

Original Article

Value of Tissue Doppler Imaging in Diagnosing Coronary Artery Involvement in Patients Suspected of Coronary Artery Disease

Farahnaz Nikdoost, MD¹ ; Hasan Zarei, MD^{1*};
Seyed Abdolhussein Tabatabaei, MD¹

Abstract

Background: There is limited evidence regarding the application of tissue Doppler imaging (TDI) parameters in assessing the severity of coronary artery disease (CAD).

Objective: To determine the value of TDI parameters to assess the presence and severity of CAD.

Methods: Fifty consecutive patients suspected of CAD and 20 gender- and age-matched healthy individuals were assessed using TDI to assess both systolic and diastolic parameters. Those with a previous history of myocardial infarction, hypertension, diabetes mellitus, or left ventricular systolic dysfunction were not included. The patients underwent coronary angiography to determine the presence and severity of CAD.

Results: Comparing echocardiographic left ventricular systolic and diastolic parameters between the patients and healthy groups showed lower mean E, A, S', E', and A' parameters as well as higher mean E/A ratio and deceleration time parameters in the patient group than in the healthy controls. Also, comparing the right ventricular systolic and diastolic parameters between the patient and healthy groups revealed that the former group had a significantly lower mean Eparameter than the healthy group.

Conclusion: TDI velocity indices had a high value in the diagnosis of the left ventricular dysfunction due to CAD and thus can be a good option to discriminate CAD from healthy condition. (*Iranian Heart Journal 2015; 16(1):6-11*)

Keywords: ■Coronary artery disease; ■Tissue Doppler imaging; ■Velocity; ■Diagnosis

¹Department of Cardiology, Shariati hospital, Tehran University of Medical Sciences, Tehran, Iran.

*Corresponding Author: Hasan Zarei, MD

Tel: 09171622688

E-mail: hasanzry@yahoo.com

Received: March 15, 2015

Accepted: May 21, 2015

Tissue Doppler imaging (TDI) is an echocardiographic technique that uses Doppler principles to measure myocardial motion velocity. TDI quantifies the higher amplitude and lower-velocity signals of myocardial tissue motion.¹⁻³ TDI has been introduced as a method to quantify the myocardial function in terms of tissue velocities, and the related results so far have been promising in this regard. As demonstrated in animal models and in patients with coronary artery disease (CAD), myocardial ischemia is characterized by a decrease in peak systolic myocardial velocity (S'), indicating the impairment of the regional contractile function.^{4,5} In this regard, some analyses, including measurement of myocardial velocities during the isovolumic contraction phase and isovolumic relaxation phase in addition to ejection velocities, are now considered for the assessment of the myocardial functional status.⁶⁻⁸ In the non-ischemic ventricle, the isovolumic contraction time (IVCT) is dominated by a positive velocity spike of short duration, which represents slight longitudinal shortening before the left ventricular (LV) ejection.^{9, 10} During the isovolumic relaxation time (IVRT), there is a pattern opposite to that during the IVCT, with a negative velocity spike of short duration, representing slight elongation before the onset of filling.¹¹ During moderate ischemia, there is a decrease in systolic shortening and then a decrease in peak early ejection and mid-ejection velocities. With progressing myocardial ischemia, the IVR velocities reverse and a large positive velocity component (post-systolic shortening of ischemic myocardium) persists throughout the entire IVR period, and in some cases continues after the early-diastolic LA/LV pressure crossover.¹²⁻¹⁴ Thus, in severely ischemic and dyskinetic myocardium, the IVC and IVR velocities are the strongest markers of myocardial dysfunction.¹⁵⁻¹⁷ Some studies have also investigated the usefulness of the time intervals in the assessment of CAD.¹⁸⁻²⁰ In

patients with acute myocardial infarction, the conventional Tei index was found to be significantly greater than that in healthy controls.^{21, 22} Furthermore, in the studies evaluating the systolic and diastolic function of the LV by TDI in patients with or without pre-infarction angina in acute myocardial infarction, the patients with preinfarction angina showed values of E' and E'/A' higher than patients without preinfarction angina, while the ratio E/E' and the MPI were significantly lower in the first group of patients.²³⁻²⁶ However, few studies have been published on the applications of TDI parameters in assessing the severity of CAD. The present study aimed to determine the value of TDI parameters to assess the presence and severity of CAD and compare them with healthy individuals.

