dilated Cardiomyopathy by Shown Ultrasonography

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Summary

Myocardial contrast echocardiography (MCE) and two-dimensional speckle tracking echocardiography (2D-STE) were used to detect left ventricular myocardial microcirculation perfusion and myocardial systolic function in dilated cardiomyopathy (DCM) and to explore the relationship between the two.

Conventional ultrasound, MCE, and 2D-STE examinations were performed on 30 patients and 30 controls. Left ventricular microcirculation perfusion, left ventricular longitudinal strain (GLS), and circumferential strain (GCS) were analyzed to further compare the correlation between left ventricular perfusion and myocardial strain parameters.

Regional myocardial perfusion was reduced in patients with DCM, manifesting as a decrease in the rising slope (A) of the mid-segment of the posterior septum, the peak intensity (PI) of the mid-segment of the anterior septum and the posterior septum, the apical segment of the lateral wall, the area under the curve (AUC) of the posterior septum, the basal segment of the posterior wall, the anterior septum, posterior septum, posterior wall, mid-segment of the lateral wall, and apical segment of the lateral wall and the overall average PI and AUC of the mid-segment, compared with that in the controls (P < 0.05). The left ventricular systolic function and the strain parameters GLS and GCS of DCM patients were lower than those of the controls (P < 0.001). Correlation analysis revealed a positive correlation between the A of the mitral valve and GCS (r = 0.372, P = 0.043), and MV-E/e‘ had a positive correlation with the AUC of the basal and intermediate segments (r = 0.379, P = 0.039; r = 0.404, P = 0.027).

In patients with DCM, regional myocardial microcirculation perfusion is reduced, and myocardial strain is impaired. Myocardial perfusion has a good positive correlation with myocardial mechanics.

Key words: Myocardial microcirculation, Myocardial strain, Myocardial contrast echocardiography, Speckle tracking imaging technology

Dilated cardiomyopathy (DCM) is a primary heart muscle disease caused by myocardial dysfunction in the absence of other abnormal loading conditions (hypertension, valvular disease, etc.) or coronary artery disease (CAD). The main symptoms include left ventricular dilatation and systolic dysfunction. Congestive heart failure may occur in the later stage, with a high mortality rate. It is well known that CAD is excluded in patients with DCM and that myocardial ischemia is generally assumed to not occur during the progression of DCM, which is also confirmed by normal coronary angiography results. However, in recent years, changes in coronary microcirculatory blood flow, especially a disturbance in the microcirculation caused by coronary endothelial dysfunction, are considered an important reason for myocardial dysfunction. Moreover, several studies have also shown that abnormal myocardial perfusion in patients with DCM is accompanied by moderate or severe adverse myocardial remodeling, that positron emission tomography and invasive angiographic assessment have demonstrated impaired myocardial perfusion reserve (MPR) and coronary flow reserve, and that endothelial dysfunction and myocardial systolic dysfunction are also associated with poor prognosis. All the above studies have confirmed that in the absence of obstructive epicardial CAD, myocardial microvascular dysfunction in patients with DCM provides support for a microvascular ischemia hypothesis. This ischemia hypothesis postulates that chronic and/or repetitive myocardial hypoperfusion resulting from microvascular dysfunction may drive progressive left ventricular (LV) systolic dysfunction and dilatation, which in turn may affect microvascular function in a vicious cycle.
Two-dimensional speckle tracking echocardiography (2D-STE) is a powerful new method for assessing subclinical heart damage. In recent years, this quantitative ultrasound technology has developed rapidly due to its unique advantages in the objective evaluation of myocardial function. Myocardial contrast echocardiography (MCE) can reveal the degree of regional myocardial perfusion, improve understanding of the myocardial blood flow reserve, and judge whether the myocardium has collateral circulation. Noninvasive detection of myocardial microcirculatory blood flow is of great significance in the early clinical evaluation of patients with DCM and is also beneficial for early treatment to slow down or reverse the disease process. Although some scholars have discussed myocardial perfusion and myocardial mechanical changes in DCM, few studies have explored the relationship between the two. The relationship between abnormal myocardial perfusion and myocardial mechanical changes in patients with DCM has not been fully elucidated. This case-control study aims to combine two ultrasound techniques, namely, MCE and 2D-STE, to evaluate myocardial microcirculatory perfusion and LV myocardial systolic function in patients with DCM and to explore the relationship between the two.

