Comparison of exercise and pharmacological stress gated SPECT in detecting transient left ventricular dysfunction

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Abstract

Objective Transient left ventricular contractile dysfunction (TLVD) is observed owing to post-exercise stunning in patients with coronary artery disease (CAD). Pharmacological stimulation differs from exercise stress because it does not cause demand ischemia. The aim of this study was to determine whether TLVD could also be seen after pharmacological stress (dipyridamole).

Methods Of the patients in whom gated single-photon emission computed tomography (GSPECT) was performed in our institution from January 2004 to April 2007, 439 subjects with known or suspected CAD were included in the study. GSPECT was performed for all patients following exercise (group I, n = 220) or pharmacological stress (group II, n = 219) according to a 2-day (stress–rest) protocol after injection of Tc-99m methoxyisobutylisonitrile (MIBI). Stress, rest, and difference (stress–rest value) left ventricular ejection fractions (SLVEF, RLVEF, and DLVEF) and transient ischemic dilatation (TID) ratio were derived automatically. Summed stress score, summed rest score, and summed difference score (SDS) for myocardial perfusion were calculated using a 20-segment model and a five-point scoring system. An SDS > 3 was considered as ischemic. On the basis of the perfusion findings, patients were subdivided into a normal (group A, n = 216) and ischemia group (group B, n = 223). DLVEF and perfusion scores of all groups were compared. Relationships between DLVEF and perfusion, and between TID ratio and DLVEF were also evaluated.

Results Stress-induced ischemia was observed in 223 of 439 patients (50.8%). In group A, the difference between stress and rest LVEF values was not significant (P = 0.670 and P = 0.200 for groups IA and IIA, respectively). However, LVEF was significantly decreased after stress compared with rest values for group B (P < 0.0001 for groups IB and IIB). TLVD (≤−5% for DLVEF) was observed in 20 of 216 (9%) and 81 of 223 subjects (36%) in patients in groups A and B, respectively (P < 0.0001). In group I, we found TLVD in 46 of 119 (39%) and 12 of 101 (12%) subjects, in patients with and without ischemia, respectively (P < 0.0001). On the other hand, in group II, TLVD was detected in 35 of 104 (34%) and 8 of 115 (7%) patients with and without ischemia, respectively (P < 0.0001). And also, we found significant good correlations between TID ratios and DLVEF values in four subgroups (r = −0.55, r = −0.62, r = −0.59, and r = −0.41; for groups IA, IB, IIA, and IIB, respectively, P < 0.0001 for all).

Conclusions Dipyridamole is believed to be less likely than exercise to induce ischemia. However, in this study, TLVD after stress was observed following not only exercise but also pharmacological stress, consistent with ischemia.

Keywords Myocardial stunning · Gated SPECT · Transient left ventricular dysfunction · Tc-99m MIBI

Introduction

In patients with coronary artery disease (CAD) transient left ventricular contractile dysfunction (TLVD) after exercise is frequently observed despite myocardial
perfusion returning to resting levels. This phenomenon of reversible impairment in left ventricular function has been well described and is known as myocardial stunning [1].

Gated single-photon emission computed tomography (GSPECT) is able to detect post-exercise stunning [2]. GSPECT offers the potential advantage of combining information about myocardial function and perfusion without any extra cost, discomfort or radiation risk to the patient [3–7]. In addition, according to several earlier studies, TLVD [wall motion abnormalities, left ventricular ejection fraction (LVEF), and volume impairments] in post-stress GSPECT images are well correlated with myocardial perfusion abnormalities and degree of stenotic vessels in coronary angiography in patients with CAD [8–11]. Pharmacological stimulation differs from exercise stress in that it does not cause demand ischemia. The number of studies that have compared exercise and pharmacological stress effects on TLVD are limited in number and conflicting [2, 11–15]. Therefore, the objective of this study was to determine whether TLVD could also be seen after pharmacological stress (dipyridamole).

Materials and methods

Patients

Of the patients in whom GSPECT was performed in our institution from January 2004 to April 2007, 439 patients (193 men and 246 women, mean age 59.5 ± 10.5 years, range 27–88 years) with known or suspected CAD were included in this retrospective study.

Stress was performed by either treadmill exercise using the Bruce protocol (group I: n = 220; 113 men, 107 women, mean age 56.0 ± 9.6 years) or dipyridamole (group II: n = 219; 80 men and 139 women, mean age 63.0 ± 10.3 years). The characteristics of the patients are listed in Table 1.

