Study The Role Of Tissue Doppler Imaging During Dobutamine Stress Echocardiography In Differentiating Of Ischemic From Non-Ischemic Cases Of Dilated Cardiomyopathy

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Abstract

Background: Dobutamine stress echocardiography (DSE) has several potential advantages over currently used non-invasive technique. It is inexpensive and provides an alternative in the patients who cannot perform leg or arm exercise. The equipment used is highly portable and thus studies can be performed in coronary care units.

Objectives: To study the role of tissue Doppler imaging during dobutamine stress echocardiography for differentiating ischemic from non-ischemic cardiomyopathy.

Patients and methods: forty six of patients were included in this study, and diagnosed as dilated cardiomyopathy by resting conventional echocardiography. The patients divided into two groups group I (18 patients) with normal coronary angiography, and group II(28 patients) with coronary artery disease. All patients subjected for full history taking, clinical examination, resting surface electrocardiograms, echocardiography, low and peak dose DSE associated with tissue Doppler with detection of mitral annulus systolic and diastolic velocities in six sectors of LV myocardium (anterior, inferior, anterior septum, posterior septum, posterior and lateral walls).

Results: In group I, there is no change in the mean value of systolic velocities of all six sectors at baseline was (6.5±1.6 cm/s) and at low dose was (6.9±2.3 cm/s) p value > 0.05, while in group II there is significant improvement in the mean value of systolic velocities of six sectors at low dose (11.3±3.1 cm/s) compared to that in the baseline resting stat (7.0±1.6 cm/s) followed by gradual decrease in the systolic velocity to peak dose (9.1±2.6 cm/s) (Biphasic response) p value < 0.01. The E'/A' ratio of diastolic function estimated by TDI at base line and during DSE, there is insignificant changes in both groups. The sensitivity of tissue Doppler with DSE for detection of coronary artery disease in patient with dilated cardiomyopathy was 85.7%, and specificity was 77.8%.

Conclusion: Tissue Doppler with DSE is non invasive simple method with high sensitivity and specificity for detection of coronary artery disease in patient with dilated cardiomyopathy.
Introduction

Dobutamine stress echocardiography (DSE) has several potential advantages over currently used non-invasive technique. It is inexpensive and provides an alternative in the patients who cannot perform leg or arm exercise. The equipment used is highly portable and thus studies can be performed in coronary care units. (1).

High quality images from DSE may be obtained more easily than those with exercise stress echocardiography because the absence of patient motion and respiratory inference, and the level of stress achieved can be controlled and the heart rate achieved can be suppressed by B-Blockers (2).

Aim of the work

To study the role of tissue Doppler imaging during dobutamine stress echocardiography for differentiation of ischemic from non-ischemic cases of dilated cardiomyopathy.

Patients and methods

This study was carried out in the cardiology department of both Benha faculty of medicine and Ain Shams faculty of medicine during the period from August 2008 to Jun 2010.

Inclusion criteria:

Included in this study, patients who have documented echocardiography diagnosis of dilated cardiomyopathy: dilated LV with generalized hypokinesis, posterior displaced mitral valve, estimated LV end diastolic diameter > 60mm and percentage ejection fraction < 45% (3)

All of those patients underwent left sided cardiac catheterization via femoral artery puncture using Seldenger's technique with left and right coronary angiography in multiple projections and angulations. According to the results of the diagnostic coronary angiography, patients were divided into two groups:

Group I :( 18 patients) with dilated cardiomyopathy and normal coronary arteries.
Group II (28 patients) with dilated cardiomyopathy and significant narrowing of one or more coronary arteries. Significant narrowing was defined as > 70% reduction in the absolute lumen diameter of a major epicardial artery or major branch vessel. (4)

**Exclusion criteria:**

1- Evidence of previous myocardial infarction.
2- Significant arrhythmias.
3- Valvular or pericardial disease.
4- Uncontrolled hypertension or diabetes mellitus.
5- History of chronic obstructive airway disease.
6- Poor echocardiography window.
7- Echocardiography evidence of left ventricular hypertrophy.
8- Decompensate heart failure.
9- Patients not complete the test due to early development of complications.

