Arch Cardiovasc Imaging. 2015 May; 3(2): e28112. DOI: 10.5812/acvi.28112

Published online 2015 May 23. **Case Report**

Reverse Left Ventricular Apical Rotation in Dilated Cardiomyopathy

Zahra Ojaghi-Haghighi [1]; Azin Alizadehasl [1,*]; Arash Hashemi [2]

[1]Echocardiography Research Center, Rajaie Cardiovascular, Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran
[2]General Cardiologist, Erfan Hospital, Tehran, IR Iran

*Corresponding author: Azin Alizadehasl, Echocardiography Research Center, Rajaie Cardiovascular, Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran. Tel: +98-2123922190, Fax: +98-2122055594, E-mail: alizadeasl@gmail.com

Received: February 27, 2015; **Accepted:** May 5, 2015

Introduction: We describe a 56-year-old woman with dilated cardiomyopathy, whose clinical assessment, including two-dimensional echocardiography, demonstrated a spherical left ventricular geometry with severe left ventricular enlargement and dysfunction as well as reverse apical rotation. Left ventricular twist and torsion were evaluated via echocardiography with velocity vector imaging; the patient was found to have reverse rotational movement. We hereby address these issues from an echocardiographic point of view.
Case Presentation: The patient was a 56-year-old woman, who referred to our clinic with complaints of dyspnea on exertion of 2 years' duration. By the time of her referral, the patient's dyspnea had exacerbated and reached New York Heart Association (NYHA) functional class III
Conclusions: These findings emphasize the potential clinical benefits of therapeutic procedures such as cardiac resynchronization therapy (CRT) or apex-sparing volume-reduction surgery in DCM. A better definition of the role and implications of reverse apical torsion in DCM and its importance and effectiveness in making therapeutic decisions like CRT implantation requires further studies.

Keywords: Torsion; Left; Dilated; Cardiomyopathies

1. Introduction

The torsional parameters of the left ventricle (LV) are sensitive indicators of cardiac performance (1, 2). The torsion/twist of the LV is the wringing motion of the heart around its long axis created by oppositely directed apical and basal rotations created by the contraction of the myofibers in the LV wall and is determined by contracting myofibers in the LV wall (3, 4) which are arranged in opposite directions between the subendocardial and subepicardial layers, (2, 5, 6) This motion is essential for regulating LV systolic and diastolic functions (7). There is a consensus that LV twist, expressed in degrees, and LV torsion, expressed in degrees per centimeter, both refer to the same phenomenon in cardiac function and define the base-to-apex gradient in a rotational angle along the longitudinal axis of the LV (5-9)

LV twist (degrees) is calculated as follows: LV twist = apical LV rotation - basal LV rotation and LV torsion = LV twist/LV length.

When viewed from the apex, the systolic rotation of the base is clockwise and that of the apex is counterclockwise. LV twist is assessed via tissue Doppler imaging (TDI) and speckle-tracking echocardiography (STE). There are two different speckle-tracking software technologies: velocity vector imaging (VVI) and two-dimensional (2D) STE.(7-11) TDI can be derived from primary velocity data with higher temporal resolution but with intrinsic angle-dependency constraints common to all Doppler methods. On the other hand, STE can be derived via the frame-to-frame tracking of the unique speckle patterns created by the interference of ultrasound beams within the tissue. Thus, VVI is a novel quantitative echocardiographic method which can track routine 2D echocardiographic images and is as such angle-independent (12-16).

There are some reports of the different degrees of decreased LV rotation and torsion and even reversed systolic apical rotation graphs in patients with dilated cardiomyopathy (DCM) (Figure 1). This finding may be associated with severe systolic and diastolic and even electrical abnormalities in these patients. It should be emphasized that the importance of a normally functioning LV apex for a normal cardiac performance is well known (13, 15, 16).

