

Tissue Doppler Imaging Values in Hypertrophic Cardiomyopathy According to Left Ventricular Outflow Gradient

Rezvaneh Salehi MD^{1*}, Azin Alizadehasl MD¹

1. Cardiovascular Research Center, Tabriz University of Medical Sciences, Tabriz, Iran.

(Received 21 May 2010; Accepted 17 Sep 2010)

Abstract

Background: The aim of the present study was to investigate the effects of the left ventricular outflow gradient on tissue doppler imaging (TDI) values in patients with hypertrophic cardiomyopathy (HCM).

Methods: This prospective observational study was conducted in a tertiary referral heart hospital (Tabriz, Iran) from March 2004 to March 2008. Fifty two patients (28 men and 24 women) with definite diagnoses of HCM were selected for the study. 2D, M-mode echocardiography and TDI were performed for all patients. In TDI, the velocities of myocardium was measured at lateral and septal corner of the mitral valve annulus in peak early diastolic (Ea), peak late diastolic (Aa) and peak systolic (Sa). Then, the TDI velocities were compared in patients with left ventricle outflow gradient less and more than 30 mmHg.

Results: The mean age of the patients was 42.13 ± 12.22 years. All patients were in New York Heart Association functional class I or II. The mean EF was $55 \pm 7\%$. It was found that TDI velocities are reduced in patients with HCM, compared to normal values and Ea in septal corners of the mitral annulus was reduced more in patients with left ventricle outflow gradient > 30 mmHg (5.3 ± 1.6 vs 7.1 ± 1.2 , $P=0.01$).

Conclusion: TDI findings specially peak early diastolic (Ea) mitral annular velocity are good parameters in HCM patients assessment.

J Cardiovasc Thorac Res 2011; Vol.2 (4): 19- 22

Keywords: Hypertrophic Cardiomyopathy—Tissue Doppler Imaging—Left Ventricular Outflow Gradient

*Corresponding Author: Rezvaneh Salehi MD, Cardiovascular Research Center, Madani Heart Hospital, Tabriz University of Medical Sciences, Tabriz, Iran. Tel: +98 411 - 3357767 Fax: +98 411 - 3344021 E-mail: Saleiir2002 @Yahoo.com



Introduction

Hypertrophic cardiomyopathy (HCM) is an autosomal dominant disorder characterized by unexplained left ventricular hypertrophy and abnormal diastolic relaxation.¹⁻³ This disease represents the leading cause of sudden cardiac death (SCD) in children and young adults.⁴ In general, HCM development is rare before puberty and may not develop until later in life.⁵ Today, more than 200 different genetic mutations have been identified to cause the disease. The development of signs and symptoms in HCM is slow, therefore, making early diagnosis often difficult.⁶⁻⁷ The early detection of the disease to begin treatment is the ultimate goal in symptomatic HCM. In past few decades the conventional echocardiography has been a way for detecting HCM.⁸ Many studies have indicated that the conventional echocardiography is unreliable in predicting the clinical status of HCM patients, including cardiac symptoms and exercise capacity, primarily due to their dependence on loading conditions.⁹ Therefore, Tissue Doppler Imaging (TDI), which uses doppler to quantify the velocity of tissue rather than blood, has been the more recent substitute method introduced as a sensitive clinical tool for detecting structural abnormalities such as HCM over a decade.^{8,10} This method allows quantitative measurement of myocardial systolic and diastolic velocities and provides the potential for load-independent assessments of diastolic function.^{1, 10} Additionally, TDI can demonstrate abnormalities in systolic and early diastolic velocities at the pre-clinical stage and can be used to identify people who carry an abnormal gene. Thus, it is a good method for early detection of HCM.⁶ Several studies have recently been conducted to investigate the usefulness of TDI in detecting HCM. For instance, Matsumura et al. (2002) measured TDI values of 85 patients with HCM and compared them with 60 normal controls. The study showed that early diastolic mitral annular velocities measured using TDI are reduced in patients with HCM, compared to normal controls.¹⁰ The aim of the present study was to investigate the TDI values in Iranian HCM patients. The effect of Left Ventricular Outflow Gradient was also investigated.

Methods

Fifty-two patients with definite diagnoses of Hypertrophic cardiomyopathy (HCM) were studied from March 2004 to March 2008. The study was conducted at the echocardiography laboratory of Madani Heart Hospital of Tabriz Medical School. The study was approved by the ethic committee of Tabriz University of medical sciences. Diagnosis of HCM was confirmed by echocardiographic evidence of hypertrophied interventricular septum (interventricular septum thickness ≥ 15 mm) without any other disease-inducing left ventricular hypertrophy. The patients were selected on the basis of the following criteria: Normal sinus rhythm and heart rate less than 90 beat /min during the time of Tissue Doppler study, left ventricle ejection fraction more than 45% with normal left ventricle cavity dimension.