Materials and Methods

This cross-sectional study recruited 50 consecutive patients suspected to have CAD and 20 gender- and age-matched healthy individuals. Those with a previous history of myocardial infarction, hypertension, diabetes mellitus, or LV systolic dysfunction were not included. Controls were selected from patients who presented to our hospital with atypical chest pain and exercise stress test and had unremarkable coronary angiography results.

All the participants were assessed using TDI (GE Vivid 7) to assess both systolic and diastolic parameters of LVEF, LVId, LAV, E , A , E/A ratio, DT , S' , E' , A' , and E/E' ratio. Also, the patients underwent coronary angiography to determine both presence and severity of coronary arteries involvement.

The results are presented as mean \pm standard deviation (SD) for the quantitative variables and were summarized by absolute frequencies and percentages for the categorical variables. The continuous variables were compared using the t -test or non-parametric Mann-Whitney U test whenever the data did not appear to have normal distribution or when

the assumption of equal variances was violated across the two study groups. The categorical variables were, on the other hand, compared using the chi-square test or the Fisher exact test when more than 20 % of cells with an expected count <5 were observed. For the statistical analysis, the statistical software SPSS version 15.0 for Windows (SPSS Inc., Chicago, IL) was used. P values ≤ 0.05 were considered statistically significant.

Results

In total, 50 CAD patients and 20 healthy ones were assessed. The two groups were matched in terms of male gender (60% vs. 60%), mean age (61.86 ± 10.96 vs. 59.50 ± 12.18 years; $P = 0.433$), and mean body mass index (BMI) (26.45 ± 6.55 vs. 26.28 ± 5.52 kg/m², $P = 0.920$). None of the participants had systemic hypertension or diabetes. The prevalence of smoking was 26 % and 15 % ($P = 0.529$) and the prevalence of dyslipidemia was 52 % and 60% ($P = 0.544$). In the patient group, 74% suffered from angina pain. Also, regarding the severity of coronary involvement, 58% had single-vessel disease, 24% had two-vessel disease, and 18% had three-vessel disease. Regarding dynamical echocardiographic parameters, the mean left ventricular ejection fraction (LVEF) in the patients and healthy subjects was 54.28 ± 4.06 % and 55.65 ± 3.59 % with no difference ($P = 0.192$). Also, the mean LAVI was similar in the groups (29.90 ± 10.13 vs. 31.95 ± 6.42 ; $P = 0.405$). However, the mean LVId was significantly lower in the patients compared to the healthy ones (47.07 ± 6.78 vs. 31.95 ± 6.42 ; $P < 0.001$).

A comparison of the echocardiographic LV systolic and diastolic parameters between the patients and healthy groups (Table 1) showed lower mean E, A, S', E', and A' parameters as well as higher mean E/A ratio and deceleration time parameters in the patients than in the healthy controls. Also, a comparison of the right ventricular systolic

and diastolic parameters between the patients and the healthy individuals revealed that the former group had a significantly lower mean E parameter compared with the healthy group (Table 1).

