Significance of ST-segment elevation during dobutamine-stress echocardiography in patients with acute myocardial infarction treated with thrombolysis

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Background Stress-induced ST-segment elevation in patients with recent myocardial infarction treated with thrombolysis has not been extensively investigated. According to the results of previous studies it may represent residual myocardial ischaemia or dyskinesia in the infarcted region. The aim of the study was to analyse the significance of dobutamine-induced ST-segment elevation in the infarcted area in a consecutive group of patients (n=42, 41 men, mean age 53 ± 7 years) with a first acute myocardial infarction treated with thrombolysis within 6 h from symptoms onset.

Methods and results All patients underwent dobutamine-stress echocardiography (up to 40 μg . kg⁻¹ . min⁻¹ + atropine) 7 ± 3 days from the acute event and coronary arteriography within 1 month from the test. Significant ST-segment elevation was defined as a shift ≥ 1 mm during dobutamine compared to baseline in at least two contiguous infarct-related leads; a correlation was made between the site of ST-segment elevation and wall motion changes during dobutamine. Dobutamine-induced ST-segment elevation in 23/42 (55%) patients (group 1) while no changes were observed in 19/23 (45%) patients (group 2). Compared to group 2, group 1 patients showed a higher asynergy score index (1-72 ± 0-24 vs 1-50 ± 0-32, P<0-02) and a higher number of asynergic segments (504 ± 1-9 vs 411 ± 1-8), at baseline, a higher incidence of baseline and/or stress-induced dyskinesia (39 vs 10%, P<0-05) in the infarct-related region and a higher percentage of occluded infarct-related arteries (48 vs 0%, P<0-001). In the 42 patients studied, a significant correlation was found between baseline ST-segment elevation and baseline asynergy score index (RS=0-56, P<0-001) and between ST-segment elevation and asynergy score index at peak stress (RS=0-55, P<0-001). The incidence of reversible wall motion abnormalities indicative of myocardial viability and residual myocardial ischaemia was similar in the two groups (87 vs 84% and 74 vs 68%, respectively), while the number of segments with irreversible akinesia indicative of myocardial necrosis was higher in group 1 compared to group 2 (1-5 ± 1-4 vs 0-9 ± 1-4). Among the 23 patients of group 1 with dobutamine-induced ST-segment elevation, six had no reversible wall motion abnormalities indicative of myocardial ischaemia; of the 17 patients with myocardial ischaemia, 11 had ≥ 50% and six had ≤ 50% of basally asynergic segments showing reversible wall motion abnormalities.

Conclusions In patients with recent thrombolysed myocardial infarction dobutamine-induced ST-segment elevation is associated with a larger akinetic area in basal conditions and either with reversible wall motion abnormalities indicative of myocardial ischaemia or with irreversible or minimally reversible wall motion abnormalities in the infarct area during the test. Thus, dobutamine echocardiography provides useful information for the interpretation of stress-induced ST-segment elevation and clinical management of these patients.

Key Words: Dobutamine-echo stress test, ST-segment elevation, recent myocardial infarction.

Introduction

The meaning of stress-induced ST-segment elevation after a recent myocardial infarction is still controversial.


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It has been previously demonstrated that in patients with recent myocardial infarction not treated with thrombolysis, transient ST-segment elevation during stress electrocardiography is related to dyskinesia of an infarcted myocardial wall and is usually associated with a significant impairment of global and regional left ventricular function due to extensive myocardial damage. However, residual myocardial ischaemia has been postulated to be the cause of ST-segment...
elevation by some authors since it was associated with reversible thallium defects within the infarcted area and was abolished by myocardial revascularization. Moreover, stress-induced ST-segment elevation has not been extensively investigated in patients treated with systemic thrombolysis. Thrombolysis may prevent the necrosis of a significant amount of myocardium within the area at risk, so that residual myocardial viability and ischaemia are frequently present after thrombolysis in acute myocardial infarction. Dobutamine stress echocardiography has been recently proposed as a useful and accurate technique for diagnosis of coronary artery disease and assessment of both myocardial viability and ischaemia in the infarct zone after thrombolysis. So this test may represent a useful method to establish whether ST-segment elevation after myocardial infarction is related to residual myocardial ischaemia or dyskinesia within the infarcted area. Thus, we have undertaken a study to assess the clinical, echocardiographic and angiographic correlates of dobutamine-induced ST-segment elevation in patients with thrombolyzed acute myocardial infarction evaluated in the early post-infarction period.

