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ORIGINAL ARTICLE

Evaluation of Myocardial Function by Pulsed Tissue Doppler in Kawasaki Disease

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Abstract Myocarditis is a well-recognized component of Kawasaki disease, with left ventricular dysfunction occurring in more than half of patients during the acute phase. The purpose of this study was to evaluate myocardial function in patients with Kawasaki disease using pulsed tissue Doppler imaging (TDI). Twenty-five patients with the diagnosis of acute Kawasaki disease were enrolled in the study. All patients underwent echocardiographic studies at the time of diagnosis of the disease, in its acute phase, prior to treatment, and then 4 weeks later. For an aged-matched control group with fever and no cardiac disease, the same echocardiographic evaluations were performed. Peak velocities of systolic (Sa), early diastolic (Ea), and late diastolic (Aa) motion of the annulus were obtained at the lateral and septal sides in apical four-chamber view, and TDI-derived myocardial performance index (TDI-MPI) was also calculated. Peak Ea velocity of lateral mitral annulus was decreased significantly during the acute phase of illness (14 ± 4.40 vs. 17.67 ± 4.41; $P = 0.028$). In seven patients with carditis, changes in Ea-to-Aa ratio of septum (1.28 ± 0.278 vs. 1.78 ± 0.49; $P = 0.018$) and lateral mitral annulus (1.23 ± 0.496 vs. 2.11 ± 0.822; $P = 0.014$) were statistically significant but TDI-MPI showed no statistically significant changes. This study showed that peak mitral annular Ea velocities obtained by TDI were significantly altered in the acute phase of Kawasaki disease. TDI- MPI does not add an incremental benefit to other indexes of myocardial performance for comprehensive myocardial function in the acute phase of Kawasaki disease.

Keywords Kawasaki disease · Myocarditis · Pulsed tissue Doppler imaging

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Kawasaki disease (KD) is an acute febrile, systemic vasculitis syndrome with an unknown etiology that occurs primarily in children under 5 years of age [1]. The principal presentations of KD are fever, bilateral nonexudative conjunctivitis, erythema of the lips, cervical lymphadenopathy, and skin rashes [2].

Coronary artery aneurysm develops in 15–25% of untreated children with this disease, which may later lead to ischemic heart disease [1]. Myocarditis manifested by tachycardia and decreased ventricular function occurs in 50% of patients [3]. Pulsed tissue Doppler imaging (TDI) is a novel technique for direct measurement of myocardial velocity. TDI can provide important information about systolic and diastolic function of the heart, not possible through conventional two-dimensional and Doppler echocardiography [4].

The Tei index (the sum of the isovolumetric contraction and relaxation times divided by the ejection time), obtained from tissue Doppler echocardiography (TDE-Tei index), modified from the Tei index by the pulsed-Doppler method [5], has the inherent advantage of measuring both systolic and diastolic components simultaneously and directly during the same cardiac cycle. Therefore, beat-to-beat variations can be avoided. Several previous studies have demonstrated that there is a moderate to strong correlation between TDE-Tei index and Tei index obtained by the

pulsed-Doppler method both in healthy subjects and in patients with heart diseases ($r = 0.62$–0.96) [6, 7]. The TDE-Tei index correlates with accepted indexes of left ventricular (LV) systolic and diastolic function acquired by cardiac catheterization, respectively [8, 9].

The purpose of this study is to evaluate myocardial function in patients with KD in the acute phase of illness using pulsed TDE.

Patients and Methods

Patients

In this study 25 patients with the diagnosis of acute KD, based on American Heart association (AHA) criteria, who were admitted to the hospitals affiliated with Shiraz University of Medical Science, Iran (Nemazee and Dastgheib hospitals), during a period of 1 year (December of 2007 to December of 2008) were included.

Study Protocol

The study was approved by the research committee of the university, and written informed consents were obtained from patients' guardians. Patients underwent echocardiographic studies first at the time of diagnosis of the disease, during its acute phase, prior to treatment with intravenous immunoglobulin (IVIG), and then 4 weeks after the onset of the acute episode. Age-matched pediatric patients underwent the same echocardiographic studies as the control group; control-group children had no heart disease and were examined during a febrile illness.