Table 1: Left and right ventricular function parameters

Parameter	Patient Group (n = 50)	Healthy Group (n = 20)	P- Value
Left ventricular			
E Velocity*	0.53 ± 0.15	0.68 ± 0.26	0.048
A velocity	0.67 ± 0.19	0.83 ± 0.18	0.023
E/A ratio	0.93 ± 0.60	0.75 ± 0.41	0.040
DT(ms)**	217.18 ± 64.89	214.45 ± 13.09	0.004
S' velocity	0.12 ± 0.09	0.16 ± 0.11	0.020
E' velocity	0.12 ± 0.07	0.14 ± 0.09	0.032
A' velocity	0.08 ± 0.02	0.14 ± 0.06	0.047
Right ventricular			
E velocity	0.44 ± 0.09	0.52 ± 0.09	0.001
A velocity	0.50 ± 0.12	0.52 ± 0.07	0.429
E/A ratio	0.86 ± 0.25	1.01 ± 0.16	0.220
DT	365.42 ± 24.03	261.84 ± 72.09	0.532
S' velocity	0.14 ± 0.06	0.15 ± 0.07	0.886
E' velocity	0.13 ± 0.09	0.14 ± 0.07	0.749
A' velocity	0.13 ± 0.07	0.17 ± 0.10	0.214

* DT: Deceleration time

** All velocities are as cm/s

Discussion

In the present study, we hypothesized that the noninvasive diagnosis of CAD by quantitative TDI is best performed using diagnostic models based on segmental velocities. In this regard, we showed that the TDI velocity indices had a high value to diagnosis LV dysfunction due to coronary artery involvement and thus can be a good option to discriminate CAD from healthy condition. Our findings were also previously described by other authors. In a study by Mädlar et al.²⁷ to develop optimal methods for the objective noninvasive diagnosis of CAD using myocardial Doppler velocities during Dobutamine stress echocardiography, tissue Doppler digital data during Dobutamine stress in 289 subjects was reviewed and measured myocardial responses by off-line analysis of 11 LV segments. Diagnostic criteria were also developed by comparing 92 normal subjects. In their study, the best cut-points from receiver-operator curves diagnosed left anterior descending, circumflex, and right

coronary disease with sensitivities and specificities of 80% and 80%, 91% and 80%, and 93 % and 82 %, respectively. In another study by Agarwal²⁸ and in a systematic review, it was shown that at rest, TDI was associated with a significant decrease in the pooled maximum systolic velocity among CAD patients compared to those without CAD, but there were no significant differences in maximum early and late diastolic velocities. Post stress, TDI was associated with a significant decrease in maximum early diastolic velocity and maximum late diastolic velocity among CAD patients compared to those without CAD. Their results finally suggested that TDI may have a role in the evaluation of CAD. In Hoffmann et al.²⁹ study to determine how the LV wall motion assessed by echocardiographic TDI is affected by the increasing severity of CAD among patients with stable angina pectoris and preserved EF, 82 patients with suspected angina pectoris, no previous cardiac history, and a normal EF were examined with color TDI prior to coronary angiography. The authors showed that global systolic and diastolic performance by TDI (in terms of global s' and E/e') were negatively correlated to the number of vessels with significant stenoses. Regional analyses revealed that in one- and two-vessel disease, e' decreased significantly in the segments supplied by a stenotic artery. In patients with one-vessel disease, a' increased compensatorily with a significant reduction in the e'/a'-ratio. Both regional s' and global s' were significantly reduced in patients with three-vessel disease. Also, Fennira et al.³⁰ revealed that compared with healthy subjects, patients with CAD have a significant increase in the IVRT and lower Ea and report Ea/Aa. These anomalies are more pronounced in akinetic segments compared with hypokinetic segments. Relying on data from coronary angiography, they found that in patients whose IVRT had increased, Ea and Ea/Aa were reduced in most segments that were hypoperfused rather than in the normally-

perfused segments. They also showed that an IVRT >70 ms and Ea < 8.3 cm/s emerged as threshold values to identify myocardial ischemia. A higher number of coronary lesions is correlated with a more severe decline in Ea and Ea/Aa and IVRT increase. In total, it seems that using different LV TDI indices, we can appropriately diagnosis coronary artery involvement in patients suspected of CAD.