**Methods:** All patients were subjected to:

1) Careful history taking.
2) Thorough clinical examination.
3) Resting electro-cardiogram: 12 leads surface resting electrocardiogram.
4) Echocardiography study:

   Trans-thoracic echocardiography examination using Vivid 5 machine and probe 3S frequency 1.5 – 3.6 MHz GE Probe was done for all subjects enrolled in the study.

M-mode, two dimensional as well as color and pulsed Doppler flow examination were done in supine and left lateral position. According to the American Society of Echocardiography guide lines the following measurements were obtained:
1) Left atrial diameter.
2) End diastolic and end systolic LV cavity dimensions and volumes. Left ventricular Ejection fraction (EF) was then calculated according to the equation: 
\[ \text{EF} \% = \frac{(\text{LV diastolic volume} - \text{LV systolic volume})}{\text{LV diastolic volume}}. \]

Using apical 4-chamber view, pulsed wave Doppler sample was put at the tips of mitral valve leaflets to record the mitral flow velocities, E and A waves. The ratio of E/A was then calculated.

**Dobutamine stress Echocardiography:**

Adequate arrangement of the testing equipment, with availability of appropriate emergency equipment, dobutamine stress echocardiography was performed to all patients in the fasting state; nitrates were stopped 24 hours before the test while beta blockers and calcium channel blockers were stopped 48 hours before the test. Dobutamine with intravenous infusion started by rate \(5 \text{ ug/kg/min. for 5 minutes increased gradually 5 ug/kg/min. every 3 minutes up to maximum dose 40 ug/kg/min.}\)

In patients not achieving 85% of their maximum heart rate without symptoms or signs of myocardial ischemia, atropine was administered intravenously; starting dose was 0.25 mg up to maximum of 2.0mg within 8 minutes while continuing infusion of dobutamine. In patients not achieving 85% of their maximum heart rate without symptoms or signs of myocardial ischemia, atropine was administered intravenously; starting dose was 0.25 mg up to maximum of 2.0mg within 8 minutes while continuing infusion of dobutamine. (6). Throughout dobutamine infusion, continuous ECG monitoring was done and at each stage heart rate and blood pressure were measured, left ventricular wall motion was evaluated with two dimensional echocardiography and mitral annular motion velocity patterns were recorded with pulsed TDI at resting and at a maximum dose of 40 ug/kg/min (7)

**End points of the test:**

- Significant chest pain.
- Extensive new wall motion abnormalities.
- Achievement of 85% of maximum predicted target heart rate.
- Additional ST segment depression or elevation about 2.0mm in at least two contiguous leads compared to the rest.

- Hypotension: systolic blood pressure < 90mm Hg or reduction more than 40mmHg of pretest level or increased systolic pressure >200.

- Significant arrhythmia.

  **Dobutamine stress echocardiography (DSE)** was considered positive for induced myocardial ischemia after detection of **biphasic response** that means at low dose of stress the contractile function improved and at higher doses the myocardial demand increased leading to ischemia and systolic function deterioration again.(8)

**Pulsed wave tissue Doppler imaging study:**

The echocardiography machine was switched to TDI mode thereby resulting in lowering of the velocity range to encode myocardial velocities.

The acoustic power and filter frequencies of the ultrasound scan system were set to the lowest values possible. Sample volumes were set at the mitral annulus. Mitral annular motion velocities were recorded on a strip chart.

On the known basis that systolic mitral annular motion velocities at 6 mitral annular sites reflect the synergy at the sites corresponding to the ischemic regions in patients with myocardial ischemia, sample volumes were set on the endocardial side at the mitral annulus in regions corresponding to:

- Anterior mitral, posterior mitral, inferior, anterior, lateral, and posterior walls. The peak velocity of the systolic mitral annular motion (S) were determined, also the peak diastolic velocities (E', A’) were determined from six consecutive beats and the mean was calculated for each parameter and expressed in centimeters per second. Recording and measurements were repeated at baseline, low dose (5ug /kg/min) and maximum dobutamine infusion rate.

