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Left Ventricular Torsional Parameters in Patients With Non-Ischemic Dilated Cardiomyopathy

Zahra Ojaghi Haghighi [1]; Azin Alizadehasl [1,*]; Hassan Moladoust [2]; Maryam Ardeshiri [3]; Atousa Mostafavi [4]; Nahid Rezaeiyan [1]; Seyed Hossein Ojaghi [5]; Fazlolah Safi [6]; Akram Mikaeilpour [5]

[1]Department of Cardiovascular Medicine, Echocardiography Research Center, Rajaie Cardiovascular, Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran
[2]Cardiovascular Research Center, Heshmat Hospital, School of Medicine, Guilan University of Medical Sciences, Rasht, IR Iran
[3]Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran
[4]Shariati Hospital, Tehran University of Medical Sciences, Tehran, IR Iran
[5]Imam Reza Hospital, Tabriz University of Medical Sciences, Tabriz, IR Iran
[6]Bahman Hospital, Tehran, IR Iran

*Corresponding author: Azin Alizadehasl, Department of Cardiovascular Medicine, Rajaie Cardiovascular, Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran. Tel: +98-2123922190, Fax: +98-2122663293, E-mail: alizadeasl@gmail.com

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Background: Velocity vector imaging (VVI) is a new echocardiography method to assess myocardial deformation in two dimensions.
Objectives: In this study, we used VVI to evaluate left ventricular (LV) main torsional parameters in non-ischemic dilated cardiomyopathy (DCM) patients in compared with normal subjects.
Patients and Methods: Twenty-six DCM patients and Twenty-four normal subjects were assessed. Echocardiographic images of the short axis apical and basal views of LV were processed by VVI software to measure peak rotation degrees and also peak rotation rates in systole. LV twist was well-defined as the net difference between apical and basal rotation values and also LV torsion was considered as LV twist divided by left ventricular diastolic longitudinal length. In addition, peak untwisting value and untwisting rate were measured in diastole too.
Results: LV twist value (5.54 ± 1.94° in DCM VS. 11.5 ± 2.45° in control group) and also LV torsion (0.71 ± 0.28°/cm in DCM VS. 1.53 ± 0.42°/cm in control group) were significantly decreased in DCM patients compared with normal group ($P < 0.001$ for both); also, the twisting rate was notably lower in DCM vs. control (38.68 ± 14.43°/s in DCM vs. 75.88 ± 17.25°/s in control; $P < 0.001$) and also untwisting rate (36.28 ± 13.48°/s in DCM vs. -73.79 ± 24.45°/s in control; $P < 0.001$), However normalization of these times for systolic duration or LV length creates different values.
Conclusions: LV twist, torsion and untwist and also rate of them are significantly impaired in DCM and this impairment is well-related to LV global systolic and diastolic dysfunction. VVI is a new noninvasive technique that can be used to evaluate LV torsional parameters.

Keywords: Cardiomyopathy, Dilated; Echocardiography; Torsional Parameters; Non-ischemic Dilated Cardiomyopathy

1. Background

Special motion of the Left ventricular (LV) is due to helical arrangement of its myofibrils, with a right handed orientation from base to apex in the endocardial layer and a left handed orientation in the epicardial layer. This spiral arrangement leads to the left ventricular wringing motion (1, 2) When we look at the LV from the apex, systolic rotation of the base is clockwise, whereas the apex tends to rotate in a counterclockwise manner.

The LV's myofibrilar geometry smoothly transforms from a marginally oblique orientation in the sub-endocardium to a circumferential orientation in the mid wall and again to an oblique orientation in the sub-epicardium. It has been postulated that this is the main mechanism for the special LV movement pattern. The LV torsion allows a uniform distribution of stress on the fibers and fiber shortening across the wall: this represents an extremely important mechanism for both ejection and filling (3, 4).

LV twist is estimated by calculating the maximal difference in rotation between the apical and basal levels in the short axis plane. Torsion is considered by dividing twist by the longitudinal length between the two recorded short axis levels. LV untwist refers to the amount of twist that happens during diastole (5, 6) Echocardiography with tissue tracking and MRI with tissue tagging are the most frequently performed techniques for assessing these parameters (7, 8). Although it is considered more precise, MRI requires more time for acquisition, has less temporal resolution, and is less accessible in most clinical environments.

2. Objectives

Velocity vector imaging (VVI) is a novel echocardiography technique to assess myocardial motion in two dimensions which incorporates speckle and endocardial border tracking and permits myocardial strains, strain rates, and velocities to be measured. The aim of our study was to compare various LV torsional parameters in systole and diastole as determined by VVI between patients with dilated cardiomyopathy (DCM) versus healthy subjects.