**Methods**

**Patient selection**

Forty-two consecutive patients (41 men, mean age 53 ± 7 years) fulfilling the following criteria were selected for the study:

1. First acute myocardial infarction diagnosed on the basis of typical chest pain lasting >30 min not relieved by nitrates, acute evolutionary ST-segment changes and an increase in CPK and CK-MB myocardial enzymes at least twice normal values with subsequent return to baseline values.
2. Treatment with systemic thrombolysis within 6 h from beginning of symptoms.
3. Age <70 years.
4. No major arrhythmias or moderate-to-severe systemic hypertension contra-indicating dobutamine stress testing.
5. A good echocardiographic window in basal conditions.

Patients with in-hospital complications including post-infarction angina, left ventricular failure, major ventricular arrhythmias as well as those with left bundle branch block or permanent pacemaker were excluded from the study.

**Dobutamine echocardiography**

The test was performed at a mean of 7 ± 3 days after acute myocardial infarction. All patients gave their informed consent to the study. Treatment with antianginal drugs was not usually withdrawn; 11/42 patients (26%) were treated with beta-adrenergic blocking drugs while 9/42 (21%) were taking long-acting nitrates and/or calcium-antagonists. Dobutamine was administered intravenously by an infusion pump at the initial dosages of 5 and 10 μg . kg⁻¹ . min⁻¹ for 5 min each, followed by increments of 10 μg . kg⁻¹ . min⁻¹ every 3 min up to a maximal dose of 40 μg . kg⁻¹ . min⁻¹. In 18/42 patients (43%) who did not reach 85% of the maximum age-predicted heart rate during the test, atropine (up to 1 mg intravenously over 4 min) was administered. End-points of the test were achievement of the maximal dose or of 85% of maximal age-predicted heart rate, new or worsening wall motion abnormality, >2 mm ST-segment depression compared to baseline, severe angina, sustained ventricular or supraventricular arrhythmias, systemic hypertension (blood pressure >230/120 mmHg) or hypotension (defined as a drop in systolic pressure >20 mmHg compared to the previous step) or other significant side-effects. During the test, a continuous low-speed (5 mm . s⁻¹) six-lead electrocardiographic recording was carried out; at the end of each step a 12-lead electrocardiogram at a paper speed of 25 mm . s⁻¹, blood pressure and heart rate were recorded. When needed the precordial leads were positioned one intercostal space below the standard position for better application of the echo transducer. Echocardiographic images were obtained with commercially available equipment (Hewlett-Packard Sonos 1000). Four standard views of the left ventricle (parasternal long- and short-axis, apical four- and two-chamber views) were recorded at baseline, at each step of the test and during recovery. All examinations were recorded on videotape for subsequent analysis.

**Echocardiographic analysis**

All examinations were reviewed by two independent observers blinded to the clinical data of the patients. In case of disagreement, a consensus was reached. For left ventricular wall motion analysis a previously described 11-segment model of the left ventricle was used. A semi-quantitative scoring system (1 = normal wall motion and thickening, 2 = hypokinesis, 3 = akinesis, 4 = dyskinesia) was used. An echocardiographic left ventricular asynergy score index, defined as the sum of the scores of the 11 segments divided by total segments considered, was calculated in basal conditions, at low doses (10 μg . kg⁻¹ . min⁻¹) and at peak doses of dobutamine. The echocardiographic site of myocardial infarction was defined by correlating the site of asynergy with that of electrocardiographic changes during the actual phase. In order to correlate electrocardiographic and echocardiographic changes, precordial electrocardiographic leads from V1 to V5 were assigned to anteroseptal, anterior and apical segments; peripheral leads II, III and aVF to inferior segments and I, aVL and precordial V6 to lateral segments.