**Methods**

**Study population:** This study included 30 patients who presented to the Heart Center of the First Affiliated Hospital of Xinjiang Medical University and eventually underwent MCE between October 2018 and May 2020 due to a DCM diagnosis. Diagnostic criteria for DCM were LV end-diastolic diameter (LVEDD) > 50 mm (female) and > 55 mm (male) or left ventricular end-diastolic diameter (LVEDD) more than 117%, predicted value corrected for age and body surface and left ventricular systolic dysfunction defined by left ventricular ejection fraction (LVEF) less than 45% (Simpson’s method) and/or fractional shortening (FS) less than 25%. The inclusion criteria for patients with DCM were as follows: patients who had undergone medical history collection, electrocardiogram (ECG) examination, general physical examination, and transthoracic echocardiography, those with secondary heart enlargement not caused by abnormal cardiac load, those with coronary angiography/CT angiography showing no CAD, those with no history of previous myocardial infarction or coronary revascularization, and those with no segmental wall motion abnormalities in cardiac ultrasound. Patients with diabetes, hypertensive heart disease, significant primary valvular heart disease, and probable secondary causes for secondary DCM were excluded. Healthy volunteers of similar age distributions, without cardiovascular disease, other organic diseases, normal cardiac ultrasound and ECG, and negative coronary angiography were recruited as control subjects (n = 30). This study was approved by the local ethics committee, and a written informed consent was provided by all patients and control subjects.

**Instrument and image acquisition method:** For all exams, a commercial ultrasound machine (Epiq 7; Philips Medical System, Andover, MA, United States) equipped with a 3.5 MHz phased array probe was used. The patients were placed in the lateral recumbent position, instructed to breathe calmly, and connected to a synchronous epicardial ECG lead. Heart size and function were measured by conventional ultrasound, which was then switched to the large vessel occlusion imaging pattern (TIS = 0.7, mechanical index (MI) = 1.4). According to the imaging situation, contrast media was injected (Sonovue, Bracco, Italy; a total of 59 mg of Sonovue was diluted in 5 mL of saline and shaken well before injection to form a milky white suspension), and dynamic images were observed and retained. The dynamic images of the LV long axial view, apical four-chamber view, and apical two-chamber view were retained from 5 cardiac cycles before the flash pattern to 15 cardiac cycles after the flash mode. Every time the “flash” flickered, the microbubbles were shattered instantly with high MI to clear microbubbles from the myocardial microcirculation, and myocardial reperfusion was observed. 2D-STE recorded the dynamic images of at least three cardiac cycle views of the LV long axis, apical four-chamber views, apical two-chamber views, LV short-axis mitral valve, papillary muscle, and apex. The image acquisition was stored in the optical disk for offline analysis.

**Image analysis:**

**MCE** Using QLAB10.8 analysis software, the regions of interest (ROIs) were placed on the LV myocardium, and manual correction of ROI placement was required, with optimization of ROI size to include as many myocardial segments as possible. Meanwhile, low-intensity signals in the pericardium or high-intensity signals in the LV cavity were avoided. The sampling frames were manually realigned frame by frame starting from the minimum intensity of the image after “flash” to keep the same anatomical position in each frame of the cardiac cycle of contrast agent refilling and dynamically tracing the blood flow changes. The software calculated the signal intensity in selected ROIs and fitted the appropriate myocardial acoustical signal intensity corresponding to the time curve in the sampling frame. Thus, a series of parameters could be estimated based on the gamma fitting formula y(t) = A*(t−t0)*exp(−α*(t−t0)) + C, such as the slope of ascending (A), time to peak (TTP), peak intensity (PI), and area under the curve (AUC). Among the related parameters extracted from TIC, the rising slope (A) reflects the change of microbubble flow velocity and flow rate with time during contrast-enhanced ultrasound in ROI. TTP refers to the time from the beginning of contrast agent into ROI to the time of enhancement to maximum intensity. PI is directly proportional to the mean blood volume of ROI, which can reflect the blood flow of local tissues roughly and is well correlated with the perfusion volume of tissues. AUC is proportional to the average blood flow in ROI, which can reflect the change of blood flow volume in blood vessels, while A × PI reflects the myocardial blood flow, namely AUC. The abnormal section of the area under the curve includes the abnormal section of the ascending slope and PI (Figure 1A, B).