3. Patients and Methods

Twenty-four healthy and twenty-six non-ischemic DCM men and women (32 ± 6 and 35 ± 4 years old respectively) were included in the present cross-sectional study. All patients underwent complete echocardiographic study, and LV torsional parameters were assessed by VVI method. All DCM patients had severe LV enlargement, pressured tissue and LV ejection fraction (LVEF) $\leq 30\%$ by transthoracic echocardiography.

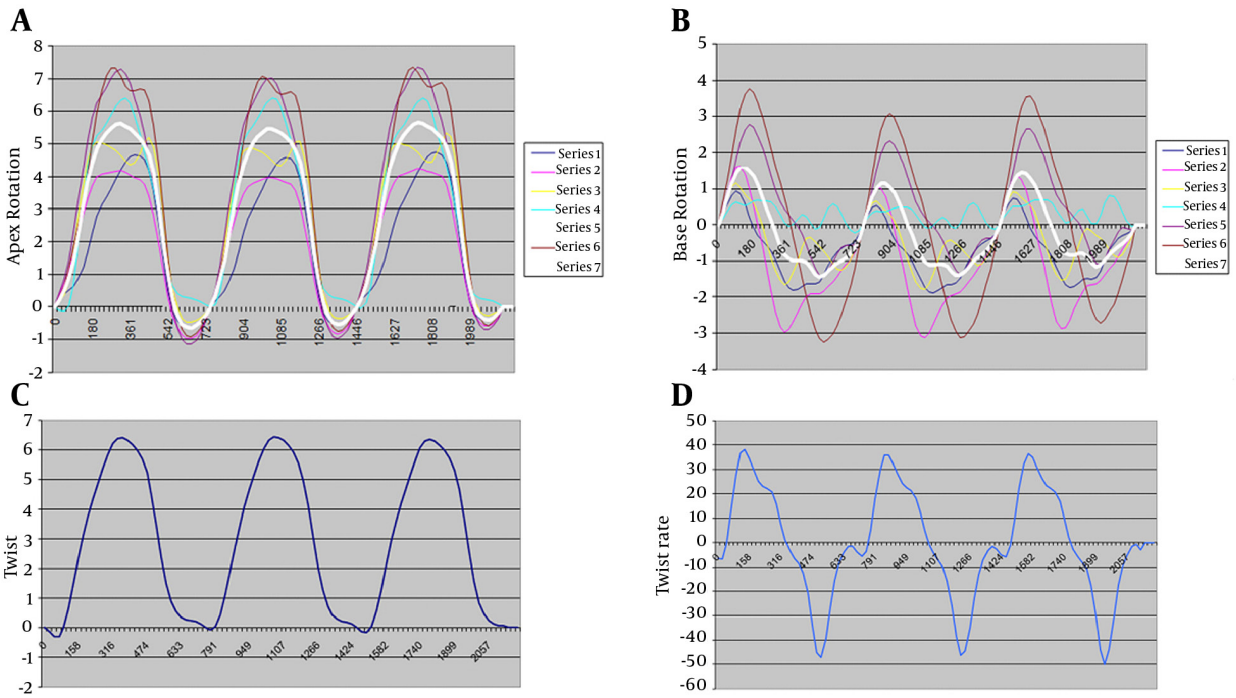
3.1. Echocardiography

Transthoracic echocardiographic study was done by MyLab60 (ESAOTE, Florence, Italy) with VVI method. Two-dimensional ECG was superimposed on the images, and end-diastole was considered at the peak R-wave of the ECG. The LV global systolic function was evaluated using a modified biplane Simpson method for calculating the LVEF by measuring end-diastolic and end-systolic volumes in the 2D images.

3.2. Velocity Vector Imaging (VVI)

We used MyLab60 (ESAOTE, Florence, Italy) which uses a VVI tracking algorithm and provides angle independent 2-dimensional velocity, strain, strain rate, and twist-related values. Endocardial borders were visually recognized and manually traced according to the manufacturer's instructions. Endocardial border tracking over one frame is then automatically performed throughout the cardiac cycle. Velocity vectors, which instantaneously represent velocity magnitude and direction, were presented throughout the cardiac cycle. Data points depicting the basal and apical LV rotation and rotational velocities were exported to Excel (Microsoft Corporation, Red-mond, WA) to calculate LV twists and twist velocities. The LV cross-section was made as circular as possible. For example, Figure 1 A, 1B, 1C, 1D show Average Basal Rotation (White line), Average Apical Rotation (White line), LV Twist and LV twist rate. Various LV torsional parameters such as peak systolic twist, peak twisting velocity, and peak untwisting velocity were measured as demonstrated. In addition, the normalized LV torsional parameters such as LV torsion, normalized peak twisting rate, normalized untwisting rate, and normalized time to peak untwisting velocity were measured by the VVI data.