Table 1 Patient characteristics

<table>
<thead>
<tr>
<th></th>
<th>Group I (n = 220)</th>
<th>Group II (n = 219)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>56.0 ± 9.6</td>
<td>63.0 ± 10.3</td>
<td>&lt;0.002</td>
</tr>
<tr>
<td>Female</td>
<td>139 (63.5%)</td>
<td>107 (48.6%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>59 (26.8%)</td>
<td>55 (25.1%)</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>126 (57.3%)</td>
<td>122 (55.7%)</td>
<td>NS</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>65 (29.5%)</td>
<td>59 (26.9%)</td>
<td>NS</td>
</tr>
<tr>
<td>Obesity (BMI &gt; 30)</td>
<td>91 (41.4%)</td>
<td>95 (43.4%)</td>
<td>NS</td>
</tr>
<tr>
<td>Smoking</td>
<td>50 (22.7%)</td>
<td>51 (23.3%)</td>
<td>NS</td>
</tr>
<tr>
<td>Family history</td>
<td>42 (19.1%)</td>
<td>46 (21.0%)</td>
<td>NS</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>19 (5.5%)</td>
<td>12 (8.2%)</td>
<td>NS</td>
</tr>
<tr>
<td>PTCA</td>
<td>39 (17.7%)</td>
<td>38 (17.4%)</td>
<td>NS</td>
</tr>
<tr>
<td>CABG</td>
<td>68 (30.9%)</td>
<td>74 (33.8%)</td>
<td>NS</td>
</tr>
</tbody>
</table>

BMI body mass index, NS non-significant, PTCA percutaneous transluminal coronary angioplasty, CABG coronary artery bypass graft surgery

GSPECT

Gated single-photon emission computed tomography was performed using a 2-day (stress–rest) protocol. Beta-blockers, calcium channel antagonists, and long-acting nitrates were discontinued 2 days prior to the GSPECT. On day 1 following pharmacological stress (with dipyridamole) or at peak effort on treadmill exercise, patients were injected with 740 MBq of Tc-99m methoxyisobutylisonitrile (MIBI). Stress acquisition was started after 45 min of Tc-99m MIBI. On day 2, rest acquisitions were performed following an injection of 740 MBq of Tc-99m MIBI under resting conditions. All data acquisitions were performed with a single-head SPECT system (ADAC Laboratories, Milpitas, CA, USA) equipped with a low-energy, high-resolution collimator. A 20% window around the 140 keV energy peak of Tc-99m was used. The acquisition matrix size was 64 × 64 × 16. A total of 64 projections (step-and-shoot mode, 30 s per projection) were obtained over a 180° circular orbit. Acquisitions were gated for eight frames per cardiac cycle. The images were reconstructed using filtered back-projection (Butterworth filter; order 10; cutoff frequency 0.55; pixel size 6.6 mm voxel⁻¹). The resulting transaxial slices were reoriented perpendicular to the heart’s long axis, yielding long- and short-axis tomograms. Images were not corrected for attenuation.

The LVEF, LV volumes, and transient ischemic dilation (TID) ratio were calculated using previously validated and commercially available automated software (Auto SPECT, Autoquant, ADAC Laboratories; developed at Cedars–Sinai Medical Center, Los Angeles, CA, USA) from the GSPECT images. The reproducibility of these measurements has been validated and reported earlier [16, 17]. The algorithm operates in three-dimensional space. It segments the left ventricle, estimates and displays the endocardial and epicardial surfaces for every gating interval, calculates end-systolic volume (ESV) and end-diastolic volume (EDV), and derives the related LVEF by taking their difference (stroke volume) and...
dividing it by EDV. Thereafter, stress (S), rest (R), and difference (D) LVEF values were calculated (DLVEF = SLVEF − RLVEF). The TID ratio was calculated as the ratio of LV chamber volumes of stress and rest studies (LV chamber volume at stress/LV chamber volume at rest).