**Statistical analysis**

Data were collected from total patients (group I and II), and expressed as expressed as the mean value, standard deviation and percentages.
Kruskal – Wallis test was used to compare different parameters between groups. When inter-group differences were found, Mann-Whitney test was performed to determine which groups were significantly different.

Categorical variables were analyzed with the Chi square test. Paired student’s t-test was used to compare different parameters of DSE in studied groups.

A value of P < 0.05 was considered statistically significant. (9)

Results

Demographic data: Forty six patients included at this study their mean age was (42.5 ± 7.1) in group I versus (59.4 ± 6.9) in group II, 11 of them were males and 7 females in group I versus 17 males and 11 females in group II.

Seven patients (38.9%) were diabetic in group I versus 18 (64.3%) in group II, 5 patients hypertensive (27.8%) in group I versus 15 (53.5%) in group II, 8 patients (44.4%) were smoker in group I versus 15 (53.5%) in group II, 7 patients (38.9) had dyslipidemia in group I versus 17(60.7) in group II, and only 3 patients (16.7%) had positive family history on group I versus 11(39.3%) in group II.

Table (1): comparisons between studied groups in demographic data

<table>
<thead>
<tr>
<th>variable</th>
<th>Group I(N=18)</th>
<th>Group II(N=28)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean value)</td>
<td>42.5 ± 7.1</td>
<td>59.4 ± 6.9</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Male</td>
<td>11(61.1%)</td>
<td>17(60.7%)</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Female</td>
<td>7 (38.9%)</td>
<td>11(39.3%)</td>
<td></td>
</tr>
<tr>
<td>Diabetics</td>
<td>7 (38.9%)</td>
<td>18 (64.3%)</td>
<td>0.032</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>5 (27.8%)</td>
<td>15 (53.5%)</td>
<td>0.039</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>7 (38.9%)</td>
<td>17 (60.7%)</td>
<td>0.042</td>
</tr>
<tr>
<td>Smokers</td>
<td>8 (44.4%)</td>
<td>15 (53.5%)</td>
<td>0.086</td>
</tr>
<tr>
<td>+ve Family history</td>
<td>3 (16.7%)</td>
<td>11 (39.3%)</td>
<td>0.023</td>
</tr>
</tbody>
</table>
**Conventional echocardiography response during dobutamine stress:**

In group I EF was \((35 \pm 3.5)\) with low dose DSE and nearly the same with high dose DSE, and in group II EF was \((45.2 \pm 4.1)\) with low dose DSE, while it was \((41.4 \pm 4.6)\) in high dose DSE, with statistically significant difference was found between both groups \((p\ value < 0.01)\). This means that in non-ischemic cardiomyopathy, dobutamine stress induces no recordable change in global systolic function from the low dose to peak dose (mono-phase response). On the other hand, in ischemic cardiomyopathy there was an initial improvement at low dose followed by impairment of systolic function (Biphasic response). Fig. (1).

![Graph showing variation of ejection fraction during DSE in both studied groups.](image)

*Figure (1): variation of ejection fraction during DSE in both studied groups.*

**LV diastolic function (E/A ratio):**

In group I E/A ratio was \((1.12 \pm 0.31)\) during resting state, \((1.1\pm0.27)\) at low dose DSE and became \((1.05\pm0.26)\) at maximum dose DSE, and in group II E/A ratio was \((0.97\pm0.26)\) during resting state, \((0.91\pm0.27)\) at low dose DSE and became \((0.85 \pm 0.46)\) at maximum dose DSE, with statistically significant difference was found between both groups \((P\ value < 0.01)\). These result means that in non-ischemic cardiomyopathy, there is insignificant change in E/A ratio during dobutamine stress, while in ischemic group there is significant decrease in this ratio, most probably due to stress induced myocardial ischemia. Fig. (2).
Figure (2): variation of diastolic function during DSE in both studied groups.