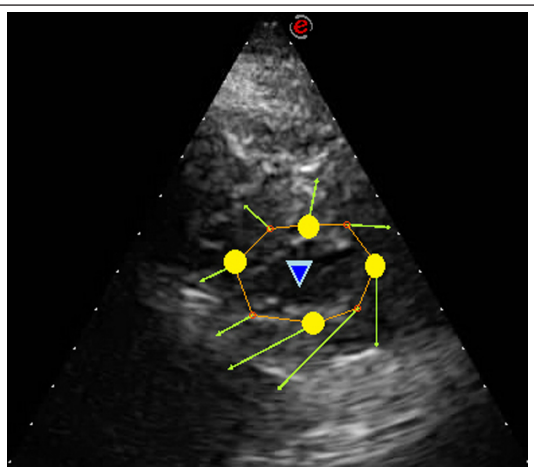

Figure 1. Delineation of the Apical Myocardial Short-Axis View During Velocity Vector Imaging Analysis

Table 1. Demographic and Echocardiographic Data of the Patients

Variable	Values
Demographic Data	
Age, y	56
Body surface area, kg/m^2	1.76
Heart rate, bpm	75
New york heart association	III
QRS duration, ms	160
Echocardiographic data	
Mitral regurgitation	Up to moderate
Left ventricular end-diastolic dimension index, mm/m^2	42
Left ventricular end-systolic dimension index mm/m^2	36
Left ventricular end-diastolic volume index, mL/m^2	187
Left ventricular end-systolic volume index, mL/m^2	163
Left ventricular sphericity index	1.69
Left ventricular ejection fraction, %	20
Peak S-wave velocity at the septal part of the mitral annulus, cm/s	4
Peak E' wave velocity at the septal part of the mitral annulus, cm/s	4
E/E' ratio	27

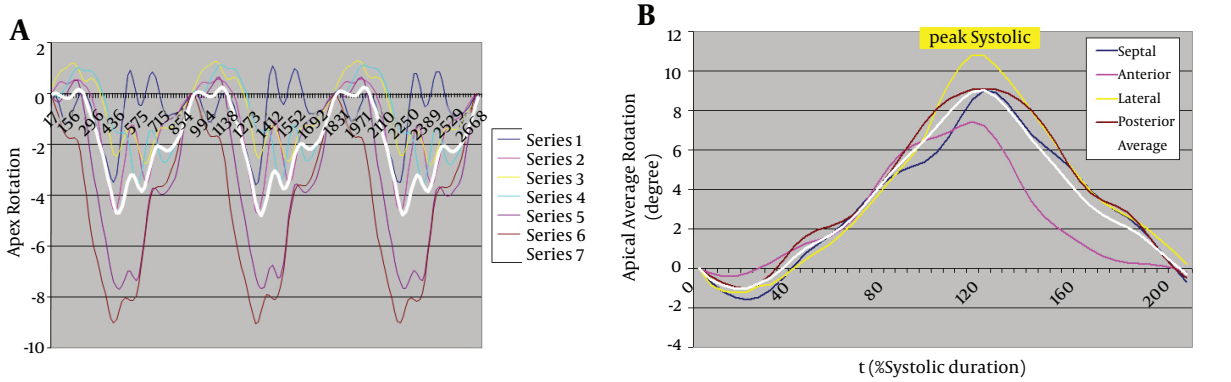

Figure 2. Reverse Left Ventricular Apical Rotation (a) Compared with Normal Left Ventricular Apical Rotation (b)

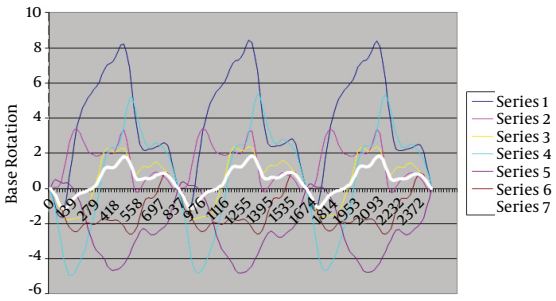

Figure 3. Left Ventricular Basal Rotation, Which is not Significantly Different From That in the Normal Subjects

2. Case Presentation

The patient was a 56-year-old woman, who referred to our clinic with complaints of dyspnea on exertion of 2 years' duration. By the time of her referral, the patient's dyspnea had exacerbated and reached New York Heart Association (NYHA) functional class III. She was given full evaluation, including coronary angiography, and was ultimately diagnosed with idiopathic DCM.