Myocardial viability was considered to be present in the infarct zone when low-dose
dobutamine systolic wall thickening and endocardial motion appeared in a basally akinetic or dyskinetic segment (from a score equal to 3 or 4 to a score 1 or 2), or normal or near-normal wall thickening and motion became apparent in a previously hypokinetic segment (from a score 2 to a score 1) included in the infarct zone defined according to the previously described criteria. Significant myocardial viability in the infarct zone was defined as an improvement in wall motion and thickening in at least two segments or in at least one segment when only two segments were basally asynergic.

Dobutamine echocardiography was considered to be indicative of myocardial ischaemia in the infarct zone when: (1) a basally akinetic or severely hypokinetic segment in the infarct area, after improving its thickening and motion at low doses, showed a significant deterioration at higher doses; (2) a hypokinetic segment during dobutamine infusion subsequently deteriorated to dyskinesia at higher doses. Akinesia deteriorating directly to dyskinesia was not considered indicative of myocardial ischaemia.

Electrocardiographic analysis

With the PR segment as the isoelectric line, ST-segment elevation was measured at 0.08 s from the J point in the infarct-related leads both in basal conditions and during stress. During dobutamine stress test an increase in ST-segment elevation \( \geq 1 \text{ mm} \) compared to baseline in at least two contiguous infarct-related leads was considered to be significant. The difference between the ST-segment shift at baseline and at peak stress in the infarct-related lead with the highest shift was calculated (Delta ST). A correlation was made between the site of ST-segment changes and the site of wall motion changes during dobutamine infusion.

Coronary angiography

All patients underwent coronary angiography by Judkins' technique and left ventricular angiography within 1 month from acute myocardial infarction. Multiple views of each coronary artery were filmed. A coronary stenosis was considered significant by qualitative analysis when the vessel diameter was narrowed by \( \geq 50\% \). The infarct-related artery was identified as the left anterior descending coronary artery for anterior infarction and the left circumflex artery or right coronary artery for inferior, lateral or infero-lateral infarction.

Statistical analysis

Continuous variables were compared using Student's t-test for paired data or one-way analysis of variance (ANOVA) for repeated measures when appropriate. When a statistically significant difference was found with one-way ANOVA, individual comparisons were made with Scheffe's test. A chi-square test was used for comparison of categorical variables. Correlations between numeric data were calculated by Spearman rank (RS) correlation coefficient. A \( P \) value of \( <0.05 \) was considered statistically significant. All data are expressed as mean \( \pm \) 1 standard deviation.

Results

Haemodynamic and electrocardiographic response to dobutamine

Heart rate increased from a basal value of 66 \( \pm \) 11 to a peak value of 112 \( \pm \) 24 beats . min\(^{-1} \) (\( P<0.001 \)), systolic blood pressure from 123 \( \pm \) 17 to 150 \( \pm \) 37 mmHg (\( P<0.001 \)) and rate-pressure product from 8202 \( \pm \) 1803 to 16836 \( \pm \) 4928 mmHg . beats . min\(^{-1} \) (\( P<0.001 \)). No major complications were induced by dobutamine. Dobutamine stress test was prematurely interrupted in only two patients (5%) because of an increase of systolic blood pressure >240 mmHg.

The test induced a new or worsening asynergy in the infarct zone indicative of residual myocardial ischaemia in 32/42 patients (76%). Significant electrocardiographic changes developed in 39/42 patients (93%): 23 had ST-segment elevation in the infarct-related leads, eight had \( >1 \text{ mm} \) ST-segment depression in leads different from those related to the infarct and eight had negative T-wave positivization. During the test three patients (7%) complained of angina.