**Tissue Doppler parameters during dobutamine stress echocardiography:**

* **Systolic velocities during DSE in group I:** there is insignificant monophasic response in all six sectors. *(P value >0.05) (Tab.2, Fig. 3)*

- **In anterior aspect** S at baseline was (6.6 ±1.9) cm/s, at low dose dobutamine (6.8 ±2.1) cm/s while S at peak dose became (7.1 ±3.1) cm/s.

- **In inferior aspect** S at baseline was (6.4 ±1.7) cm/s, S at low dose (6.6 ±2.8) cm/s while S at peak dose became (7.2 ±2.3) cm/s.

- **In posterior aspect** S at baseline was (5.5 ±1.9) cm/s, S at low dose (5.9 ±2.5) cm/s while S at peak dose became (6.2 ±3.1) cm/s.

- **In anterior septal aspect** S at baseline was (5.9 ±1.7) cm/s, S at low dose dobutamine was (6.1 ±2.4) cm/s while S at peak dose became (6.4 ±2.8) cm/s.

- **In posterior septal aspect** S at baseline was (7.0 ±1.9) cm/s, S at low dose dobutamine was (8.2 ±2.1) cm/s while S at peak dose became (9.1 ±3.5) cm/s.

- **In lateral aspect** S at baseline was (7.7 ±1.4) cm/s, S at low dose dobutamine was (8.3 ±1.9) cm/s while S at peak dose became (8.6 ±2.1) cm/s.

* **Diastolic velocities E'/A’ ratio during DSE in group I:** The mean value of diastolic velocity (E'/A') in group I at baseline was (1.0 ±0.3) cm/s, at low dose dobutamine (1.0 ±0.2) cm/s, and became (0.9±0.3) cm/s at high dose. *(P value > 0.05)*
Table (2): Tissue Doppler parameters during DSE in group I.

<table>
<thead>
<tr>
<th></th>
<th>Resting</th>
<th>Low dose</th>
<th>High dose</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior wall</td>
<td>6.6±1.9</td>
<td>6.8±2.1</td>
<td>7.1±3.1</td>
<td>0.22</td>
</tr>
<tr>
<td>Inferior wall</td>
<td>6.4±1.7</td>
<td>6.6±2.8</td>
<td>7.2±2.3</td>
<td>0.10</td>
</tr>
<tr>
<td>Posterior wall</td>
<td>5.5±1.9</td>
<td>5.9±2.5</td>
<td>6.2±3.1</td>
<td>0.19</td>
</tr>
<tr>
<td>Anterior septal</td>
<td>5.9±1.7</td>
<td>6.1±2.4</td>
<td>6.4±2.8</td>
<td>0.09</td>
</tr>
<tr>
<td>Posterior septal</td>
<td>7.0±1.9</td>
<td>8.2±2.1</td>
<td>9.1±3.5</td>
<td>0.12</td>
</tr>
<tr>
<td>Lateral wall</td>
<td>7.7±1.4</td>
<td>8.3±1.9</td>
<td>8.6±2.1</td>
<td>0.18</td>
</tr>
<tr>
<td>E′/A′ ratio</td>
<td>1.0±0.3</td>
<td>1.0±0.2</td>
<td>0.9±0.3</td>
<td>0.07</td>
</tr>
</tbody>
</table>

Figure (3): systolic velocities in all myocardial aspects during DSE in group I.

* Systolic velocities of myocardial aspects during DSE in group II: there is biphasic response with significant changes in all six sectors. (P value < 0.01) (Tab.3, Fig.4)

- In anterior aspect S at baseline was (7.4 ± 1.3) cm/s, S at low dose dobutamine was (14.6 ±4.7) cm/s, and became (9.1 ± 3.1) cm/s at high dose.