We considered TLVD when DLVEF was smaller than −5%. This limit has been used by other authors earlier [18]. Non-gated (summed) SPECT images were used for the assessment of semiquantitative myocardial perfusion in 20 segments according to a five-point scoring system (0, normal; 1, slight reduction of uptake; 2, moderate reduction; 3, severe reduction; and 4, absent uptake). Three summed scores were automatically derived: summed stress score (SSS), summed rest scores (SRS), and summed difference score (SDS, the difference between SSS and SRS). Ischemia was recognized if SSS was higher than SRS by more than 3 points (SDS > 3). The DLVEF and perfusion scores of all groups were compared with each other. And also, relationships between DLVEF and perfusion, and between TID ratio and DLVEF were evaluated.

Coronary angiography

Selective left and right coronary angiograms were obtained using Judkins catheters (Siemens Axiom Artis and Philips H3000 systems, ADAC Laboratories). Lesions were considered as significant if they exceeded 50% of the lumen diameter in two projections. Two trained observers evaluated the lesions together. Mostly, visual estimation was used. Quantitative analysis was also done in the case of any suspicion regarding the significance of narrowing. Coronary angiography (CAG) could be available in 74 (33.2%) patients with ischemia.

Statistical analysis

Continuous data were expressed as the mean ± SD and were compared using paired t test, independent samples t test and Mann–Whitney U test when appropriate. Pearson’s correlation coefficients were used to determine the correlation between TID ratios and DLVEF values. A P value of <0.05 was considered to be statistically significant.

Results

On the basis of the perfusion findings, patients were subdivided into a normal group (group A, n = 216) and an ischemia group (group B, n = 223). Thus, totally four groups were formed: group IA, exercise-normal perfusion (n = 101, 44 men and 57 women, mean age 55.7 ± 8.6 years, range 40–78 years); group IIA, dipyridamole-normal perfusion (n = 115, 30 men and 85 women, mean age 62.4 ± 10.7 years, range 38–88 years); group IB, exercise ischemia (n = 119, 50 men and 69 women, mean age 56.3 ± 10.4 years, range 27–82 years); and group IIB, dipyridamole ischemia (n = 104, 54 men and 50 women, mean age 63.7 ± 9.8 years, range 41–85 years).

Stress-induced ischemia was observed in 223 of 439 patients (50.8%). In group I (exercise) 101 patients had normal perfusion (group IA) and 119 patients had ischemia (group IB). On the other hand, in group II (dipyridamole) 115 patients had normal perfusion (group IIA) and 104 patients had ischemia (group IIB). Incidence of ischemia in groups I and II was not different (54.1% and 47.5%, respectively, P = 0.167).

The mean SLVEF, RLVEF, and DLVEF were 64.15 ± 11.78%, 63.98 ± 11.79%, and 0.17 ± 3.93%; 53.24 ± 14.80%, 56.59 ± 15.39%, and −3.35 ± 4.39% for groups A and B, respectively (Table 2). Mean SLVEF, RLVEF and DLVEF obtained for subgroups were 64.52 ± 9.36%, 64.69 ± 9.85%, −0.17 ± 3.96% for group IA; 53.98 ± 13.88%, 57.60 ± 13.28%, −3.61 ± 4.54% for group IB; 63.82 ± 13.59%, 63.35 ± 13.27%, 0.47 ± 3.90% for group IIA; 52.38 ± 15.80%, 55.43 ± 16.56%, −3.05 ± 4.20% for group IIB, respectively (Table 2).

According to these results, for group B DLVEF was significantly decreased after stress compared with rest values (P < 0.0001 for groups B, IB, and IIB). But, in group A the differences between stress and rest LVEF values were not significant (P = 0.523, P = 0.670, and P = 0.200 for groups A, IA, and IIA, respectively; Table 2).

In addition, the difference between the DLVEF values of the normal and ischemic groups was significant for all group and subgroups (exercise and dipyridamole groups; P < 0.0001 for all groups).

Mean TID ratios were given in Table 2. We found significant good correlations between TID ratios and DLVEF values in four subgroups (r = −0.55, r = −0.62, r = −0.59, r = −0.41; for groups IA, IB, IIA, and IIB, respectively, P < 0.0001 for all; Figs. 1, 2, 3, 4).

Transient left ventricular contractile dysfunction was observed in 20 of 216 (9%) patients and in 81 of 223 patients (36%) in groups A and B, respectively. The incidence of TLVD was statistically different for groups A and B (P < 0.0001). In group II, TLVD was detected in 35 of 104 (34%) and 8 of 115 (7%) patients, in subjects with and without ischemia, respectively. Again, the incidence of TLVD was statistically different for groups IA and IB (P < 0.0001). On the other hand, in group I, we found TLVD in 46 of 119 (39%) and 12 of 101 (12%)
patients, in subjects with and without ischemia, respectively. Therefore, the incidence of TLVD was statistically different in groups IIA and IIB ($P < 0.0001$; Fig. 5).