Dobutamine-induced ST-segment elevation

On the basis of the electrocardiographic response to dobutamine stress test, two groups of patients were identified: group 1 (\( n=23 \); 55%) with dobutamine-induced ST-segment elevation in the infarct-related leads and group 2 (\( n=19 \); 45%) without ST-segment elevation during the test. The characteristics of the two groups of patients are illustrated in Table 1. They were similar in relation to sex distribution, mean age, site of acute myocardial infarction, percentage of Q wave infarction, number of pathological Q waves, and peak level of total CK enzyme distribution. The incidence of a \( >1 \text{ mm} \) ST-segment elevation in the resting electrocardiogram was significantly higher in group 1 compared to group 2 (78 vs 42%, \( P<0.05 \)). Table 2 shows the echocardiographic parameters of the two groups at baseline and during dobutamine stress echocardiography. Compared to group 2, group 1 patients also showed a significantly
Table 1  Clinical and electrocardiographic characteristics of patients with (group 1) and without (group 2) dobutamine-induced ST-segment elevation

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
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<tbody>
<tr>
<td>Number of patients</td>
<td>23 (55%)</td>
<td>19 (45%)</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>22/1</td>
<td>19/0</td>
</tr>
<tr>
<td>Mean age (years)</td>
<td>54 ± 7</td>
<td>52 ± 7</td>
</tr>
<tr>
<td>Site of myocardial infarction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior/inferior</td>
<td>14/9</td>
<td>10/9</td>
</tr>
<tr>
<td>Peak creatine kinase (mIU. ml⁻¹)</td>
<td>2370 ± 1321</td>
<td>2385 ± 1719</td>
</tr>
<tr>
<td>Q-wave infarction</td>
<td>20/23</td>
<td>13/19</td>
</tr>
<tr>
<td>Number of Q waves</td>
<td>3 ± 45</td>
<td>2 ± 77</td>
</tr>
<tr>
<td>Baseline ST elevation (mm)</td>
<td>18/23</td>
<td>5 ± 19</td>
</tr>
</tbody>
</table>

*P<0.05 vs group 1.

Table 2  Echocardiographic parameters and results of dobutamine-echo stress test in patients with (group 1) and without (group 2) dobutamine-induced ST-segment elevation

|                          | Group 1 (n=23) | Group 2 (n=19)
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Asynergy score index</td>
<td></td>
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</tr>
<tr>
<td>Baseline</td>
<td>1·72 ± 0·24</td>
<td>1·50 ± 0·32*</td>
</tr>
<tr>
<td>Low dose dobutamine</td>
<td>1·46 ± 0·26</td>
<td>1·26 ± 0·29*</td>
</tr>
<tr>
<td>High dose dobutamine</td>
<td>1·84 ± 0·36</td>
<td>1·58 ± 0·35</td>
</tr>
<tr>
<td>Number of basally asynergic segments</td>
<td>5·04 ± 1·9</td>
<td>4·11 ± 1·8</td>
</tr>
<tr>
<td>Number of ischaemic segments at peak stress</td>
<td>3·41 ± 1·9</td>
<td>3·43 ± 1·7</td>
</tr>
<tr>
<td>Dyskinesia at baseline and/or during stress</td>
<td>9/23 (39%)</td>
<td>2/19 (10%)*</td>
</tr>
<tr>
<td>Viability at low-dose dobutamine</td>
<td>20/23 (87%)</td>
<td>16/19 (84%)</td>
</tr>
<tr>
<td>Ischaemia at high dose dobutamine</td>
<td>17/23 (74%)</td>
<td>13/19 (68%)</td>
</tr>
<tr>
<td>Angina during stress</td>
<td>2/23 (9%)</td>
<td>1/19 (5%)</td>
</tr>
<tr>
<td>Mean dobutamine dose infused (µg. kg⁻¹)</td>
<td>35 ± 7</td>
<td>35 ± 7</td>
</tr>
</tbody>
</table>

*P<0.02 vs group 1; †P<0.05 vs group 1.