Echocardiographic assessment showed severe LV systolic dysfunction with a left ventricular ejection fraction (LVEF) of about 20% and up to moderate mitral regurgitation with concomitant significant diastolic dysfunction. The patient's echocardiographic data are presented in Table 1.

Two-dimensional conventional, pulse Doppler transthoracic echocardiography was performed with a commercial GE Vivid™ 7 system (Horten, Norway), equipped with an M3S multi-frequency harmonic phased-array transducer. Additionally, the torsional parameters were evaluated using MyLab™ 60 for VVI (Figure 2). The images were acquired with the subject at rest, lying in the lateral supine position at the end of expiration. A 2D electrocardiogram (ECG) was superimposed on the images, and end-diastole was considered the peak R-wave of the ECG. Additionally, LV global systolic function was evaluated via the modified biplane Simpson method for calculating LVEF by measuring the end-diastolic and end-systolic volumes in the 2D images. The patient's basal and apical LV rotation and LV torsion were quantified by STE (Figures 1, 3 and 4).

An advanced echocardiographic method applied on the routine grey scale echocardiographic images, VVI

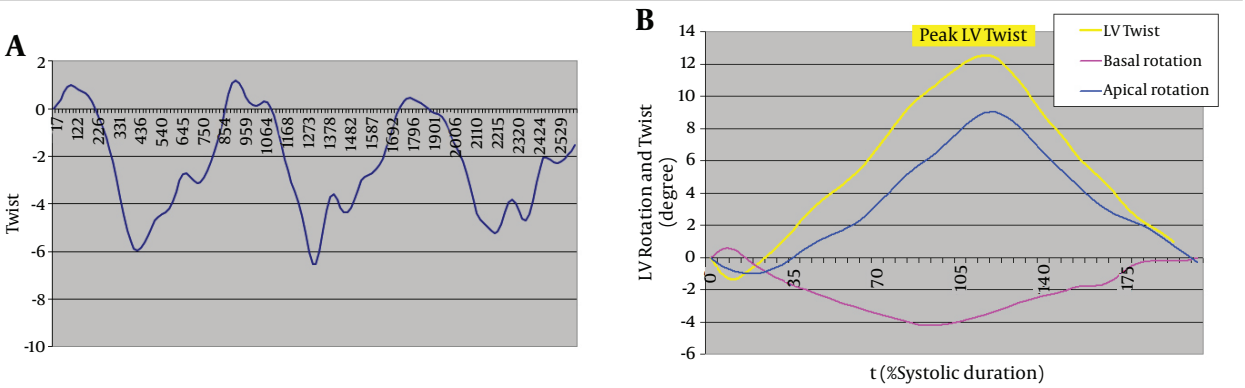

Figure 4. Reverse Left Ventricular Twist (a) Compared with Normal Left Ventricular Twist (b)

was originally developed for the analysis of the LV myocardium (1-5). Indeed, VVI is based on myocardial feature tracking and assesses myocardial motion in 2D, permitting an angle-independent measurement of tissue velocity and deformation (8-12). An endocardial tracing of a single frame is manually derived from a routine digital cine loop, and the periodic displacement of these regions is tracked in the subsequent frames. By tracking the moving tissue, the volume changes of the heart chambers can be calculated automatically, and the tracking data of all the sample regions (six segments for the basal and four segments for the apical levels) are transferred to an Excel Spreadsheet for LV average rotation and rotational velocity calculation (7-13).

3. Discussion

The main findings of this case presentation are that in patients with DCM, severe LV dilation and increased sphericity are associated with reduced or even reversed systolic apical rotation and that reversed systolic apical rotation with consequent loss of LV torsion reflects a more advanced disease stage with more severe LV remodeling, LV dyssynchrony, and more severe systolic and diastolic dysfunction compared to patients with DCM with normally directed apical rotation (11-15).