Table 2 Mean of LVEF values and median of perfusion scores

<table>
<thead>
<tr>
<th></th>
<th>All patients</th>
<th>Group I (n = 216)</th>
<th>Group II (n = 223)</th>
<th>Group I (n = 101)</th>
<th>Group II (n = 119)</th>
<th>Group I (n = 115)</th>
<th>Group II (n = 104)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SLVEF (%)</td>
<td>$64.15 \pm 11.78$</td>
<td>$53.24 \pm 14.80$</td>
<td></td>
<td>$64.52 \pm 9.36$</td>
<td>$53.98 \pm 13.88$</td>
<td></td>
<td>$63.82 \pm 13.59$</td>
</tr>
<tr>
<td>RLVEF (%)</td>
<td>$63.98 \pm 11.79$</td>
<td>$56.59 \pm 15.39$</td>
<td></td>
<td>$64.69 \pm 9.85$</td>
<td>$57.60 \pm 13.28$</td>
<td></td>
<td>$63.35 \pm 13.27$</td>
</tr>
<tr>
<td>DLVEF (%)</td>
<td>$0.17 \pm 3.93$</td>
<td>$-3.35 \pm 4.39$</td>
<td></td>
<td>$-0.17 \pm 3.96$</td>
<td>$-3.61 \pm 4.54$</td>
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<td>$0.47 \pm 3.90$</td>
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<tr>
<td>SESV (ml)</td>
<td>$34.01 \pm 25.87$</td>
<td>$56.51 \pm 42.16$</td>
<td></td>
<td>$32.10 \pm 16.82$</td>
<td>$54.84 \pm 41.44$</td>
<td></td>
<td>$35.69 \pm 31.74$</td>
</tr>
<tr>
<td>RESV (ml)</td>
<td>$33.05 \pm 25.75$</td>
<td>$50.64 \pm 41.08$</td>
<td></td>
<td>$31.51 \pm 17.05$</td>
<td>$48.79 \pm 39.82$</td>
<td></td>
<td>$34.39 \pm 31.50$</td>
</tr>
<tr>
<td>DESV (ml)</td>
<td>$0.96 \pm 5.17$</td>
<td>$5.87 \pm 8.77$</td>
<td></td>
<td>$0.58 \pm 4.99$</td>
<td>$6.05 \pm 9.72$</td>
<td></td>
<td>$1.30 \pm 5.32$</td>
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<tr>
<td>SEDV (ml)</td>
<td>$86.09 \pm 31.03$</td>
<td>$109.12 \pm 45.26$</td>
<td></td>
<td>$85.21 \pm 24.55$</td>
<td>$107.94 \pm 44.58$</td>
<td></td>
<td>$86.87 \pm 35.86$</td>
</tr>
<tr>
<td>REDV (ml)</td>
<td>$83.74 \pm 32.64$</td>
<td>$103.53 \pm 46.01$</td>
<td></td>
<td>$84.35 \pm 23.77$</td>
<td>$102.63 \pm 44.65$</td>
<td></td>
<td>$83.20 \pm 38.90$</td>
</tr>
<tr>
<td>DEDV (ml)</td>
<td>$2.36 \pm 10.46$</td>
<td>$5.59 \pm 11.05$</td>
<td></td>
<td>$0.86 \pm 7.32$</td>
<td>$5.31 \pm 10.91$</td>
<td></td>
<td>$3.67 \pm 12.48$</td>
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<tr>
<td>TID ratio</td>
<td>$1.02 \pm 0.11$</td>
<td>$1.07 \pm 0.13$</td>
<td></td>
<td>$0.99 \pm 0.09$</td>
<td>$1.07 \pm 0.12$</td>
<td></td>
<td>$1.05 \pm 0.12$</td>
</tr>
<tr>
<td>SSS</td>
<td>0.0</td>
<td>7.0</td>
<td></td>
<td>0.0</td>
<td>7.0</td>
<td></td>
<td>0.0</td>
</tr>
<tr>
<td>SRS</td>
<td>0.0</td>
<td>1.0</td>
<td></td>
<td>0.0</td>
<td>1.0</td>
<td></td>
<td>0.0</td>
</tr>
<tr>
<td>SDS</td>
<td>0.0</td>
<td>5.0</td>
<td></td>
<td>0.0</td>
<td>5.0</td>
<td></td>
<td>0.0</td>
</tr>
</tbody>
</table>

