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DIAGNOSTIC VALUE OF POSTSYSTOLIC SHORTENING OF THE LEFT VENTRICULAR MYOCARDIUM ASSESSED DURING SPECKLE TRACKING STRESS ECHOCARDIOGRAPHY ON THE TREADMILL IN PATIENTS WITH CORONARY ARTERY DISEASE

<i>Aim</i>	To evaluate the diagnostic capacity of left ventricular (LV) postsystolic shortening (PSS) values obtained by speckle-tracking stress-echocardiography (stress-EchoCG) using a treadmill test in determining the functional significance of the degree of coronary artery (CA) stenosis.
<i>Material and methods</i>	The study included 132 patients (80 men aged 65.0±9.3 years) with suspected or previously verified diagnosis of ischemic heart disease. Stress-EchoCG with the treadmill test was performed for all patients. Strain parameters were determined by two-dimensional speckle-tracking on gray-scale images before and after the exercise. Values of LV postsystolic index (PSI) and LV mean postsystolic time (PST) were calculated. Coronary angiography was performed for all patients. Patients were divided into 3 groups based on the severity of CA stenosis according to the G. G. Gensini score.
<i>Results</i>	LV PSS values at rest did not significantly differ between the patient groups. After completion of the exercise, the mean LV PSI was significantly higher for patients with pronounced CA stenosis than in the group without CA stenosis or with moderate CA stenosis: 8.9% [3.8; 10.7%] vs. 3.8% [2.2; 6.8%] (p=0.012) and 3.4% [2.2; 6.2%] (p=0.012), respectively. The mean LV PSI after completion of the exercise indicated the presence of pronounced CA stenosis with a sensitivity of 75% and a specificity of 61% (area under the curve, AUC, 0.74±0.06; p<0.001). After completion of the exercise, the mean LV PST was significantly greater for patients with pronounced CA stenosis than in the group without CA stenosis or with moderate CA stenosis: 27.4 [18.7; 34.7] ms vs. 18.4 [10.8; 26.5] ms (p=0.036) and 20.9 [14.2; 29.5] ms (p=0.036), respectively. The mean LV PST after completion of the exercise exceeding 23.5 ms suggests pronounced CA stenosis with a sensitivity of 71% and a specificity of 65% (AUC 0.69±0.06; p=0.004). A complex evaluation of the LV PSI, the LV local contractility disorder (LCD) index, the LV PST, and LV LCD index allows enhancement of the test sensitivity in diagnosing pronounced CA stenosis.
<i>Conclusion</i>	Determination of LV PSS in speckle-tracking stress-EchoCG may be useful for evaluating the functional significance of the degree of CA stenosis to enhance the sensitivity of stress-EchoCG in patients with pronounced CA stenosis.
<i>Keywords</i>	Postsystolic shortening; postsystolic index; speckle-tracking echocardiography; stress-echocardiography; ischemic heart disease
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Stress echocardiography is used to assess transient myocardial ischemia and viability of the myocardium, as well as in the prognosis in diverse groups of patients with coronary artery disease (CAD) based on regional left ventricular wall motion abnormalities (LVWMA) [1]. However, the visual assessment of LVWMA is subjective [2]. Speckle tracking echocardiography (STE) can be used to quantify LV myocardial strain and longitudinal post-systolic strain or post-systolic shortening (PSS).

PSS involves a shortening of LV myocardium, which occurs with closure of the aortic valve [3]. Although it is not clear whether the onset of PSS is associated with viable myocardium or caused by passive wall motion, several studies evaluating LV PSS in patients with ST elevation myocardial infarction shown by resting echocardiography demonstrated that LV PSS was associated with decreased systolic function and worse prognosis for patients [4, 5]. It was demonstrated that the presence of LV PSS is a predictor of severe coronary artery stenosis in patients with stable

exertional angina [6]. In two studies, dobutamine stress echocardiography demonstrated in patients with suspected CAD that LV PSS assessed by tissue Doppler imaging increased in ischemic segments and was relatively highly sensitive and specific in detecting ischemia [7, 8].