higher asynergy score index at baseline (1·72 ± 0·24 vs 1·50 ± 0·32, P<0·02) and a higher number of basally asynergic segments in the infarct-related region (5·04 ± 1·9 vs 4·11 ± 1·8). Compared to group 2, group 1 patients showed a higher incidence of baseline and/or stress-induced dyskinesia in the infarct region (39 vs 10%, P<0·05). At baseline, dyskinesia was present in one group 1 patient and in no patients in group 2; stress-induced dyskinesia was detected in eight patients in group 1 and in two group 2 patients. In both groups the asynergy score index significantly improved after low-dose dobutamine and subsequently deteriorated at higher doses of dobutamine. Myocardial viability and myocardial ischaemia in the infarct area were detected in a similar proportion of patients in the two groups (87 vs 84% and 74 vs 68%, respectively). The intake of medications did not significantly limit the detection of myocardial viability and ischaemia in patients under treatment or off treatment (100 vs 73% and 80 vs 64%, respectively). The number of segments showing reversible wall motion abnormalities indicative of myocardial viability and ischaemia was similar in the two groups (3·4 ± 1·7 vs 3·4 ± 1·7), while the number of segments with irreversible akinesia indicative of myocardial necrosis was higher in group 1 compared to group 2 (1·5 ± 1·4 vs 0·9 ± 1·4). Among the 23 patients of group 1 with dobutamine-induced ST-segment elevation, the majority of patients (12/23, 52%) had no peri-infarction ischaemia (six patients) or peri-infarction ischaemia involving less than 50% of basally asynergic segments (six patients).

The incidence of angina, the mean dose of dobutamine infused (Table 2), the heart rate, the systolic blood pressure and the double product at baseline and at peak stress (Table 3) were similar in the two groups.

Relation between ST-segment elevation and regional wall motion

In the 42 patients studied, a significant linear correlation was found between baseline ST-segment elevation and baseline asynergy score index (RS=0·56, P<0·001) (Fig. 1).

Regression analysis also showed a significant correlation between peak ST-segment elevation and maximal asynergy score index during dobutamine.
Table 3 Haemodynamic parameters during dobutamine-echo stress test in patients with (group 1) and without (group 2) dobutamine-induced ST-segment elevation

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n = 23)</th>
<th>Group 2 (n=19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats . min⁻¹)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>68 ± 10</td>
<td>65 ± 12</td>
</tr>
<tr>
<td>Peak stress</td>
<td>112 ± 23*</td>
<td>115 ± 25*</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>122 ± 15</td>
<td>125 ± 20</td>
</tr>
<tr>
<td>Peak stress</td>
<td>144 ± 34*</td>
<td>159 ± 41*</td>
</tr>
<tr>
<td>Double product (mmHg . beats . min⁻¹)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>8300 ± 1780</td>
<td>8085 ± 1870</td>
</tr>
<tr>
<td>Peak stress</td>
<td>16 020 ± 4740*</td>
<td>17 825 ± 5100*</td>
</tr>
</tbody>
</table>

*P<0-001 vs baseline.

Figure 1 Linear correlation between baseline ST-segment elevation and baseline asynergy score index in the 42 patients studied. A significant direct linear correlation was found between the two parameters (RS=0-56, P<0-001).

Figure 2 Linear correlation between peak ST-segment elevation and asynergy score index at peak stress in the 42 patients studied. A significant direct linear correlation was found between the two parameters (RS=0-55, P<0-001).

In the pre-thrombolytic era stress-induced ST-segment elevation after myocardial infarction was usually interpreted as a marker of left ventricular dysfunction and extensive myocardial damage not related to ischaemia. However, according to other studies this electrocardiographic response to exercise stress is associated with reversible thallium perfusion defects in the infarct area and is abolished by coronary revascularization and may therefore be considered indicative of residual myocardial ischaemia. Recently, Margonato studied 25 patients with myocardial infarction within 6 months from the acute event with exercise thallium-201 scintigraphy. Among the 17 patients (68%) who developed ST-segment elevation during stress, 16 (94%) demonstrated reversible perfusion defects in the infarcted region indicative of tissue viability and residual ischaemia compared to only four out of the eight patients without ST-segment elevation. These results are at variance with those reported by Dunn and by Lahiri who found reversible ischaemia on thallium-201 scintigraphy in only 52% and 36%, respectively of patients with exercise-induced ST-segment elevation.