Echocardiographic Examination

Echocardiography was performed with a GE Vivid 3 echocardiographic machine, using a 3-MHz probe with TDI software. TDI was obtained with the sample volume placed at the lateral corner of the mitral annulus and, subsequently, on the medial (or septal) corner from the apical four-chamber view. In each region, systolic (S) wave, early diastolic (Ea), and late diastolic (Aa) velocities and ejection time (ET) were recorded. Also, the isovolumetric contraction time (ICT) and the isovolumetric relaxation time (IRT) were measured from the end of the mitral annular velocity pattern to the onset of the S-wave and from the end of the S wave to the onset of the mitral annular velocity pattern, respectively. The TDI-derived Tei index (TDI-Tei), sum of ICT and IRT divided by ET, was determined for the lateral mitral annulus (Fig. 1). Time interval measurements were obtained from three consecutive beats, and then the data were averaged to obtain the mean value.

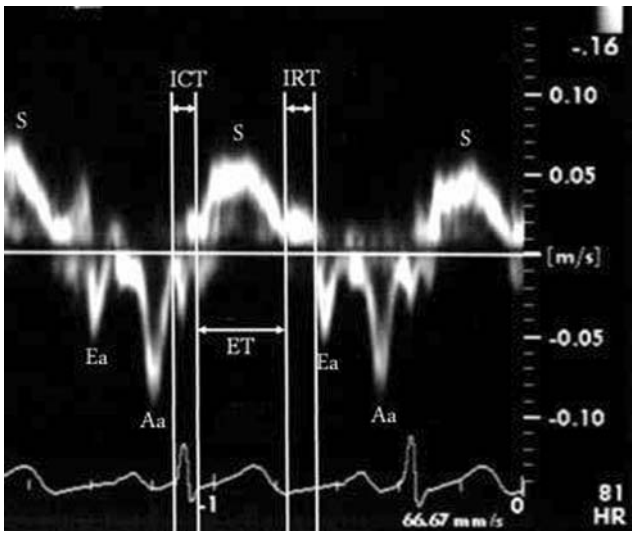

Fig. 1 Time intervals measured from pulsed tissue Doppler (ICT = tissue isovolumic contraction time; ET = ejection time; IRT = tissue isovolumic contraction time). TDE-Tei index is calculated as (ICT + IRT)/ET

Statistical Analysis

All data are expressed as mean ± 1 standard deviation (SD); data were compared by paired-sample *t*-test and Mann–Whitney test, and a P value < 0.05 was considered statistically significant. SPSS version 15 statistical software was used for all statistical analyses.

Results

Comparison of the results of TDI in patients in the acute phase of KD and 4 weeks after treatment with IVIG is presented in Table 1. Velocities of early diastolic flow of the mitral annulus (Ea_M) and early diastolic flow of the septum (Ea_S) in the acute phase were significantly lower than those 4 weeks after treatment (14 ± 4.40 vs. 17.67 ± 4.41, $P = 0.028$, and 12.35 ± 3.06 vs. 13.93 ± 1.58, $P = 0.025$, respectively).

The results of TDI of patients in the acute phase of disease and the febrile control group are presented in Table 2. Ea_M velocities (14 ± 4.40 vs. 17.13 ± 3.20; $P = 0.16$) showed statistically significant differences between these two groups.

In addition, TDI of patients 4 weeks after treatment of the disease and the febrile control group were obtained (Table 3). A statistically significant difference was seen in ET but no significant differences were noted in the velocities between the two groups.

Five patients in the acute phase had tachycardia more than expected due to fever alone, pericardial effusion, or significant valvular regurgitation (signs of carditis) and two

Table 1 TDI parameters in KD patents (group 1) in the acute phase and 4 weeks after receiving immunoglobulin (group 2)