Figure 1. LV Torsional Parameters (VVI Method)



A, LV apex rotation; B, LV base rotation; C, LV twist; D, LV twist rate.

3.3. Statistical Analysis

All the continuous variables are presented as mean (SD). The normal distribution was tested using the Kolmogorov-Smirnov (K-S) test. To determine whether the difference in the values between the two groups was statistically significant, an independent samples t-test was performed. A $P \leq 0.05$ was considered statistically significant. All the statistical analyses were performed using the SPSS version 18.0 software package (SPSS Inc. Chicago, IL, USA). Also the study protocol was approved by institutional ethic committee. Written informed consent was obtained from all of the patients.

4. Results

4.1. Clinical and Echocardiographic Characteristic

The clinical characteristics data of the two groups are summarized in Table 1.

4.2. Twist and Torsion Results

The twist degree was significantly lower in the DCM group versus control group (5.54 ± 1.94 vs. 11.05 ± 2.45°; P = 0.000). The LV torsion was significantly lower in the DCM group than normal group (0.71 ± 0.28°/cm vs. 1.53 ± 0.42°/cm; P = 0.000).

4.3. Twisting Rate and Untwisting Rate Results

The twisting rate was notably lower in DCM vs. control (38.68 ±14.43°/s in DCM vs. 75.88 ± 17.25°/s in control; P = 0.000) and also untwisting rate (-36.28 ± 13.48°/s in DCM vs. -73.79 ± 24.45°/s in control; P = 0.000) , but when the untwisting rate was normalized by the systolic duration (t = 100% at end systole), there was no significant difference between the two groups (134.05 ± 26.92% in DCM vs. 136.77 ± 21.3% in control; P = 0.701.

Similarly, when the untwisting rate was normalized by the LV length, there was a significant decline in the normalized peak untwisting rate in the DCM group (- 4.59 ± 1.85°/s/cm vs. - 10.08 ± 3.33°/s/cm; P = 0.000), also the peak twisting rate normalized by the LV length was predominantly lower in DCM group (4.89 ± 1.95°/s/cm vs. 10.53 ± 3.12°/s/cm; P = 0.000). Finally the peak untwisting rate normalized by the LV torsion was not statistically different between the two groups (-6.78 ± 2.08 1/s vs. -6.77 ± 2.31 1/s; P = 0.993) (Table 2).

Table 1. Demographic and Hemodynamic Characteristics of the Study Participants [a,b]

Variable	Control (n = 24)	DCM Group (n = 26)	P Value
Male/female [c]	11/13	11/15	0.924
Body surface area, m^2	1.82 ± 0.3	1.83 ± 0.1	> 0.99
Age, y	32 ± 6	35 ± 4	0.750
Heart rate, beats/min	82 ± 12	78 ± 15	0.321
SBP, mmHg	124 ± 12.5	118 ± 13.2	0.631
DBP, mmHg	76 ± 12.9	72 ± 10.3	0.44
LVEF, %	60 (5)	17.5 (10)	< 0.001
LVEDD, mm	46 ± 7	70 ± 10	0.01
LVESD, mm	30 ± 5	55 ± 10	0.001

[a] Abbreviations: DBP, Diastolic Blood Pressure; DCM, dilated cardiomyopathy; LVEDD, Left Ventricular End Diastolic Diameter; LVEF, Left Ventricular Ejection Fraction; LVESD, Left Ventricular End Systolic Diameter; SB, Systolic Blood Pressure.
[b] Data are presented as Mean (SD) No. (%).
[c] Data are presented as No.

Table 2. Torsional Parameters in Two Groups [a,b]

Variables	Control Group	DCM Group	P value
LV apical rotation	10 ± 3.2	4 ± 2.6	< 0.001
LV basal rotation	-4 ± 2.5	-2 ± 1.5	0.01
LV twist	11.05 ± 2.45	5.54 ± 1.94	< 0.001
LV torsion	1.53 ± 0.42	0.71 ± 28	< 0.001
LV twisting rate	75.88 ± 17.25	38.68 ± 14.43	< 0.001
LV untwisting rate	-73.79 ± 24.45	-36.28 ± 13.48	< 0.001

[a] Abbreviations: DCM, dilated cardiomyopathy; LV, left ventricule.
[b] Units; Rotation: degree, Twist: degree, Torsion: degree/cm, Twisting rate: degree/second, Untwisting rate: degree/second.