- In inferior aspect S at baseline was (7.4 ±1.9) cm/s, S at low dose dobutamine was (12.3±3.6) cm/s, and became at peak dose (10.7 ±2.8) cm/s.
- **In posterior aspect** S at baseline was \((5.3 \pm 1.1)\) cm/s, S at low dose dobutamine was \((10.4 \pm 2.7)\) cm/s while became at peak dose \((7.8 \pm 2.5)\) cm/s.

- **In anterior septal aspect** S at baseline was \((5.6 \pm 1.6)\) cm/s, at low dose dobutamine was \((9.6 \pm 3.1)\) cm/s while S at peak dose of dobutamine was \((7.2 \pm 2.7)\) cm/s.

- **In posterior septal aspect** S at baseline was \((7.6 \pm 1.2)\) cm/s, at low dose dobutamine was \((11.9 \pm 3.8)\) cm/s while S at peak dose of dobutamine was \((8.9 \pm 2.9)\) cm/s.

- **In lateral aspect** S at baseline was \((8.7 \pm 1.3)\) cm/s, at low dose dobutamine was \((15.2 \pm 3.9)\) cm/s while S at peak dose of dobutamine was \((10.4 \pm 2.3)\) cm/s.

* **Diastolic velocities \(E'\)/\(A'\) ratio in Group II**: there is insignificant changes during DSE; the mean value at baseline was \((0.93 \pm 0.3)\) cm/s, at low dose \((97.0 \pm 0.2)\) cm/s while S at peak dose became \((0.96 \pm 0.2)\) cm/s. (P value > 0.05)

**Table (3): Tissue Doppler parameters during DSE in group II.**

<table>
<thead>
<tr>
<th></th>
<th>Resting</th>
<th>Low dose</th>
<th>High dose</th>
<th>p. value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior wall</td>
<td>7.4±1.3</td>
<td>14.6±1.7</td>
<td>9.1±3.1</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Inferior wall</td>
<td>7.4±1.9</td>
<td>12.3±3.6</td>
<td>10.7±2.8</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Posterior wall</td>
<td>5.3±1.1</td>
<td>10.4±2.7</td>
<td>7.8±2.5</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Anterior septal</td>
<td>5.6±1.6</td>
<td>9.6±3.1</td>
<td>7.2±2.7</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Posterior septal</td>
<td>7.6±1.2</td>
<td>11.9±3.8</td>
<td>8.9±2.9</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Lateral wall</td>
<td>8.7±1.3</td>
<td>15.2±3.9</td>
<td>10.4±2.3</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Mean value E/A ratio</td>
<td>0.93±0.3</td>
<td>0.97±0.2</td>
<td>0.96±0.2</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>
Figure (4): Systolic velocities in all myocardial aspects during DSE in group II.

Complications during dobutamine stress echocardiography:

-Patient excluding from the test due to development of complications:

1- Two patients developed significant hypotension.

2- Three patients developed rapid atrial fibrillation.

3- One patient developed runs of ventricular tachycardia.

Specificity and sensitivity Dobutamine Stress Echocardiography:

- The test was positive for stress induced myocardial ischemia in 28 patients, 23 of them was true positive while 5 patients was false positive, also the test was negative for 18 patients, 13 of them was true negative while 5 patients was false negative.

- The specificity of the test was 72.2% and the sensitivity was 82.1%.

Table (4): Specificity and sensitivity of DSE.

<table>
<thead>
<tr>
<th>DSE</th>
<th>All cases</th>
<th>True cases</th>
<th>False cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ve cases</td>
<td>28 patients</td>
<td>23 patients</td>
<td>5 patients</td>
</tr>
<tr>
<td>-ve cases</td>
<td>18 patients</td>
<td>13 patients</td>
<td>5 patients</td>
</tr>
<tr>
<td>Specificity</td>
<td>72.2%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>sensitivity</td>
<td>82.1%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Specificity and sensitivity of TDI with DSE according to systolic velocity:

- The test was positive for stress induced myocardial ischemia in 28 patients, 24 of them was true positive and only 4 patients were falsely negative, also the test was negative in 18 patients, 14 of them were true and only 4 were false.