**2D-STE** Two-dimensional echocardiographic images for the LV were analyzed from the long axis of the left ventricle, four apical chamber view, two apical chamber view,
mitral valve of the short axis of the left ventricle, papillary muscle, and apical section into the CMQ interface for quantitative analysis of cardiac movement, sketching the endocardium, manually fine-tuning the region of interest, obtaining the strain versus time curve and peak strain of each segment of the left ventricle, and generating the overall longitudinal strain of the left ventricle. The endocardial border or region of interest was readjusted to obtain each myocardial segment of the strain versus time curve, generating a series of parameters, such as global longitudinal strain (GLS) and global circumferential strain (GCS) (Figure 1C-F).

Conventional ultrasonic measurement These parameters, which are obtained by conventional ultrasound, included left atrial volume, left atrial volume index (LAVI), left ventricular mass (LVM), left ventricular mass indexed to body surface area (LVM/BSA, LVMI), LVEF, left ventricular fractional shortening, left ventricular end-diastolic volume, left ventricular end-systolic volume, stroke volume (SV), left ventricular internal diameter during diastole (LVIDD), and relative wall thickness (RWT). The RWT was measured using the formula 2 * LVPWD/LVIDD. 2D imaging of the LV was performed from the parasternal long axis, parasternal short axis (basal, mid, and apical), apical four-chamber, apical two-chamber, and apical long-axis views. Transmitral flow velocities (E and A) were obtained using pulsed-wave Doppler in the apical four-chamber view. The E/A velocity ratio was measured. Tissue Doppler imaging was used to measure mitral annular velocities. The early diastolic velocity (E’) was meas-
Table I. General Information Features of the Studied Patients (X ± s)

| Variables               | DCM (n = 30)     | Control (n = 30)   | P     |
|-------------------------|------------------|-------------------|-------|
| Age (years)             | 45.6 ± 11.80     | 44.1 ± 13.86      | 0.46  |
| Gender (n)              |                  |                   | 0.646 |
| Male                    | 23               | 13                | 6.944 |
| Female                  | 7                | 17                | 0.008 |
| BSA (m²)                | 1.89 ± 0.22      | 1.82 ± 0.19       | 0.232 |
| Weight (kg)             | 80.57 ± 12.55    | 70.27 ± 12.47     | 0.232 |
| Systolic blood pressure (mmHg) | 128.47 ± 18.77  | 117.83 ± 10.94    | 0.010 |
| Diastolic blood pressure (mmHg) | 85.47 ± 14.66  | 71.63 ± 7.82      | < 0.001 |
| BNP (ng/L)              | 1737.4 ± 2826.28 | 166.47 ± 193.91   | 3.005 |

Data are shown as mean ± SD or number. BSA indicates body surface area; and BNP, brain natriuretic peptide.

Table II. Measures of Conventional and Tissue Doppler (X ± s)

| Variables               | DCM (n = 30)     | Control (n = 30)   | t     | P     |
|-------------------------|------------------|-------------------|-------|-------|
| LVM (g/m²)              | 179.13 ± 44.81   | 99.81 ± 15.06     | 9.191 | < 0.001 |
| LAVI (mL/m²)            | 39.35 ± 13.01    | 23.96 ± 6.31      | 5.832 | < 0.001 |
| RWT (mm)                | 0.29 ± 0.07      | 0.38 ± 0.04       | −5.934| < 0.001 |
| MV-E (cm/s)             | 0.77 ± 0.17      | 0.90 ± 0.13       | −3.256| 0.002  |
| MV-A (cm/s)             | 0.59 ± 0.25      | 0.75 ± 0.15       | −3.001| 0.004  |
| MV-E/A                  | 1.71 ± 1.12      | 1.24 ± 0.26       | 2.245 | 0.032  |
| MV-E/e'                 | 13.23 ± 6.16     | 8.46 ± 1.63       | 4.097 | < 0.001 |
| LVEF (%)                | 41.48 ± 14.62    | 61.43 ± 8.64      | −6.434| < 0.001 |
| LVFS (%)                | 21.03 ± 8.91     | 33.27 ± 2.75      | −7.184| < 0.001 |
| LVEDV (mL)              | 234.26 ± 63.69   | 103.17 ± 21.80    | 10.665| < 0.001 |
| LVESV (mL)              | 140.88 ± 60.03   | 38.90 ± 7.48      | 9.234 | < 0.001 |
| SV (mL)                 | 93.38 ± 33.29    | 64.27 ± 16.66     | 4.282 | < 0.001 |