Thus, given that tissue Doppler stress echocardiography has demonstrated the possibility of using PSS for evaluating myocardial ischemia, speckle tracking stress echocardiography using treadmill exercise to determine LV PSS is also a promising method of assessing the functional significance of coronary stenosis.

Objective

To evaluate the diagnostic possibilities of LV PSS determined by stress STE on treadmill in the estimation of functional significance of coronary artery stenosis.

Material and Methods

The design of the study was observational, comparative and single center. The study included 132 patients (from 42 to 85 years old, with a mean age 65.0 ± 9.3 years; 52 (39.4%) female and 80 (60.6%) male patients).

Inclusion criteria: signed consent on stress STE with the analysis of LV strain and diagnostic coronary artery angiography (CAG); sinus rhythm; stress STE on treadmill performed and strain indicators calculated; diagnostic CAG.

Exclusion criteria: a history of high functional class (FC) heart failure (HF); cardiomyopathy, cancer; supraventricular or ventricular arrhythmia at the time of examination; high-grade conduction abnormalities; severe heart valve defects; a history of myocardial infarction (MI); heart valve birth defects; contraindications for stress echocardiography on treadmill or CAG [9, 10]; failure to calculate LV strain at rest or following exercise; absence of patient consent to participate in the study or refusal to sign informed consent for stress STE with post-processing evaluation of LV strain / CAG.

Beta-blockers were discontinued 48 hours before stress STE. Pretest probability of CAD was assessed in all patients following the guidelines [11]. All patients underwent CAG within three months before/after stress STE. As shown in Table 1, all patients were divided to three groups based on the CAG findings. Group 1 included 42 patients without coronary stenosis according to CAG (Gensini score: 0), Group 2 was made up of 63 patients with moderate coronary stenosis (Gensini score: <34), while Group 3 comprised 27 patients with severe coronary stenosis (Gensini score: ≥ 35).

In 70% of patients, CAG was performed after stress echocardiography. In Group 1, stress STE was performed in 8 (19%) patients after CAG due to suspected CAD with normal coronary arteries. In Group 2 and Group 3, stress STE was performed after CAG in 22 (34.9%) and 6 (22%) patients,

respectively, in order to assess the functional significance of coronary artery stenosis shown by CAG.

Male patients prevailed in the group of severe coronary stenosis, as compared to patients without coronary artery stenosis ($p=0.042$). Patients with severe coronary stenosis were relatively older than patients without coronary stenosis ($p=0.036$). The groups were essentially comparable in terms of clinical data (see Table 1). The pretest probability of CAD in patients with severe coronary stenosis was statistically significantly higher than in patients without coronary artery stenosis ($p<0.001$).

Standard stress echocardiography was performed using a Vivid E95 ultrasound system [12]. Treadmill exercise was performed on a GE Healthcare Series 2100 treadmill following the Bruce protocol. Electrocardiogram (ECG) and HR were registered at rest and during exercise, while blood pressure (BP) was measured using a manual tonometer at each step of the exercise. The protocol of stress echocardiography was as described earlier [13].

In addition to following the standard longitudinal strain assessment protocol, mean LV post-systolic index (PSI) and post-systolic time (PST) were evaluated. Longitudinal LV systolic strain was defined as the maximum longitudinal strain at end systole. Left ventricular global longitudinal systolic strain (LVGLS) was calculated as the mean values of LV longitudinal systolic strain for all 17 segments of interest.

LV PSI was defined as the ratio of the difference between peak global strain to peak systolic strain to peak global strain multiplied by 100% [14]. LV PST was calculated as the time from the aortic valve closure to the maximum value of peak global strain. Mean values of LV PSI and PST were calculated for 17 LV myocardial segments. Delta LVGLS, mean LV PSI and mean LV PST were calculated as the difference between the baseline values and the values registered following the exercise. Changes in mean LV PSI and mean LV PST were evaluated before and after the exercise in all patients. CAG was performed by an independent interventional surgeon using the Judkins technique. The severity of coronary artery stenosis was assessed using the Gensini score: no stenosis – 0; moderate stenosis – 1–34; severe stenosis – ≥ 35 [15].