	Group 1	Group 2	*P*-value
SS	7.45 ± 1.932	8.20 ± 1.424	0.08
ES	12.35 ± 3.066	13.93 ± 1.580	0.025
AS	8.05 ± 2.235	8.67 ± 1.543	0.240
SM	7.95 ± 2.305	8.87 ± 1.885	0.114
EM	14.00 ± 4.401	17.67 ± 4.419	0.027
AM	8.70 ± 3.114	9.60 ± 2.947	0.364
ET	216.75 ± 45.348	229.73 ± 38.588	0.542
ICT	65.30 ± 21.404	67.87 ± 22.087	0.586
IRT	64.15 ± 14.982	55.33 ± 16.855	0.106
EF	72.65 ± 7.989	67.60 ± 5.501	0.080
SF	36.65 ± 6.260	35.27 ± 5.147	0.282
HR	114.65 ± 29.231	105.33 ± 13.028	0.050
TDI-Tei	0.622 ± 0.175	0.5454 ± 0.105	0.168
E/A mitral	1.806 ± 0.832	1.9903 ± 0.685	0.364
E/A septum	1.613 ± 0.488	1.6715 ± 0.440	0.705

TDI Tissue Doppler imaging, *KS* Kawasaki disease, *SS* velocity of systolic wave of septum, *ES* velocity of early diastolic flow of septum, *AS* velocity of A wave of septum, *SM* velocity of systolic wave of mitral annulus, *EM* velocity of early diastolic flow of mitral annulus, *AM* velocity of A wave of mitral annulus, *ET* ejection time, *ICT* isovolumic contraction time of mitral annulus, *IRT* isovolumic relaxation time of mitral annulus, *EF* ejection fraction, *SF* shortening fraction, *HR* heart rate, *TDI-Tei* pulsed TDI-derived Tei index

Table 2 TDI parameters in KD patents (group 1) in the acute phase and in febrile control patients (group 3)

	Group 1	Group 3	*P*-value
SS	7.45 ± 1.932	7.94 ± 1.569	0.290
ES	12.35 ± 3.066	12.81 ± 2.105	0.421
AS	8.05 ± 2.235	8.06 ± 2.594	0.648
SM	7.95 ± 2.305	8.31 ± 1.922	0.440
EM	14.00 ± 4.401	17.13 ± 3.202	0.016
AM	8.70 ± 3.114	8.94 ± 3.623	0.962
ET	216.75 ± 45.348	219.69 ± 51.672	0.765
ICT	65.30 ± 21.404	59.38 ± 18.424	0.741
IRT	64.15 ± 14.982	54.63 ± 14.028	0.049
EF	72.65 ± 7.989	75.20 ± 6.723	0.302
SF	36.65 ± 6.260	43.00 ± 6.000	0.060
HR	114.65 ± 29.231	122.50 ± 40.223	0.694
TDI-Tei	0.622 ± 0.175	0.5259 ± 0.09106	0.057
E/A mitral	1.806 ± 0.832	2.199 ± 0.825	0.131
E/A septum	1.613 ± 0.488	1.717 ± 0.509	0.335

TDI Tissue Doppler imaging, *KS* Kawasaki disease, *SS* velocity of systolic wave of septum, *ES* velocity of early diastolic flow of septum, *AS* velocity of A wave of septum, *SM* velocity of systolic wave of mitral annulus, *EM* velocity of early diastolic flow of mitral annulus, *AM* velocity of A wave of mitral annulus, *ET* ejection time, *ICT* isovolumic contraction time of mitral annulus, *IRT* isovolumic relaxation time of mitral annulus, *EF* ejection fraction, *SF* shortening fraction, *HR* heart rate, *TDI-Tei* pulsed TDI-derived Tei index

Table 3 TDI parameters in KD patients 4 weeks after treatment (group 2) and in febrile control patients (group 3)

	Group 2	Group 3	*P*-value
SS	8.20 ± 1.424	7.94 ± 1.569	0.653
ES	13.93 ± 1.580	12.81 ± 2.105	0.065
AS	8.67 ± 1.543	8.06 ± 2.594	0.188
SM	8.87 ± 1.885	8.31 ± 1.922	0.494
EM	17.67 ± 4.419	17.13 ± 3.202	0.519
AM	29.60 ± 2.947	8.94 ± 3.623	0.264
ET	229.73 ± 38.588	219.69 ± 51.672	0.830
ICT	67.87 ± 22.087	59.38 ± 18.424	0.337
IRT	55.33 ± 16.855	54.63 ± 14.028	0.770
EF	67.60 ± 5.501	75.20 ± 6.723	0.098
SF	35.27 ± 5.147	43.00 ± 6.000	0.140
HR	105.33 ± 13.028	122.50 ± 40.223	0.048
TDI-Tei	0.545 ± 0.105	0.525 ± 0.091	0.740
E/A mitral	1.990 ± 0.685	2.199 ± 0.825	0.337
E/A septum	1.671 ± 0.440	1.717 ± 0.509	0.770