Data are shown and mean ± SD. LV indicates left ventricular; LVM, left ventricular mass; LAVI, left ventricular mass index; LAVI, left atrium volume; LAVI, left atrium volume index; RWT, relative wall thickness; E, early transmitral velocity; A, late transmitral velocity; LVEF, left ventricular ejection fraction; LVFS, left ventricular fractional shortening; SV, stroke volume; LVEDV, left ventricular end-diastolic volume; and LVESV, left ventricular end-systolic volume.

Results

General information features: Table I shows the basic clinical characteristics of the groups, including 23 males and 7 females in the DCM group, with an average age of 45.67 ± 11.80 years, and 13 males and 17 females in the control group, with an average age of 44.13 ± 13.86 years. Age and body surface area did not differ significantly between the groups (P > 0.05), but patients with DCM had higher weight, systolic blood pressure, diastolic blood pressure, and brain natriuretic peptide than the control group (P < 0.05).

Conventional ultrasonic parameters: Conventional ultrasound was feasible for inspecting changes in LV function in all subjects. As shown in Table II, compared with the control group, LVM, LAVI, EDV, ESV, and SV reflecting LVEF in the DCM group increased, while RWT, EF, and FS decreased. Moreover, the standard indices of diastolic function, including MV-E and MV-A, decreased, while MV-E/A and MV-E/e' increased compared with those in the control group; these differences reached statistical significance (P < 0.05).

Myocardial perfusion parameters: Quantitative MCE was feasible in all patients. By comparing the myocardial microcirculation perfusion parameters among the groups, it was found that myocardial perfusion parameters were reduced, including the rising slope (A) of the mid-segment of the posterior septum, the PI of the mid-segment of the anterior septum and the posterior septum, the apical segment of the lateral wall, the AUC of the posterior septum, the basal segment of the posterior wall, and the anterior septum, posterior septum, posterior wall, and...
Correlation between perfusion and strain: Based on the two groups (and the absolute value of GCS in the observation group versus −7.62 ± 3.03% versus −26.20 ± 2.36%), statistically significant differences were not found in other variables (r = 0.039; P = 0.027), and other values of segments and MV-E/e′ were not correlated (P > 0.05) (Table IV).