Echocardiography Protocol

Echocardiographic studies were performed with a commercially available echocardiography equipment (VIVID7, GE, USA) using 2.5 MHz transducer. 2D, M-mode and Tissue Doppler Imaging (TDI) data were obtained through transthoracic echocardiography (TTE). For the purpose of further investigation, our patients were divided into two groups according to their left ventricular outflow gradient (group A: LV outflow gradient ≤ 30 mmHg, and group B: LV outflow gradient more than 30 mmhg. In this study the velocity of myocardium was measured at lateral and septal site of mitral valve annulus in peak early diastolic (Ea), peak late diastolic (Aa) and peak systolic (Sa).

Statistical Analysis

All the obtained data were analyzed by SPSS software version 16.0 (SPSS Inc, Chicago, IL). Continuous data are expressed as mean values \pm SD. Student's t-test was used to assess the significant differences of mean values between patients with left ventricle out flow gradient and intraventricular septum thickness. A P-value ≤ 0.05 was considered statistically significant.



Results

Fifty-two patients with definite diagnosis of hypertrophic cardiomyopathy participated in the present study. Twenty-four patients (46.15 %) were females and 28 patients were males. The mean age of the patients was 42.13 ± 12.22 years. All patients were in New York Heart Association functional class I or II and the mean EF of the patients was $55 \pm 7\%$. As mentioned before, in this study the velocity of myocardium was measured at lateral and septal site of mitral valve annulus in peak early diastolic (Ea), peak late diastolic (Aa) and peak systolic (Sa). Table 1 shows the measured TDI velocities of all patients. The comparison of TDI velocities between the two groups are shown in Table 2.

Table 1- Color TDI velocities in patients with Hypertrophic cardiomyopathy

	Patients (n=52)
Ea (Lat)	7.2 ± 2.1
Aa (Lat)	6.3 ± 1.2
Sa (Lat)	9.9 ± 1.6
Ea (Sept)	6.9 ± 1.5
Aa (Sept)	6.4 ± 1.3
Sa (Sept)	9.8 ± 2.3

Peak velocities, values in cm/sec
Mean \pm SD

Table 2- Comparison the results of color TDI velocities according to left ventricle outflow gradient

	Group ALV n=31	Group BLV n=21	P Value
Ea (Lat)	7.6 ± 2.2	7.1 ± 2.4	0.12
Aa (Lat)	6.3 ± 1.3	5.9 ± 1.4	0.04
Sa (Lat)	9.8 ± 1.3	10.3 ± 2.1	0.25
Ea (Sept)	7.1 ± 1.2	5.3 ± 1.6	0.01
Aa (Sept)	5.9 ± 1.1	5.1 ± 1.5	0.03
Sa (Sept)	10.0 ± 2.3	9.3 ± 1.8	0.22

TDI: Tissue Doppler Imaging; LV: Left Ventricle;
Group ALV: LV outflow gradient ≤ 30 mmHg;
Group BLV: LV outflow gradient > 30 mmHg;
Peak velocities, values in cm/sec
Mean \pm SD

Discussion

We have demonstrated that echocardiographic evaluations of the patients with HCM based on left ventricular outflow gradient had good correlation with diastolic dysfunction severity. As shown in table I, all myocardial velocities decreased in the patients with HCM in comparison to the normal values. Recent studies have shown that TDI is a reliable method for early detection of HCM in genotype-positive patients before the onset of hypertrophy.⁹⁻¹² Reduced systolic and diastolic TDI velocities have a high sensitivity and specificity for identifying mutation of HCM carriers. Serial echocardiographic evaluation of patients with initially genotype positive but phenotype negative showed further reduction in TDI velocities and subsequent development of hypertrophy and diastolic function impairment during follow up.⁶⁻⁷ Impaired LV relaxation is a prominent diastolic abnormality in patients with HCM.¹¹⁻¹³ However, mitral inflow and pulmonary venous flow assessments may not be sensitive for the detection of high filling pressures. Despite the patients who had LV systolic dysfunction, the simultaneous echocardiography and invasive hemodynamic study revealed no significant correlation between mitral inflow or pulmonary venous flow parameters and filling pressures.¹⁴ There is a good correlation between LV filling pressure, mitral inflow E velocity, and TDI diastolic MV annular velocities (Ea).¹⁶ Reduced TDI early diastolic velocities that have been detected in our study indicate that diastolic abnormalities may precede the onset of hypertrophy.¹⁴⁻¹⁶ In addition, TDI has been used to monitor the response of invasive therapies such as ethanol ablation or myectomy. Therefore, TDI could be a valuable tool to select for the HCM patients; also interestingly we observed positive correlation between reduced Ea septal and LVOT gradient and this finding could be useful in diagnosis obstructive forms of HCM.