TDI Tissue Doppler imaging, *KS* Kawasaki disease, *SS* velocity of systolic wave of septum, *ES* velocity of early diastolic flow of septum, *AS* velocity of A wave of septum, *SM* velocity of systolic wave of mitral annulus, *EM* velocity of early diastolic flow of mitral annulus, *AM* velocity of A wave of mitral annulus, *ET* ejection time, *ICT* isovolumic contraction time of mitral annulus, *IRT* isovolumic relaxation time of mitral annulus, *EF* ejection fraction, *SF* shortening fraction, *HR* heart rate, *TDI-Tei* pulsed TDI-derived Tei index

patients had coronary artery involvement (one had enhanced echo brightness of the soft tissue surrounding the coronary artery lumen that regressed after 4 weeks, and the other had two aneurysms with diameters of 5 and 3 mm on the left main coronary artery and right coronary artery, respectively, that persisted after 4 weeks). The results of TDI in these patients were compared with those in the other 13 patients without carditis, and the data are reported in Table 4. The velocity of Ea_M (11.71 ± 1 5.438, 15.23 ± 3.345), Aa_s wave of septum (9.43 ± 2.225, 7.31 ± 1.932), Ea_S-to-Aa_S ratio (1.28 ± 0.278, 1.78 ± 0.493), Ea_M-to-Aa_M ratio (1.23 ± 0.496, 2.11 ± 0.822), ET (239.00 ± 25.492, 175.43 ± 46.342), and ICT 57.43 ± 23.95, 69.54 ± 19.56) revealed significant differences.

Percentage ejection fraction and fractional shortening in the three groups showed no statistically significant differences ($P > 0.05$) as reported in Tables 1, 2, 3, 4. Only two patients in the acute phase had fractional shortening <28% that subsided after 4 weeks of treatment with IVIG.

Discussion

Cardiac involvement is the most important manifestation of KD. Myocarditis, pericarditis, coronary artery aneurysms, and valvular regurgitation are the major cardiac

Table 4 Results of TDI in patients with signs of carditis compared with other patients (no carditis) in the acute phase of KD (no carditis)

Parameter	Carditis	No carditis	*P*-value
SS	8.14 ± 2.795	7.08 ± 1.256	0.588
ES	12.29 ± 4.608	12.38 ± 2.063	0.392
AS	9.43 ± 2.225	7.31 ± 1.932	0.045
SM	8.00 ± 3.317	7.92 ± 1.706	0.757
EM	11.71 ± 5.438	15.23 ± 3.345	0.045
AM	10.14 ± 3.436	7.92 ± 2.753	0.114
S-D	175.43 ± 46.342	239.00 ± 25.492	0.003
ICT	57.43 ± 23.957	69.54 ± 19.564	0.081
IRT	62.86 ± 8.112	64.85 ± 17.916	0.588
EF	69.29 ± 10.372	74.46 ± 6.091	0.392
SF	34.86 ± 7.515	37.62 ± 5.561	0.392
HR	146.14 ± 26.617	97.69 ± 10.419	0.000
TDI-Tei	0.725 ± 0.215	0.566 ± 0.126	0.096
E/A mitral	1.231 ± 0.496	2.115 ± 0.822	0.014
E/A septum	1.288 ± 0.278	1.789 ± 0.493	0.018

TDI Tissue Doppler imaging, *KS* Kawasaki disease, *SS* velocity of systolic wave of septum, *ES* velocity of early diastolic flow of septum, *AS* velocity of A wave of septum, *SM* velocity of systolic wave of mitral annulus, *EM* velocity of early diastolic flow of mitral annulus, *AM* velocity of A wave of mitral annulus, *S-D* ejection time of mitral annulus, *ET* ejection time, *ICT* isovolumic contraction time of mitral annulus, *IRT* isovolumic relaxation time of mitral annulus, *EF* ejection fraction, *SF* shortening fraction, *HR* heart rate, *TDI-Tei* pulsed TDI-derived Tei index