The special motion of the LV is a function of its helically oriented myofibrils. When we look at the LV from the apex, the systolic rotation of the base is clockwise, whereas the apex tends to rotate in a counterclockwise manner. A normally functioning LV apex is vitally important for a normal cardiac performance. LV apical rotation represents the foremost determinant of global LV systolic torsion; however, rapid apical back rotation plays a pivotal role in the suction of the blood into the LV cavity, supporting its filling at low pressures. It has been previously demonstrated that in normal subjects, LVEF is allied to apical, but not to basal, rotation, although only basal rotation is age-related: this suggests the clinical importance of apical rotation in the evaluation of LV systolic performance even without the complex calculation of LV twist. The loss of this particular motion pattern is probably associated with systolic and diastolic and even electrical abnormalities observed in DCM patients (13-16).

The myofibrillar geometry of the LV smoothly transforms from a slightly oblique orientation in the subendocardium to a circumferential orientation in the mid wall and again to an oblique orientation in the subepicardium layer. It has been postulated that this is the main mechanism for the special LV movement pattern. LV twist allows a uniform stress distribution on the fibers and fiber shortening across the wall: this represents an extremely important mechanism for both ejection and filling (12-15).

LV twist is calculated as the net difference in LV rotation between the apical and basal short-axis planes at isochronal time points. The short-axis views at the apical and basal levels are recorded with identical or very similar and close heart rates for the measurement of the basal and apical rotations (13-15). Peak LV twist in heart failure patients is 4.8 ± 2.6 degrees in comparison with 15.0 ± 3.6 degrees in control subjects according to Braunwald's Heart Disease, ninth edition, 2011 textbook.

Reversed apical rotation and loss of LV torsion in patients with DCM is very rare and is associated with significant LV remodeling, increased electrical dyssynchrony, impaired systolic function, and increased filling pressures - indicating a more advanced disease stage (13, 15-17). Bogdan A. et al. (13) (2009) demonstrated that in normal subjects, LVEF is associated with apical, but not with basal, rotation; however, only basal rotation is age-related. This finding signifies the clinical importance of the apical rotation in the valuation of LV systolic function even without the complex measurement of LV torsion.

The short-axis plane of the apex provides better acoustic settings than that of the base, and through-plane motion is minimal due to the limited longitudinal motion of the LV apex. Furthermore, the much smaller size of the circular cut of the apex simplifies its recording in DCM patients, while the basal cut is usually difficult to contain in a short-axis view in patients with extremely dilated LV.16 A reversed systolic apical rotation with the loss of LV torsion has also been observed in patients with noncompaction cardiomyopathy (13, 16-19).

LV apical rotation represents the foremost determinant of global LV systolic twist, while rapid apical back rotation plays a pivotal role in the suction of the blood into the LV cavity, stimulating its filling at low pressures (13, 15-17).

These findings emphasize the potential clinical benefits of therapeutic procedures such as cardiac resynchronization therapy (CRT) or apex-sparing volume-reduction surgery in DCM. A better definition of the role and implications of reverse apical torsion in DCM and its importance and effectiveness in making therapeutic decisions like CRT implantation requires further studies.