- The specificity of the test was 77.8% and the sensitivity was 85.7%.

Table (5): Specificity and sensitivity of TDI with DSE:

<table>
<thead>
<tr>
<th>DSE</th>
<th>All cases</th>
<th>True cases</th>
<th>False cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ve cases</td>
<td>28 patients</td>
<td>24 patients</td>
<td>4 patients</td>
</tr>
<tr>
<td>-ve cases</td>
<td>18 patients</td>
<td>14 patients</td>
<td>4 patients</td>
</tr>
<tr>
<td>Specificity</td>
<td>77.8%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sensitivity</td>
<td>85.7%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Fig. (5) Tissue Doppler parameters on patient No (1)
Discussion

Tissue Doppler imaging, obtained from the apical windows during dobutamine stress, offers a quantitative and highly sensitive mean for measuring systolic and diastolic alterations due to myocardial ischemia (10).

The present study aimed to compare the changes in myocardial velocities as reflected by measuring annular mitral velocities at rest and during dobutamine stress in ischemic and non-ischemic cardiomyopathy and thus define criteria for differentiating between them.

For this purpose, in the present study, forty-six patients with dilated cardiomyopathy had been evaluated by TDI at rest and during dobutamine stress echocardiography. All patients underwent coronary angiography within one week before the stress test. According to the results of coronary angiography, the patients were classified into two groups; group I who had normal coronary arteries and group II who had significant coronary artery disease.

In the present work, group II patient were significantly older than group I patients (mean age in years = 59.4 ± 6.9 versus 45 ± 7.1, p<0.05) (table 1)

In contrary, Vogel et al. (2003) reported that there is no significant difference in age in ischemic cardiomyopathic patients and idiopathic cardioyomyopathic patients (56±6.3ys versus 53±4ys). (11)

In present study, there is a significant increase in the prevalence of diabetes in group II (18) (64.3%) versus (7) patients (38.9%) in group I (P < 0.05). (Table 1)

Peter, et al. (2001) agrees with this, who found a higher prevalence of diabetes in patients with ischemic cardiomyopathy (22%) than those who had non-ischemic cardiomyopathy (5%). (12)

In the present study there is a higher prevalence of dyslipidamia in group II than in group I (60.7% versus 38.9%, p< 0.05). (Table 1)
This is in accord with Vogel et al. (2003) who found a higher prevalence of hypercholesterolemia in patients with ischemic cardiomyopathy than in patients with non-ischemic cardiomyopathy (62% Vs 18%), p <0.05. (11)

In the present study, atropine was used to reach the target stress level in 10 cases (55.5%) of group I and in 12 (42.8%) cases of group II.

This finding is in agreement with that of Rambaldi et al. (1998) who found that 47% of group I needed addition of atropine compared to 45% of the group II. (13)

In the current study, the global systolic function as calculated by EF in both groups, there was nearly no changes in group I while in group II there is initial increase then decrease in EF from low dose to peak dose (Biphasic response) (fig.1).

Pauliks et al. (2005) detect that there is an increase in global LV systolic function in low dose dobutamine stress echocardiography in ischemic cardiomyopathy that decrease again with high doses dobutamine stress. (14)

As regard diastolic parameters during dobutamine stress in the present work, there is insignificant decrease in the E/A ratio in group I while there is significant decrease in that ratio in group II (Fig. 2)

This is agree with Alison et al. (2003), who found that marked deterioration of diastolic function in ischemic than in idiopathic cardiomyopathic patient during dobutamine stress echocardiography. This deterioration of diastolic function may be due to the increase of myocardial rigidity during stress as a result of ischemia in group II. (5)

But this result not agree with, Hanekom et al. (2005), who reported that there is insignificant changes of diastolic function during dobutamine stress echocardiography in ischemic and idiopathic cardiomyopathy. (15)