References

1. Araujo AQ, Arteaga E, Ianni BM, Salemi VM, Ramires FJ, Matsumoto AY et al. Usefulness of a new proposed tissue Doppler imaging global function index in hypertrophic cardiomyopathy. *Echocardiography* 2006; 23:197-201.
2. Koffas H, Dukes-McEwan J, Corcoran BM, Moran CM, French A, Sboros Vet al. Pulsed tissue Doppler imaging in normal cats and cats with hypertrophic cardiomyopathy. *J Vet Intern Med* 2006; 20: 65-77.
3. MacDonald KA, Kittleson MD, Garcia-Nolen T, Larson RF, Wisner ER. Tissue Doppler imaging and gradient echo cardiac magnetic resonance imaging in normal cats and cats with hypertrophic cardiomyopathy. *J Vet Intern Med* 2006; 20(3): 627-634.
4. Michels M, Soliman OI, Kofflard MJ, Hoedemaekers YM, Dooijes D, Majoor-Krakauer D et al. Diastolic abnormalities as the first feature of hypertrophic cardiomyopathy in Dutch myosin-binding protein C founder mutations. *JACC Cardiovasc Imaging* 2009; 2: 58-64.
5. Carlos Sampedrano C, Chetboul V, Gouni V, Nicolle AP, Pouchelon JL, Tissier R. Systolic and diastolic myocardial dysfunction in cats with hypertrophic cardiomyopathy or systemic hypertension. *J Vet Intern Med* 2006; 20(5): 1106-1115.
6. Teske AJ, De Boeck BW, Melman PG, Sieswerda GT, Doevendans PA, Cramer MJ. Echocardiographic quantification of myocardial function using tissue deformation imaging, a guide to image acquisition and analysis using tissue Doppler and speckle tracking. *Cardiovasc Ultrasound* 2007; 5: 27-30.
7. Kato T, Noda A, Izawa H, Nishizawa T, Somura F, Yamada A, et al. Myocardial velocity gradient as a noninvasively determined index of left ventricular diastolic dysfunction in patients with hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2003 16; 42: 278-285.
8. Nagueh SF, McFalls J, Meyer D, Hill R, Zoghbi WA, Tam JW, et al. Tissue Doppler imaging predicts the development of hypertrophic cardiomyopathy in subjects with subclinical disease. *Circulation* 2003; 108: 395-398.
9. Yamada H, Oki T, Tabata T, Mishiro Y, Abe M, Onose Y, et al. Assessment of the Systolic Left Ventricular Myocardial Velocity Profile and Gradient Using Tissue Doppler Imaging in Patients with Hypertrophic Cardiomyopathy. *Echocardiography* 1999; 16: 775- 783.
10. Matsumura Y, Elliott PM, Virdee MS, Sorajja P, Doi Y, McKenna WJ. Left ventricular diastolic function assessed using Doppler tissue imaging in patients with hypertrophic cardiomyopathy: relation to symptoms and exercise capacity. *Heart* 2002 ; 87: 247-251.
11. Spirito P, Belone P, Harris KM, et al: Magnitude of left ventricular hypertrophy and risk of sudden death in hypertrophic cardiomyopathy. *N Engl J Med* 2000; 324: 1778-1785.
12. Maron BJ, Towbin JA, Thiene G, et al: Contemporary definitions and classification of the cardiomyopathies: An American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. *Circulation* 2006; 113: 1807-1816.
13. Solomon SD, Wolff S, Watkins H, et al: Left ventricular hypertrophy and morphology in familial hypertrophic cardiomyopathy associated with mutations of the beta-myosin heavy chain gene. *J Am Coll Cardiol* 1993; 22: 498-505.
14. Maron BJ, McKenna WJ, Danielson GK, et al: A report of the American College of Cardiology Foundation Task Force on Clinical Expert Consensus Documents and the European Society of Cardiology Committee for Practice Guidelines. *J Am Coll Cardiol* 2003; 42: 1687-1713.
15. Henry WL, Clark CE, Epstein SE: Asymmetric septal hypertrophy: echocardiographic identification of the pathognomonic anatomic abnormality of IHSS. *Circulation* 1973; 47: 225- 233.
16. Gilbert BW, Pollick C, Adelman AG, Wigle ED: Hypertrophic cardiomyopathy: Subclassification by M-mode echocardiography. *Am J Cardiol* 1980; 45: 861-872.