5. Discussion

Our main and broad result in this study indicated the significant decline in all torsional parameters of LV in DCM patients; so to explain the reason of these data, we illustrate some mechanisms and previous studies in concomitant with our results.

As mentioned before, the LV twist is considered as the net difference in the LV rotation at isochoronal time points between the apical and basal short axis planes, it means that there is gradient between apex and base in the rotation along left ventricular longitudinal axis (9) The short-axis views at the apical and basal levels are recorded immediately with identical or very similar HR for the measurement of the apical and basal rotations (8-10). LV torsion is a measurement derived from the twisting or wringing motion of the heart along its long axis, it plays an important role in squeezing the blood out of the heart (11) this calculation is made by measuring the amount of rotation at the apex and subtracting the amount of basal rotation (12).

Several techniques had been applied for study of LV twist and torsion with tagged cardiac magnetic resonance imaging (CMR) is considered the gold standard (13), but the most recent VVI technique used in the present study.

Tissue Doppler imaging method (TDI) is derived from velocity data with higher temporal resolution but with intrinsic angle dependency and STE derived from frame by frame tracking of speckles created by the interference of ultrasound beams within tissue. VVI is an advance echocardiographic method based on STE that is faster than conventional STE, has not limitation of angle dependency as TDI method (14).

Deformation and twisting of LV wall measured by torsion, provides insight into its regional and global contractile function (15) and is a sensitive indicator of cardiac dysfunction. Regional myocardial function allows clinician to early detection of myocardial dysfunction and therefore is important for patient management and evaluation of therapeutic efficacy (16). Systolic torsion in LV dysfunction was characterized by discontinuing counterclockwise rotation of the apex to base before end systole (3, 7). The size and shape of the LV change in patients with DCM as ventricle develops spherical configuration and LV rotational behavior changes (13), rotation in these patients is regionally heterogeneous and abnormal in magnitude and pattern (14).

Meluzin et al. (5) evaluated LV torsion, rotation and longitudinal, circumferential and radial strain and strain rate, in patient with idiopathic DCM by means of STE method and found all torsional systolic and diastolic parameters were decreased, another study used magnetic resonance imaging myocardial tagging method to study LV torsion in LV dysfunction also showed diminution in torsion and rotation and also decline in timing of peak torsion and also disorganization of untwisting in patients with DCM (4, 11). Corresponding three dimensional component of systolic and diastolic deformation, considerable variation exist in direction of basal and apical rotation in a subset of patients (5).

Matsumoto et al. (17) used 3D speckle tracking echocardiography to assess if LV dyssynchrony may affect LV torsional mechanics in patients with idiopathic dilated cardiomyopathy and found all rotational indexes were significantly impaired in patients with DCM and LV dyssynchrony negatively affect LV torsional mechanics and that in patients with wide QRS, LV torsion is significantly smaller than the patients with narrow QRS. Even when DCM is remodeled, circumferential and longitudinal myocardial function may not be normal, even when ejection fraction are normal as Okada et al. noted in their study (18).

Tibayan et al. evaluated systolic torsion in animal model and found in sheeps with tachycardia induced CMP there was decreased and delayed LV torsional deformation and loss of early diastolic recoil (19).

Global LV torsion was evaluate in children patients with DCM as well, Jin et al. (20) found in this group of patients that decreased systolic torsion and loss of elastic recoil contribute to decreased systolic and diastolic dysfunction, this diminution of torsion is mainly by diminution of counter clockwise apical rotation and augmented by somewhat less reduction in clock wise basal rotation.

Burns et al. (21) have shown in normal subjects with invasive measures of LV pressure that indexes of untwisting value are correlated to parameters of early diastolic filling phase but not events happening later in diastole. Decreases in the rate and degree of untwisting were associated with deteriorating of diastolic relaxation and reduced early diastolic suction. These findings lend further weight to the hypothesis that untwisting is so important in creating early diastolic LV suction, an important part of early diastolic filling phase. So, there was a close association observed between systolic and diastolic torsion parameters, suggesting that torsion might be an important mechanistic link between the phases of the cardiac cycles (21, 22). Our data are in keeping with previously confirmed findings; so, in conclusion, LV torsion (twisting and untwisting) allows a uniform distribution of fiber stress and fiber shortening across the wall, representing a critically important mechanism for both ejection and filling.

In our study twist and untwist and torsion are decreased as mentioned in above studies but time to peak untwist was not delayed but when we normalized this parameter to LV length, there was decline in time to peak untwisting, but when was normalized to LV torsion there was not significant change in this parameter, Both peak twist and untwist rate was decreased as previous studies showed. We think that by serial assessment of LV torsional behavior in clinical setting, we can take concepts for better management of patients with DCM.

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