manifestations of KD [2]. Myocarditis, manifested by tachycardia and decreased ventricular function, occurs in at least 50% of patients during the acute phase of the disease [3]. Echocardiography obtained in the acute phase may show a small amount of pericardial effusion, mild LV dilatation, and a decrease in systolic function indexes such as fractional shortening in more than 50% of patients [3, 10]. There may also be mitral regurgitation and, rarely, aortic root dilation, which occurs within the first 3 weeks of the disease and persists during the first year of follow-up; mild aortic regurgitation is seen in 4% of cases [11]. Rarely, the patient can have heart failure with markedly reduced LV function. These acute changes, which are due to myocarditis, tend to improve spontaneously as the degree of systemic inflammation subsides; rapid clinical and echocardiographic improvements occur with IVIG therapy [3]. In the current study 28% (7 of 25) of the patients had carditis and only two patients had fractional shortening <27% in the acute phase of KD, which may be due to small sample size. Other modalities can increase the rate of detection of myocarditis in KD, for example, endocardial and cardiac biopsies of patients have provided histological evidence of myocarditis in the acute phase of KD [12, 13]. Antibody titers to human cardiac myosin are significantly elevated in patients with KD [14]. About 80% of patients with KD showed myocarditis in the acute phase, using gallium-67 myocardial imaging with single-photon emission computed tomography [15], and 57% of patients in the acute phase of KD had myocarditis detected by ^{99m}Tc-HMPAO-labeled WBS scan [16–18]. Noninvasive stress-shortening and stress-velocity analysis using echocardiograms revealed that more than half of patients with KD had abnormal contractility at the time of clinical presentation [3].

TDI is a relatively recent method for assessment of cardiac function that provides direct, local measurements of myocardial velocities throughout the cardiac cycle with ultrasound waves [4]. In this study, a decrease in velocity of the early diastolic flow of the mitral annulus (Ea_M) showed a statistically significant difference in the acute phase of illness in comparison with the same data obtained 4 weeks after treatment and the findings in the febrile control group. This result shows that the myocarditis of KD, and not the fever, is the cause of these changes. Patients with carditis have a statistically significant decrease in Ea-to-Aa velocity ratio.

In a study by Jang et al. [19] that evaluated patients with KD, peak velocities of systolic (Sa), early diastolic (Ea), and late diastolic (Aa) motion of the mitral annulus were obtained. The results showed a decrease of Sa and Ea in patients with KD. In another study, by Daiji et al., that evaluated TDI changes and plasma brain natriuretic peptide and increased oxidative stress in KD showed a statistically significant decrease in Ea in the first week of KD compared with controls, which subsequently normalized during the convalescent stage [20].

The Tei index obtained by the pulsed-Doppler method has been found to be a reproducible and simple index for assessing global LV function, and it correlates closely with invasive measurements of LV systolic and diastolic function [8, 9]. However, its components are sometimes obtained sequentially from different heartbeats if mitral inflow and LV ejection signals cannot be clearly recorded simultaneously, and its application may be limited by heart rate fluctuations.

In contrast, the TDE-Tei index has the inherent advantage of recording the systolic and diastolic velocity signals simultaneously during the same cardiac cycle. Therefore, beat-to-beat variations can be avoided. Some previous studies have demonstrated that the TDE-Tei index correlates well with the Tei index obtained by the pulsed-Doppler method [7, 21]. Su et al. [9] demonstrated fair correlations between the TDE-Tei index and the accepted indexes of LV diastolic and systolic function acquired from cardiac catheterization. In contrast, some studies have shown that the TDE-Tei index recorded at the mitral annulus does not seem to be a suitable substitute for the traditional LV Tei index as a noninvasive indicator of

combined systolic and diastolic myocardial performance [22].

This study shows that the tissue Doppler-derived Tei index cannot evaluate the diastolic dysfunction that occurs in the acute phase of KD. The results of our study suggest that in the acute phase of KD there is a statistically significant decrease in Ea velocity that subsequently normalizes during the convalescent stage, it is correlated with carditis in this disease, and the TDE-Tei index cannot evaluate systolic and diastolic changes in the acute phase of the disease.

Limitations of the Study

The relatively small sample size may be a limitation of this study; hopefully, this report may initiate more research in this field.

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