$s$, stress, $R$, rest, $D$, difference, $LVEF$, left ventricular ejection fraction, $SSS$, summed stress score, $SRS$, summed rest score, $SDR$, summed difference score, $ESV$, end-systolic volume, $EDV$, end-diastolic volume, $TID$, transient ischemic dilatation

Fig. 1 Correlation between difference left ventricular ejection fractions (DLVEF) and transient ischemic dilatation (TID) ratio in group IA

Fig. 2 Correlation between DLVEF and TID ratio in group IB

Fig. 3 Correlation between DLVEF and TID ratio in group IIA

Fig. 4 Correlation between DLVEF and TID ratio in group IIB

Patients with ischemia were regrouped according to the SDS. SDS = 4–5, SDS = 6–8, and SDS ≥ 9 were considered, mild ischemia (MI), moderate ischemia (ModI), and severe ischemia (SI), respectively. The incidences of TLVD in patients with MI, ModI SI were greater than in patients with normal perfusion on
The incidence of TLVD was statistically different between groups A and B ($P < 0.0001$) in whole group. The incidence of TLVD was statistically different between groups IA and IB and groups IIA and IIB (all $P < 0.0001$). However, the incidence of TLVD was not significantly different between groups I and II ($P = 0.440$).

Coronary angiography results were available for the 74 (33.2%) patients with ischemia. Sixty of them (81.1%) were pathological (>50% stenosis). CAG was abnormal for 25 patients (78.1%) and 35 patients (83.3%) in groups I and II, respectively. There was no significant difference in the incidence of abnormal CAG between groups I and II ($P = 0.574$).

In present study, we found LVEF to be significantly decreased after stress compared with rest values in patients with ischemia. This observation is valid for not only the exercise group but also the pharmacological stress group. However, in patients with normal perfusion, the difference between stress and rest LVEF values were not significant. In addition, the incidence of TLVD in patients with ischemia was more than that in patients with normal perfusion (36% vs. 9%). Similar results were obtained for subgroups. Thus, the frequency of TLVD was greater in patients with ischemia than in patients with normal perfusion in the exercise and pharmacological subgroups (39% vs. 12% and 34% vs. 7%, respectively). This observation supported the suggestion that TLVD after stress might be seen following not only post-exercise but also pharmacological stress consistent with ischemia. LVEF worsening after stress in patients with MI, ModI, and SI was more frequent than in patients with normal perfusion. Conversely, we could not find any difference between the frequencies of LVEF worsening in patients with MI and ModI, MI and SI, and ModI and SI. Thus, the worsening of LVEF was not in concordance with the severity of perfusion abnormalities.

Pharmacological stimulation differs from exercise stress because it does not cause demand ischemia. In patients with severe stenosis, dipyridamole administration can cause a "steal" of blood flow, which may result in true ischemia in the presence of increased oxygen demand and therefore may also cause stunning [19]. The number of studies that have compared exercise and pharmacological stress effects on TLVD using GSPECT are limited in number and conflicting [2, 11–15].

Iwado et al. [2] performed GSPECT on 362 patients, after exercise ($n = 199$) or short acting pharmacological stress ($n = 163$) to determine whether TLVD could also be seen after short acting pharmacological stress (adenosine triphosphate). They found LVEF to be significantly decreased after exercise stress when compared with rest values in ischemia and fixed defect groups. However, the difference was not significant after pharmacological stress. They concluded that a transient decrease in left ventricular ejection fraction after stress was observed following exercise, but not following a short acting pharmacological stress (adenosine). In contrast, Hung et al. [12] demonstrated dipyridamole-induced worsening of LVEF by Tl-201 GSPECT in 126 patients. They correlated the
GSPECT results with those of CAG and found the sensitivity of LVEF worsening in detecting CAD to be only 35\%, whereas the specificity was as high as 93\%. Interestingly, the specificity of LVEF worsening was found superior to the specificity of perfusion images in detecting CAD (93\% vs. 60\%). In another study Druz et al. [15] reported that following adenosine, myocardial stunning occurred in one-third of the patients with severe reversible defects, consistent with ischemia. The results of the present study were compatible with the studies of Druz et al. [12] and Hung et al. [15]. However, a discrepancy was found between our results and the conclusions reached by Iwado et al. [2] and Tanaka et al. [14]. Although the mechanisms underlying the effects of adenosine and dipyridamole were similar, dipyridamole can produce a much longer effect than adenosine because its half-life is considerably longer than that of adenosine (up to 20–45 min vs. less than 10 s) [20]. So, myocardial ischemia and also side effects may be prolonged after dipyridamole stress. The inconsistency between the previous studies could be attributed to the longer half-life of dipyridamole. But, interestingly, Druz et al. [15] also performed their study using dipyridamole and found myocardial stunning in one-third of the patients with severe ischemia. Further studies in ischemic patients comparing dipyridamole and adenosine on post-stress stunning will have to be performed to explain this statement.