In the present study the peak systolic velocities in tissue Doppler during DSE study in group I, at the six sectors show insignificant and monophasic response. (Fig.3, Table 2)
While there is significant increase in systolic velocity at low dose compared to that in
the baseline resting stat and followed by gradual decrease in the systolic velocity toward
the peak dose in all six sectors (Biphasic response). (Fig.4, Table 3)

This in accord with Peteiro et al. (2001) who used pulsed tissue Doppler imaging
during dobutamine - atropine stress testing to detect significant coronary lesions and they
found that at rest peak systolic velocity in territories supplied by diseased artery was
insignificantly less than that of non-ischemic cardiomyopathy but with low stress the
peak systolic velocity was significantly higher in ischemic cardiomyopathy. (16)

This in agreement with those results of Elzaky et al., (2005), who found that
systolic velocities as assessed by pulsed tissue Doppler imaging were significantly
reduced in ischemic region when compared with the corresponding walls in healthy
patients and high in ischemic patient when compared with myopathic patients. (17)

On the other hand Tsutsi et al. (1998) demonstrated that pulse-wave Doppler
tissue imaging at rest could not differentiate between ischemic cardiomyopathy and non-
ischemic cardiomyopathy segments depending on the peak systolic velocity. This
difference may be related to different demographic data and involvement of multi-vessel
disease in CAD group. (18)

Also the results of current study are in accord with Alison et al., (2003) who used
dobutamine tissue Doppler with measurement of mitral trans-annular systolic velocities,
the study showed a reduction in systolic velocities in non-ischemic myopathic patients
compared to CAD group. (5)

The diastolic parameters of tissue Doppler detected by E'/A' in the present work
were evaluated during dobutamine stress, we found in group I, the E'/A' ratio at rest was
1.0±0.3, at low dose it was 1.0±0.2 and at peak dose it was 0.9±0.3 (Table 2). This means
that there is insignificant decrease in E'/A' ratio in this group with non-ischemic
 cardiomyopathy.
Similarly, in group II, the E'/A' ratio during the test were insignificant changes as in group I (Table 3).

So, there were no changes in diastolic parameters as estimated by TDI during dobutamine stress in both groups.

This may be explained by non-linear changes in diastolic myocardial velocities in response to ischemia; the ratio may increase with restrictive response or decreased with abnormal relaxation response. The different responses of diastolic function may be related to the amount of myocardial fibrosis and level of end-diastolic pressure which is depending on patient age and duration of disease.

Similar conclusions were obtained by Alison et al. (2003), who found that diastolic velocities as assessed by pulsed tissue Doppler imaging were insignificantly reduced in ischemic region when compared with the corresponding walls in idiopathic cardiomyopathic patients. (5)

Contradictory results were obtained by Elzaky et al., (2005) who found that diastolic velocities as assessed by pulsed tissue Doppler imaging were significantly reduced in ischemic region when compared with the corresponding walls in healthy patients and high in ischemic patient when compared with other myopathic patients. (17)

Hegazy et al., (2007) concluded that mitral trans-annular systolic and diastolic velocities were an indicator for predicting significant coronary stenosis, their study revealed that the impaired TDI derived variables; S, E', A' and E'/A' ratio, were an indicator for CAD. (19)

In the present study the sensitivity and specificity of TDI during DSE in prediction of coronary artery disease in myopathic patients were significantly higher than that of the conventional dobutamine stress echocardiography; it was in tissue Doppler 85.7% versus 82.1% in conventional echocardiography, specificity was 77.8% in TDI versus 72.2%.(Table 4).
This is in accord to Alison et al. (2003) who found that, tissue Doppler imaging with dobutamine stress echocardiography identified 41 of 48 of myopathic patients with CAD and 22 of 25 without CAD with a sensitivity 85% and specificity 88%. (5)

The difference in values between present study and other studies may be attributed to the differences age, gender, and other risk factors like hypertension, diabetes mellitus, and dyslipidemia, also maybe due to different degrees of diameter stenosis, and various combinations of ischemic, hibernating, and infarcted myocardium.

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