All patients signed informed consent before being included in the study. The study protocol was approved by the Ethics Committee of the Central State Medical Academy under the Directorate of the President of the Russian Federation (Protocol No. 12-1 / 2019 approved on 09/17/2019).

The data obtained were processed using the SPSS 23.0. The normality of distribution was verified using the Lilliefors test based on the Kolmogorov–Smirnov test. The normally distributed quantitative data were expressed as the mean values and standard deviations ($M \pm SD$) and estimated using Student's t-test. The non-normally distributed quantitative indicators were described using the median values and the

Table 1. Clinical and anthropometric characteristics of the patient groups (n=132)

Parameter	Group 1 (n=42)	Group 2 (n=63)	Group 3 (n=27)	P
Sex (male / female), n (%)	18 (42.9) / 24 (57.1)	42 (66.7) / 21 (33.3)	20 (74.1) / 7 (25.9)	P ₁₋₂ =0.078 P ₁₋₃ =0.042* P ₂₋₃ =0.621
Age, years (M±SD; 95% CI)	62.4±8.3 59.8–65.0	65.5±9.7 63.1–68.0	67.9±9.1 64.3–71.5	P ₁₋₂ =0.273 P ₁₋₃ =0.036* P ₂₋₃ =0.852
Height, cm (M±SD; 95% CI)	169.2±8.8 166.5–172.0	171.1±8.8 168.9–173.3	171.0±8.8 167.5–174.5	P ₁₋₂ =0.870 P ₁₋₃ =0.411 P ₂₋₃ =0.977
Weight, kg (M±SD; 95% CI)	81.4±16.2 76.4–86.5	84.9±17.3 80.5–89.3	81.2±15.3 75.2–87.3	P ₁₋₂ =0.918 P ₁₋₃ =0.958 P ₂₋₃ =0.344
Body mass index, kg/m ² (M±SD; 95% CI)	28.4±4.9 26.9–29.9	27.7±4.5 26.1–29.9	27.6±4.1 26.0–29.3	P ₁₋₂ =0.659 P ₁₋₃ =0.498 P ₂₋₃ =0.732
Pre-test probability of CAD, % (Me [Q1; Q3])	16.0 [6.0; 27.0]	22.0 [10.5; 27.0]	24.0 [22.0; 44.0]	P ₁₋₂ =0.657 P ₁₋₃ <0.001* P ₂₋₃ =0.078
Hypertensive heart disease, n (%)	30 (71.4)	52 (82.5)	22 (81.5)	P ₁₋₂ =0.690 P ₁₋₃ =0.403 P ₂₋₃ =0.999
History of exertional angina FC I–II, n (%)	16 (38.1)	21 (33.3)	13 (48.1)	P ₁₋₂ =0.679 P ₁₋₃ =0.466 P ₂₋₃ =0.711
History of heart rhythm disorders, n (%)	12 (28.6)	14 (22.2)	7 (25.9)	P ₁₋₂ =0.495 P ₁₋₃ =0.999 P ₂₋₃ =0.787
History of AF, n (%)	7 (16.7)	13 (20.6)	6 (22.2)	P ₁₋₂ =0.800 P ₁₋₃ =0.753 P ₂₋₃ =0.999
Diabetes mellitus, n (%)	6 (14.3)	11 (17.5)	6 (22.2)	P ₁₋₂ =0.790 P ₁₋₃ =0.518 P ₂₋₃ =0.573
Bronchial asthma, n (%)	1 (2.4)	3 (4.8)	1 (3.7)	P ₁₋₂ =0.648 P ₁₋₃ =0.999 P ₂₋₃ =0.999
COPD, n (%)	3 (7.1)	7 (11.1)	4 (15.4)	P ₁₋₂ =0.736 P ₁₋₃ =0.415 P ₂₋₃ =0.724
Smoking, n (%)	5 (11.9)	16 (25.4)	7 (25.9)	P ₁₋₂ =0.402 P ₁₋₃ =0.582 P ₂₋₃ =0.999
Dyslipidemia, n (%)	11 (26.2)	17 (27.0)	8 (29.6)	P ₁₋₂ =0.999 P ₁₋₃ =0.788 P ₂₋₃ =0.802