References

1. Correale M, Caivano M, Totaro A, et al. Tissue Doppler Imaging in the clinical practice It. J Practice Cardiol 2010;2: 27-38.
2. Vinereanu D, Khokhar A, Fraser AG. Reproducibility of pulsed wave tissue Doppler echocardiography. J Am Soc Echocardiogr 1999;12: 492-99.
3. Carolyn Y Ho, Scott D, Solomon A. Clinician's Guide to Tissue Doppler Imaging. Circulation 2006;113:e396-e398.
4. Van de Veire NR, Yu CM, Ajmone-Marsan N, et al. Triplane tissue Doppler imaging: a novel three-dimensional imaging modality that predicts reverse left ventricular remodelling after cardiac resynchronisation therapy. Heart 2008; 94:e9.
5. Cui W, Roberson DA, Chen Z, Madronero LF, Cuneo BF. Systolic and diastolic time intervals measured from Doppler tissue imaging: normal values and Z-score tables, and effects of age, heart rate, and body surface area. J Am Soc Echocardiogr 2008; 21:361-70.
6. Correale M, Totaro A, Ieva R, Brunetti ND, Di Biase M. Time intervals and myocardial performance index by tissue doppler imaging. Intern Emerg Med 2011; 6: 393-402.
7. Tei C, Nishimura AR, Seward JB, Tajik AJ. Noninvasive Doppler-derived myocardial performance index: Correlation with simultaneous measurements of cardiac catheterization measurements. J Am Soc Echocardiogr. 1997;10: 169-78.
8. Sohn D-W, Chai I-H, Lee D-J, et al. Assessment of mitral annulus velocity by Doppler tissue imaging in the evaluation of left ventricular