**Table III.** Comparison of Myocardial Perfusion Between the Two Groups of 17 Segments (X ± s)

| Variables | Base | Control | Base | Control | Apex | Control |
|-----------|------|---------|------|---------|------|---------|
| A (dB/second) | | | | | | |
| A &gm; ± 13.90 | 7.47 ± 7.88 | 5.03 ± 7.35 | 5.22 ± 4.55 | 4.45 ± 5.58 | 8.12 ± 9.27 |
| AS | 9.96 ± 10.67 | 8.11 ± 7.56 | 7.53 ± 9.00 | 9.73 ± 10.55 |
| PS | 15.51 ± 22.38 | 10.49 ± 11.10 | 8.93 ± 12.35 | 19.94 ± 23.06 | 9.76 ± 15.33 | 5.90 ± 5.71 |
| I | 11.16 ± 21.27 | 9.07 ± 15.69 | 12.61 ± 19.71 | 8.67 ± 10.62 | 7.48 ± 9.37 | 7.12 ± 5.59 |
| P | 7.23 ± 6.98 | 11.09 ± 12.87 | 8.38 ± 10.83 | 15.13 ± 15.38 |
| L | 7.94 ± 12.35 | 8.51 ± 9.61 | 7.71 ± 9.62 | 7.05 ± 11.48 | 12.22 ± 19.14 | 13.44 ± 16.66 |
| Apex | 15.21 ± 27.08 | 9.00 ± 9.82 |
| TTP (seconds) | | | | | | |
| A | 1.46 ± 0.86 | 1.46 ± 0.80 | 1.59 ± 0.72 | 1.58 ± 0.83 | 1.63 ± 0.59 | 1.60 ± 0.84 |
| AS | 1.39 ± 0.70 | 1.70 ± 0.79 | 1.46 ± 0.70 | 1.64 ± 0.79 |
| PS | 1.34 ± 0.90 | 1.59 ± 0.77 | 1.53 ± 0.59 | 1.37 ± 0.73 | 1.43 ± 0.66 | 1.69 ± 0.82 |
| I | 1.49 ± 0.75 | 1.68 ± 0.84 | 1.42 ± 0.75 | 1.50 ± 0.74 | 1.67 ± 0.62 | 1.57 ± 0.75 |
| P | 1.57 ± 0.63 | 1.57 ± 0.85 | 1.66 ± 0.79 | 1.36 ± 0.84 |
| L | 1.60 ± 0.83 | 1.55 ± 0.71 | 1.43 ± 0.78 | 1.73 ± 0.77 | 1.47 ± 0.65 | 1.49 ± 0.77 |
| Apex | 1.39 ± 0.81 | 1.59 ± 0.76 |
| PI (dB) | | | | | | |
| A | 17.77 ± 7.06 | 18.93 ± 5.07 | 16.76 ± 6.40 | 18.50 ± 5.83 | 19.19 ± 6.54 | 20.22 ± 8.00 |
| AS | 22.30 ± 7.03 | 23.21 ± 8.29 | 20.54 ± 6.43 | 25.26 ± 8.00 |
| PS | 26.45 ± 6.01 | 29.67 ± 6.51 | 24.71 ± 6.19 | 29.82 ± 5.53 | 20.06 ± 5.59 | 21.03 ± 7.23 |
| I | 22.71 ± 6.35 | 21.45 ± 7.92 | 23.92 ± 5.78 | 20.45 ± 7.91 | 22.78 ± 6.36 | 20.46 ± 7.00 |
| P | 23.04 ± 6.48 | 25.13 ± 6.85 | 22.04 ± 5.67 | 24.96 ± 6.53 |
| L | 20.83 ± 6.56 | 22.60 ± 7.01 | 17.78 ± 4.70 | 20.41 ± 6.23 | 23.01 ± 6.82 | 26.79 ± 6.72 |
| Apex | 23.67 ± 6.26 | 25.88 ± 7.31 |
| AUC (dB-second) | | | | | | |
| A | 39.06 ± 18.21 | 40.94 ± 13.88 | 35.58 ± 14.88 | 39.65 ± 13.01 | 41.82 ± 18.49 | 42.72 ± 19.51 |
| AS | 47.43 ± 18.02 | 52.93 ± 24.02 | 44.40 ± 17.79 | 59.68 ± 26.12 |
| PS | 57.29 ± 18.03 | 68.71 ± 21.68 | 53.82 ± 17.23 | 68.47 ± 18.65 | 43.28 ± 14.45 | 49.02 ± 20.71 |
| I | 48.62 ± 15.45 | 44.32 ± 16.94 | 51.25 ± 16.19 | 45.09 ± 18.16 | 47.47 ± 17.62 | 44.91 ± 18.86 |
| P | 48.48 ± 16.06 | 60.40 ± 20.95 | 47.08 ± 15.96 | 60.25 ± 21.96 |
| L | 43.46 ± 16.23 | 48.94 ± 16.89 | 37.87 ± 13.23 | 46.02 ± 17.07 | 49.52 ± 18.25 | 62.63 ± 21.11 |
| Apex | 51.04 ± 15.54 | 58.20 ± 19.18 |