References

1. Jarnert C, Melcher A, Caidahl K, Persson H, Ryden L, Eriksson MJ. Left atrial velocity vector imaging for the detection and quantification of left ventricular diastolic function in type 2 diabetes. *Eur J Heart Fail.* 2008;**10**(11):1080–7.
2. Liu XW, Li ZA. [Assessment of cardiac twist in dilated cardiomyopathy using echocardiography velocity vector imaging]. *Zhonghua Yi Xue Za Zhi.* 2009;**89**(27):1892–6.
3. Merih B, Cihangir E. Predictive accuracy of tissue Doppler imaging for assessment of noninfarct myocardial region in patients with acute myocardial infarction. *Echocardiography.* 2007;**25**(2):79–85.
4. Hegazy AM, Akbar MA, Al-Sayegh A, Abdulkader BA. Predictive accuracy of tissue Doppler imaging for assessment of noninfarct myocardial region in patients with acute myocardial infarction. *Med Princ Pract.* 2007;**16**(1):40–6.
5. Deng Y, Alharthi MS, Thota VR, Yin L, Li C, Emani UR, et al. Evaluation of left ventricular rotation in obese subjects by velocity vector imaging. *Eur J Echocardiogr.* 2010;**11**(5):424–8.
6. Notomi Y, Setser RM, Shiota T, Martin-Miklovic MG, Weaver JA, Popovic ZB, et al. Assessment of left ventricular torsional deformation by Doppler tissue imaging: validation study with tagged magnetic resonance imaging. *Circulation.* 2005;**111**(9):1141–7.
7. Helle-Valle T, Crosby J, Edvardsen T, Lyseggen E, Amundsen BH, Smith HJ, et al. New noninvasive method for assessment of left ventricular rotation: speckle tracking echocardiography. *Circulation.* 2005;**112**(20):3149–56.
8. Notomi Y, Lysyansky P, Setser RM, Shiota T, Popovic ZB, Martin-Miklovic MG, et al. Measurement of ventricular torsion by two-dimensional ultrasound speckle tracking imaging. *J Am Coll Cardiol.* 2005;**45**(12):2034–41.
9. Pirat B, Khoury DS, Hartley CJ, Tiller L, Rao L, Schulz DG, et al. A novel feature-tracking echocardiographic method for the quantitation of regional myocardial function: validation in an animal model of ischemia-reperfusion. *J Am Coll Cardiol.* 2008;**51**(6):651–9.
10. Perk G, Tunick PA, Kronzon I. Non-Doppler two-dimensional strain imaging by echocardiography–from technical considerations to clinical applications. *J Am Soc Echocardiogr.* 2007;**20**(3):234–43.
11. Sirbu C, Herbots L, D'Hooge J, Claus P, Marciniak A, Langeland T, et al. Feasibility of strain and strain rate imaging for the assessment of regional left atrial deformation: a study in normal subjects. *Eur J Echocardiogr.* 2006;**7**(3):199–208.
12. Wang J, Khoury DS, Yue Y, Torre-Amione G, Nagueh SF. Left ventricular untwisting rate by speckle tracking echocardiography. *Circulation.* 2007;**116**(22):2580–6.
13. Popescu BA, Beladan CC, Calin A, Muraru D, Deleanu D, Rosca M, et al. Left ventricular remodelling and torsional dynamics in dilated cardiomyopathy: reversed apical rotation as a marker of disease severity. *Eur J Heart Fail.* 2009;**11**(10):945–51.
14. Hori M, Yeliin EL, Sonnenblick EH. Left ventricular diastolic suction as a mechanism of ventricular filling. *Jpn Circ J.* 1982;**46**(1):124–9.
15. Suga H, Goto Y, Igarashi Y, Yamada O, Nozawa T, Yasumura Y. Ventricular suction under zero source pressure for filling. *Am J Physiol.* 1986;**251**(1 Pt 2):H47–55.
16. Kim HK, Sohn DW, Lee SE, Choi SY, Park JS, Kim YJ, et al. Assessment of left ventricular rotation and torsion with two-dimensional speckle tracking echocardiography. *J Am Soc Echocardiogr.* 2007;**20**(1):45–53.
17. van Dalen BM, Caliskan K, Soliman OI, Nemes A, Vletter WB, Ten Cate FJ, et al. Left ventricular solid body rotation in non-compaction cardiomyopathy: a potential new objective and quantitative functional diagnostic criterion? *Eur J Heart Fail.* 2008;**10**(11):1088–93.
18. Notomi Y, Popovic ZB, Yamada H, Wallick DW, Martin MG, Oryszak SJ, et al. Ventricular untwisting: a temporal link between left ventricular relaxation and suction. *Am J Physiol Heart Circ Physiol.* 2008;**294**(1):H505–13.
19. Haghighi ZO, Alizadehasl A, Moladoust H, Ardeshiri M, Mostafavi A, Rezaeiyan N, et al. Left Ventricular Torsional Parameters in Patients With Non-Ischemic Dilated Cardiomyopathy. *Arch Cardiovasc Imaging*. 2015 Feb; 3(1): e26751.