- diastolic function. *J Am CollCardiol.* 1997;30: 474-480.
9. Tekten T, Onbasili AO, Ceyhan C, Unal S, Discigil B. Novel approach to measure myocardial performance index: pulsed-wave tissue Doppler echocardiography. *Echocardiography.* 2003;20: 503-10.
 10. Rojo EC, Rodrigo JL, de Isla LP, et al. Disagreement between tissue Doppler imaging and conventional pulsed wave Doppler in the measurement of myocardial performance index. *Eur J Echocardiogr* 2006; 7: 356-64.
 11. Gaibazzi N, Petrucci N, Ziacchi V. Left ventricle myocardial performance index derived either by conventional method or mitral annulus tissue-Doppler: a comparison study in healthy subjects and subjects with heart failure. *J Am SocEchocardiogr.* 2005;18(12):1270-6.
 12. Tayyareci Y, Tayyareci G, Nişancı Y, et al. Evaluation of the severity of mitral stenosis with a new index: isovolumic myocardial acceleration. *Turk KardiyolDernArs* 2008; 36: 388-94.
 13. Lyseggen E, Rabben SI, Skulstad H, et al. Myocardial acceleration during isovolumic contraction: relationship to contractility. *Circulation* 2005; 111:1362-69.
 14. Hashimoto I, Li XK, Bhat AH, Jones M, Sahn DJ. Quantitative assessment of regional peak myocardial acceleration during isovolumic contraction and relaxation times by tissue Doppler imaging. *Heart* 2005; 91: 811-16.
 15. Lindqvist P, Waldenström A, Wikström G, Kazzam E. Potential use of isovolumic contraction velocity in assessment of left ventricular contractility in man: A simultaneous pulsed Doppler tissue imaging and cardiac catheterization study. *Eur J Echocardiogr* 2007; 8: 252-8.
 16. Vogel M, Schmidt MR, Kristiansen SB, et al. Validation of myocardial acceleration during isovolumic contraction as a novel noninvasive index of right ventricular contractility: comparison with ventricular pressure-volume relations in an animal model. *Circulation* 2002; 105:1693-99.
 17. Vogel M, Cheung MMH, Li J, et al. Noninvasive Assessment of left ventricular force-frequency relationships using tissue Doppler-derived isovolumic acceleration. *Circulation* 2003;107: 1647.
 18. Edvardsen T, Urheim S, Skulstad H, Steine K, Ihlen H, Smiseth OA. Quantification of leftventricularsystolicfunction by tissue Doppler echocardiography: added value of measuringpre- and post ejection velocities in ischemic myocardium. *Circulation* 2002; 105: 2071-7.
 19. Margulescu AD, Thomas DE, Ingram TE, et al. Can isovolumic acceleration be used in clinical practice to estimate ventricular contractile function? Reproducibility and regional variation of a new noninvasive index. *J Am SocEchocardiogr* 2010; 23:423-31.
 20. Yu CM, Chau E, Sanderson JE, et al. Tissue Doppler echocardiographic evidence of reverse remodeling and improved synchronicity by simultaneously delaying regional contraction after biventricular pacing therapy in heart failure. *Circulation* 2002;105: 438-45.
 21. Yu CM, Fung WH, Lin H, et al. Predictors of left ventricular reverse remodeling after cardiac resynchronization therapy for heart failure secondary to idiopathic dilated or ischemic cardiomyopathy. *Am J Cardiol* 2003;91:684-8.
 22. Correale M, Ceglia M, Brunetti ND, et al. Myocardial tissue Doppler echocardiography and atorvastatin in Heart Failure. *Euro Heart J* 2009;30(Abtract Supplement):343.
 23. Edvardsen T, Urheim S, Skulstad H, et al. Quantification of Left Ventricular Systolic Function by Tissue Doppler Echocardiography. Added Value of Measuring Pre- and Postejction Velocities in Ischemic Myocardium. *Circulation* 2002;105:2071.
 24. Lakoumentas JA, Panou FK, Kotseroglou VK, Aggeli KI, Harbis PK. The Tei Index of Myocardial Performance: Applications in Cardiology. *Hellenic J Cardiol* 2005;46: 52-58.
 25. Yilmaz R, Celik S, Baykan M, et al. Pulsed wave tissue Doppler-derived myocardial performance index for the assessment of left ventricular thrombus formation risk after acute myocardial infarction. *Am Heart J* 2004; 148: 1102-1108.
 26. Baykan M, Yilmaz R, Celik S, et al. Assessment of left ventricular systolic and diastolic function by Doppler tissue imaging in patients with

preinfarction angina. J Am Soc Echocardiogr 2003; 16: 1024-30.

27. Madler CF, Payne N, Wilkenshoff U, Cohen A, Derumeaux GA, Pierard LA, Engvall J, Brodin LA, Sutherland GR, Fraser AG; Myocardial Doppler in Stress Echocardiography (MYDISE) Study Investigators. Non-invasive diagnosis of coronary artery disease by quantitative stress echocardiography: optimal diagnostic models using off-line tissue Doppler in the MYDISE study. Eur Heart J. 2003; 24:1584-94.
28. Agarwal R, Gosain P, Kirkpatrick JN, Alyousef T, Doukky R, Singh G, Umscheid CA. Tissue doppler imaging for diagnosis of coronary artery

disease: a systematic review and meta-analysis. Cardiovasc Ultrasound. 2012;10:47.

29. Hoffmann S, Mogelvang R, Olsen NT, Sogaard P, Fritz-Hansen T, Bech J, Galatius S, Madsen JK, Jensen JS. Tissue Doppler echocardiography reveals distinct patterns of impaired myocardial velocities in different degrees of coronary artery disease. Eur J Echocardiogr. 2010; 11: 544-9.
30. Fennira S, Ben Moussa F, Ellouze Y, Kraiem S, Slimane ML. Pulse-wave Doppler Tissue Imaging in the assessment of regional left ventricular diastolic function in patients with coronary artery disease. Tunis Med 2010; 88: 492-6. [Article in French]

Archive of SID