CI – confidence interval; FC – functional class; AF – atrial fibrillation; COPD – chronic obstructive pulmonary disease. * p<0.05.

lower and upper quartiles (Me [Q1; Q3]) and estimated using the Mann-Whitney U-test. The Bonferroni correction was used to nullify the effect of multiple comparisons. The categorical indicators were expressed as a percentage. Pearson's chi-squared test was used to assess statistical significance of

the differences between percentages. A ROC analysis was performed; ROC curves were constructed for LV PSI and LV PST at rest and after exercise, as well as for LVWMA index and the combinations of LV PSI with LVWMA index and LV PST with LVWMA index. The significance was p<0.05.

Results

A total of 4,488 segments were analyzed during stress STE. Values of regional longitudinal LV strain could not be obtained for 16 segments at rest and 50 segments after exercise. The stress echocardiography findings are provided in Table 2.

Only patients with severe coronary artery disease complained of typical angina pain. HR at maximum load was statistically significantly higher in patients with severe coronary stenosis than in patients without coronary artery stenosis (p=0.009 and p=0.048, respectively). According to stress STE, 5 (11.9%) positive tests, 31 (73.8%) negative tests and 6 (14.3%) ambiguous tests were reported in Group 1, while in Group 2, 6 (9.5%) positive tests, 49 (77.7%) negative tests, and 8 (12.8%) ambiguous tests were registered; 12 (44.4%) positive tests, 10 (37%) negative tests and 5 (18.6%) ambiguous tests were registered in Group 3. With statistical significance, there were more patients with positive stress STE test in the group with severe coronary stenosis than patients without stenosis and with moderate coronary stenosis (p=0.012 and p=0.001, respectively).

Stress STE on treadmill and CAG showed that the provisional diagnosis of CAD should be reviewed in 88.1% of patients in Group 1. CAD with normal coronary arteries was suspected in 11.4% of patients without coronary artery stenosis and with positive stress STE test. Angina pain was recorded during stress STE in a small percentage of cases, while the history of exertional angina was established in 33.3–48.1% of cases.

In the severe coronary stenosis group, LVWMA indices after the termination of exercise were statistically significantly higher compared to the groups without coronary stenosis and with moderate coronary stenosis (1.13±0.21 versus 1.00±0.03 (p<0.001) and 1.01±0.04 (p<0.001), respectively).

The ROC analysis performed in order to assess the diagnostic value of LVWMA index in detecting severe coronary stenosis after exercise demonstrated good model quality (AUC was 0.74±0.07) with a sensitivity of 58% and specificity of 91% for the index of 1.02.

Table 3 shows the results of stress STE. The values of LVGLS were not statistically significantly different between the groups. After exercise, LVGLS was statistically significantly lower in patients with severe coronary stenosis than in patients without coronary stenosis and with

Table 2. Stress echocardiography (n=132)