In our study the prevalence of dobutamine-induced ST-segment elevation was 55%. This figure is lower than the value of 68% reported by Margonato with exercise and that of 75 and 85% reported by Coma-Canella with dobutamine. This difference may be due to the fact that 100% of our patients, but only 30-60% of the patients in the reported studies were treated with systemic thrombolysis which can reduce infarct size, extent of wall motion abnormalities and therefore the incidence of ST-segment elevation.

In our study dobutamine-induced ST-segment elevation was associated with a significantly larger infarct size as documented by the higher asynergy score index and the higher number of basally asynergic segments in patients with, compared to those without, dobutamine-induced ST-segment elevation. Also the prevalence of dyskinesia in the infarct zone was significantly higher (39 vs 10%, P<0-05) in patients with, compared to those without, dobutamine-induced ST-segment elevation. In the patients with stress-induced dyskinesia, a progressive deterioration of wall motion...
from baseline akinesia to dyskinesia developed during the test. This finding is in keeping with the results of Arnese and probably reflects the passive systolic bulging of the necrotic segments that may generate stress-induced ST-segment elevation.

We found a significant positive correlation between the degree of ST-segment elevation and the asynergy score index both at baseline and at peak stress. On the other hand no significant correlation was found between Delta ST and the difference between stress and baseline asynergy score index that reflects the extent of peri-infarction ischaemia. Also, other authors recently found a significant correlation between ST-elevation and thallium defect score both at rest and during stress in a group of 88 patients with recent myocardial infarction undergoing dobutamine stress test; in this study stress-induced ST-segment elevation was associated with a smaller increase in regional ejection fraction, reflecting a more severe asynergy of necrotic segments.

In our study myocardial viability and ischaemia in the infarct area were detected in a similar percentage of patients in the two groups. The incidence of ischaemic wall motion abnormalities in our patients with ST-segment elevation was similar to that of peri-infarction redistribution at thallium scintigraphy (74 vs 72%) found by Coma-Canella in a comparable group of patients. Among the 23 patients with ST-segment elevation, six had no evidence of reversible asynergy in the infarcted zone and 17 showed reversible wall motion abnormalities indicative of peri-infarction ischaemia. Of the 17 patients with reversible asynergy, six patients had ≤50% and 11 ≥50% of basally asynergic segments showing peri-infarction ischaemia. These findings suggest that in patients with recent myocardial infarction dobutamine-induced ST-segment elevation may be associated both with irreversible and reversible ischaemia-induced wall motion abnormalities in the peri-infarction area and cannot be considered a specific marker of myocardial ischaemia.

A possible limitation of the study is that 47% of the patients were treated with anti-anginal drugs that can modify the echocardiographic and electrocardiographic signs of viability and ischaemia; however the proportion of patients with myocardial viability or ischaemia was similar in the group of patients treated or not with anti-anginal drugs. Compared with patients without ST-segment elevation during dobutamine stress those with ST-segment elevation showed a significantly higher percentage of occlusion of the infarct-related artery (52 vs 0%). Although in the minority (4/11, 36%) of patients with an occluded infarct-related artery and a well developed collateral circulation stress-induced ST-segment elevation could be caused by myocardial ischaemia, the majority of these patients (7/11, 64%) had an occluded coronary artery without a significant collateral blood supply which may favour infarct expansion, post-infarction remodelling and stress-induced systolic bulging of the necrotic segments.

Clinical implications

Our study suggests that stress-induced ST-segment elevation after a recent myocardial infarction treated with thrombolysis is not a specific marker of ischaemia at echocardiography. When ST-segment elevation is associated with a large akinetic area and irreversible or only minimally reversible wall motion abnormalities in the infarct area and no other signs of myocardial ischaemia are present, the test cannot be considered positive for residual myocardial ischaemia in the infarct zone. On the other hand, the evidence of a small area of irreversible akinesia and of a significant amount of myocardium in the infarct area showing reversible wall motion abnormalities associated with ST-segment elevation should be considered indicative of significant residual myocardial ischaemia. In these patients myocardial revascularization has been shown to abolish stress-induced ST-segment elevation and seems indicated, therefore, to prevent myocardial ischaemia and improve left ventricular regional function.
References