The differences between DLVEF values in patients with normal perfusion and ischemic patients may be attributed to day-to-day or test–retest variability. Because we previously reported the day-to-day variability of GSPECT results in our department in another study, we did not repeat it for the present study [7]. In this study, no significant difference was found in the day-to-day measurements of EDV, ESV, and LVEF, with mean differences and SD of $-2.6 \pm 17.8$ ($P = 0.6$), $-3.6 \pm 12.5$ ($P = 0.3$), and $0.9 \pm 3.5$ ($P = 0.4$), respectively [7]. There were excellent correlations and close limits of agreement between day-to-day measurements of EDV, ESV, and LVEF measured with gated GSPECT ($r = 0.93$, 0.98, and 0.99 for LVEF, EDV, and ESV, respectively) [7].

We also found significant good correlations between TID ratios and DLVEF values in four subgroups ($r = -0.55$, $r = -0.62$, $r = -0.59$, and $r = -0.41$, for groups IA, IB, IIA, and IIB, respectively; all $P < 0.0001$). When we carefully examined the correlation plots (Figs. 1, 2, 3, 4) TID ratio increased when negative values of DLVEF increased. Conversely, TID ratio decreased when positive values of DLVEF increased. Thus, patients who had higher SLVEF than RLVEF had lower TID ratios. On the other hand, patients who possessed lower SLVEF than RLVEF had higher TID ratios. According to these results in this group of patients TID ratios were concordant with TLVD. Our results were compatible with earlier studies [21–23].

Interestingly, we observed TLVD in 20 of 216 (9\%) patients with normal perfusion. In some patients a hypertensive response to exercise could cause systolic dysfunction and wall motion abnormalities in the absence of CAD according to earlier studies [24, 25]. Therefore, we retrospectively reassessed the stress data of these patients. However, we found a hypertensive response to exercise in only two of them. TLVD in the remaining patients could not be attributed to a hypertensive response to exercise. In another study, Cholewinski et al. [26] found a significant depression of LVEF in 2 of 10 patients with normal myocardial perfusion. This finding is similar (9\% vs. 20\%) to our results. According to these authors, possible explanations for this situation could have been a non-detectable microcirculatory blood flow abnormality, a non-specific incidental stimulation resulting in an increase in the rest LVEF, and some other cardiac pathology causing decreased functional reserve of the myocardium. However, they concluded that the mechanism of post-stress dysfunction in normal myocardium was unclear.

Because the present study was performed retrospectively, CAG results were not available for all patients with ischemia. This is a limitation of our study. However, 81.1\% of the ischemic patients whose CAG results were accessible had pathological coronary arteries. Additionally, we could not find any significant difference in the frequency of abnormal CAG between groups I and II ($P = 0.574$). Therefore, the two groups were comparable with respect to the CAG results.

Although stress-induced myocardial stunning may persist for up to 24 h usually it resolves during the first 30 min to 60 min [27]. In the present study, we also demonstrated that TLVD could be seen after 45 min not only after exercise but also after pharmacological stress. Hence, care must be taken when evaluating post-stress GSPECT images and LVEF values because post-stress values do not represent the rest values.

**Conclusions**

In conclusion, dipyridamole is believed to be less likely than exercise to induce ischemia. However, in this study, TLVD after stress was observed following not only exercise but also pharmacological stress consistent with ischemia. The prognostic significance of this result should be investigated with prospective studies in groups with large numbers of patients.
References