Statistical significance (P < 0.05) is indicated by triangles. A indicates ascending slope; B, time to peak; C, peak intensity; D, area under the curve; Base, basal segment; Mid, middle segment; Apex, Apical segment; A, anterior wall; AS, anterior septum; PS, posterior septum; I, inferior wall; P, posterior wall; and L, lateral wall.

mid-segment of the lateral wall. Perfusion parameters of each segment of the LV short-axis mitral valve level, papillary muscle level, and apical level were added together to obtain the overall myocardial perfusion at each level. The results showed that the apical segment of the lateral wall and the overall average PI and AUC of the mid-segment in patients with DCM were obviously decreased compared with those in the control group (P < 0.05). Statistically significant differences were not found in other segments (P > 0.05) (Table III, Figure 2).

**Strain analysis:** Quantitative 2D-STE analysis was feasible in all patients. The absolute value of GLS in the observation group had a positive relationship with parameters of GCS (r = 0.372, P = 0.043), and other values of myocardial perfusion, GLS and GCS, were not correlated (P > 0.05) (Table IV); MV-E/e′ had a positive correlation with the AUC of the basal and middle segments (r = 0.379, P = 0.039; r = 0.404, P = 0.027), and other values of segments and MV-E/e′ were not correlated (P > 0.05) (Table IV).

**Repeatability test:** In terms of the intra-observer variability, there was an excellent ICC for A (0.877; 95% CI 0.594–0.968, P < 0.001), TTP (0.845; 95% CI 0.491–0.959, P = 0.001), PI (0.960; 95% CI 0.863–0.989, P < 0.001), AUC (0.984; 95% CI 0.946–0.995, P < 0.001), LVGLS (0.997; 95% CI 0.991–0.999, P < 0.001), and GCS (0.937; 95% CI 0.828–0.978, P < 0.001). Similar, although slightly worse, concordance values were found for the interobserver evaluation: A (0.806; 95% CI 0.382–0.948, P = 0.002), TTP (0.828; 95% CI 0.445–0.954, P = 0.001), PI (0.956; 95% CI 0.835–0.989, P < 0.001), AUC (0.976; 95% CI 0.912–0.994, P < 0.001), LVGLS (0.984; 95% CI 0.952–0.994, P < 0.001), and LVGCS (0.897; 95% CI 0.699–0.965, P < 0.001) (Table V).
DCM is a significant cause of mortality and heart transplant worldwide and is principally driven by pump failure and sudden cardiac death, seriously affecting the quality of life and health of patients. Severe coronary artery endothelial dysfunction affects the regulation of myocardial blood perfusion in patients with DCM, and some scholars maintain that a myocardial oxygen deficit caused by microvascular dysfunction may contribute to deterioration of LV systolic and diastolic function in DCM. In addition, endothelial dysfunction and myocardial dysfunction have also been associated with an adverse prognosis. Therefore, a deeper understanding of myocardial microcirculation and cardiac motor function in patients with DCM contributes to early clinical diagnosis and treatment as well as an evaluation of the effect on treatment to improve clinical symptoms of patients and reduce the hospitalization and fatality rates.

Some studies have found that the pattern of heterogeneity varied widely among the regional LV function of patients and reduced function in the inferior, septal, or ante-
Due to the increase in wall tension, the LV cavity of DCM patients expands, and myocardial fibers are passively elongated, displaced, and thinned. These changes affect the original movement and deformability of the myocardium, resulting in overall LV dysfunction. In this study, conventional ultrasound tests showed that the overall cardiac function in the DCM group was damaged, and myocardial strain changes were further detected, showing that LV longitudinal strain and circumferential strain decreased, which indicated that myocardial movement in the longitudinal and circular directions was impaired and compliance was decreased. Decreased myocardial deformability leads to different degrees of damage to LV diastolic and systolic function, and myocardial hypokinesia is the cornerstone of cardiac pump dysfunction. GLS integrates the global long-axis strain of all myocardial segments and may be more sensitive in reflecting the early damage to left ventricular segmental systolic function than LVEF, which is thought to be more sensitive to subendocardial ischemia. In addition, it was found that the GLS of patients with improved LVEF after treatment was less than 16%, still indicating that the patients might face long-term left ventricular dysfunction. GLS is a good indicator of early cardiac damage, an adverse prognosis, and therapeutic effect monitoring. Our results were consistent with those of other studies, suggesting that myocardial mechanics are an important feature of DCM.

In recent years, studies have found that myocardial blood flow impairment is multifactorial in nature and may be caused by myocardial structural abnormalities, effects of increased hemodynamic load on the coronary microvas-
circular bed, and structural alteration of the coronary microvasculature itself. Depressed coronary blood flow reserve measured by left cardiac catheterization also confirms this view. Previous studies have shown that reduced myocardial perfusion in nonischemic cardiomyopathy is usually associated with overall eccentric left ventricular hypertrophy, enlargement of the left ventricular chamber, and reduced LVEF. GLS and GCS are mainly related to subendocardial myocardial contraction. This study found that GCS was positively correlated with the rising slope of the basal segment, which confirmed the correlation between myocardial microcirculation perfusion and myocardial mechanical changes. Some factors could explain why GCS alone was independently associated with event occurrence in our study. First, in agreement with the studies of Schuster and Claus, et al., GCS was considered the most robust parameter in studies of myocardial strain, which was not affected by poor tracking of the subannular region, unlike GLS. In addition, our population had more advanced disease at diagnosis, reflected by the lower LVEF values and worse myocardial strain parameters. In this state, systolic function predominately depends on the circumferential and radial contraction, and its alteration could influence prognosis. We speculated that the degree of myocardial microcirculation damage might reflect the degree of myocardial systolic function damage, that is, the more seriously impaired the myocardial microcirculation was, the more serious the regional myocardial dysfunction, and the worse the clinical prognosis of patients. In fact, the left ventricle of DCM patients is spherically dilated and exhibits an increase in wall tension, affecting myocardial perfusion, especially subendocardial involvement of the myocardium, which leads to myocardial ischemia, necrosis, and fibrosis and to a reduction in LV myocardial contractility (such as longitudinal, radial, and circumferential contractility). At the same time, LV diastolic impairment leads to decreased coronary blood supply and further aggravates myocardial microcirculation perfusion injury. In conclusion, severely damaged endocardial myocardial structures and myocardial microcirculation disorders lead to LV dysfunction.

Limitations: In this study, the changes in myocardial perfusion and myocardial strain in patients with DCM were investigated by grasping the inclusion and exclusion criteria, and the problems associated with hypertension and diabetes (a common clinical problem) that might affect the study results were eliminated. However, some limitations still exist in the study. First, the two-dimensional speckle tracking technique can provide complex cardiac spatial motion information, but this technology inevitably has some problems, such as manually sketching out the endocardium and automatically tracing speckle signals through software. Although strict quality control was carried out in image retention and data reading, there may still be some measurement errors. Moreover, the lateral resolution of tissue Doppler-based strain at the base of the heart is limited, as is the limited feasibility of measuring radial strain. Finally, the sample size of this study was relatively small, and no loading test was performed to evaluate the reserve capacity of coronary arteries. If we want to comprehensively and objectively evaluate myocardial perfusion and myocardial function in DCM patients, we still need to expand the sample size in the future.

Conclusion

MCE and 2D-STI were used to obtain LV myocardial perfusion and strain-related indicators in DCM patients and to explore the relationship between LV microcirculation and changes in myocardial systolic function in this study. The regional myocardial tissue blood flow and tissue perfusion in DCM patients were reduced, and the papillary muscle level was significantly affected. At the same time, ventricular compliance in DCM was impaired, and the longitudinal contractility and circumferential contractility were reduced. The myocardial microcirculation was consistent with changes in myocardial mechanics, and there was a positive correlation between the two. Decreased myocardial microcirculation perfusion may be the cause of myocardial fibrosis and myocardial systolic dysfunction. In addition, myocardial microcirculation perfusion may be an independent prognostic factor that can predict LVEF recovery and the occurrence of major cardiovascular events. Thus, we can combine these two techniques to more objectively and accurately detect cardiac disorders at a subclinical level, improve risk stratification of patients with various cardiac conditions, and potentially monitor treatment effect.

Disclosure

Conflicts of interest: The authors declare that there is no conflict of interest.

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