Parameter	Group 1 (n=42)	Group 2 (n=63)	Group 3 (n=27)	P
SBP at rest, mm Hg (Me [Q1; Q3])	125.0 [120.0; 135.0]	130.0 [120.0; 140.0]	130.0 [125.0; 130.0]	$p_{1-2}=0.365$ $p_{1-3}=0.963$ $p_{2-3}=0.986$
SBP after exercise, mm Hg (Me [Q1; Q3])	180.0 [160.0; 200.0]	180.0 [157.5; 195.0]	170.0 [160.0; 180.0]	$p_{1-2}=0.583$ $p_{1-3}=0.510$ $p_{2-3}=0.398$
DBP at rest, mm Hg (Me [Q1; Q3])	80.0 [70.0; 80.0]	80.0 [70.0; 80.0]	80.0 [72.5; 80.0]	$p_{1-2}=0.884$ $p_{1-3}=0.812$ $p_{2-3}=0.714$
DBP after exercise, mm Hg (Me [Q1; Q3])	80.0 [80.0; 90.0]	80.0 [80.0; 90.0]	80.0 [80.0; 90.0]	$p_{1-2}=0.847$ $p_{1-3}=0.685$ $p_{2-3}=0.834$
HR at rest, bpm (Me [Q1–Q3])	66.5 [61.0; 76.0]	68.0 [63.0; 73.5]	66.0 [59.0; 72.5]	$p_{1-2}=0.379$ $p_{1-3}=0.834$ $p_{2-3}=0.888$
HR after exercise, bpm (Me [Q1–Q3])	134.0 [127.0; 142.0]	133.0 [123.0; 140.0]	126.0 [113.5; 133.0]	$p_{1-2}=0.502$ $p_{1-3}=0.009^*$ $p_{2-3}=0.048^*$
Chest pain, n (%)	2 (4.8)	5 (7.9)	5 (18.5)	$p_{1-2}=0.700$ $p_{1-3}=0.306$ $p_{2-3}=0.477$
Angina pain treated with nitrates, n (%)	–	–	2 (7.4)	–
New onset rhythm disorders, n (%)	31 (73.8)	43 (68.3)	18 (66.7)	$p_{1-2}=0.663$ $p_{1-3}=0.592$ $p_{2-3}=0.999$
including:				
• Isolated / pair of supraventricular / ventricular extrasystoles	31 (100)	42 (97.7)	18 (100)	$p_{1-2}=0.999$ $p_{1-3}=0.867$ $p_{2-3}=0.999$
• VT runs	–	1 (2.3)	–	–
Dyspnea, n (%)	17 (41.5)	21 (33.3)	14 (51.9)	$p_{1-2}=0.413$ $p_{1-3}=0.461$ $p_{2-3}=0.321$
Sub-maximum HR achieved, n (%)	35 (83.3)	48 (76.2)	17 (63.0)	$p_{1-2}=0.467$ $p_{1-3}=0.255$ $p_{2-3}=0.633$
Mean load, METS (Me [Q1–Q3])	7.0 [7.0; 7.0]	7.0 [4.6; 7.0]	4.6 [4.6; 7.0]	$p_{1-2}=0.906$ $p_{1-3}=0.303$ $p_{2-3}=0.474$
Load time, min (Me [Q1–Q3])	5.5 [4.5; 6.4]	6.1 [4.3; 7.2]	5.0 [3.3; 6.5]	$p_{1-2}=0.541$ $p_{1-3}=0.870$ $p_{2-3}=0.552$
Positive stress echocardiography, n (%)	5 (11.9)	6 (9.5)	12 (44.4)	$p_{1-2}=0.751$ $p_{1-3}=0.012^*$ $p_{2-3}<0.001^*$

SBP – systolic blood pressure;
DBP – diastolic blood pressure; HR – heart rate;
VT – ventricular tachycardia. * $p<0.05$.

moderate coronary stenosis ($16.0\pm4.1\%$ versus $20.7\pm3.8\%$ ($p<0.001$) and $20.0\pm3.2\%$ ($p=0.001$), respectively).

The values of LV PSI were not statistically significantly different between the groups. After exercise, LV PSI was statistically significantly higher in patients with severe coronary stenosis than in patients without coronary stenosis and with moderate coronary stenosis (8.9 [3.8; 10.7] % versus 3.8 [2.2; 6.8] % ($p=0.012$) and 3.4 [2.2; 6.2] % ($p=0.012$), respectively).

Analysis of the diagnostic value of the LV PSI after exercise in detecting coronary artery stenosis showed poor model quality (AUC was 0.57 ± 0.05). Assessment of LV PSI after exercise in detecting severe coronary artery stenosis showed good model quality (AUC was 0.74 ± 0.06) with sensitivity of 75% and specificity of 61% for the index of 4.9%. The ROC curve of the evaluation of severe coronary stenosis using LV PSI after exercise is shown in Figure 1.

The mean values of LV PST at rest were not statistically significantly different between the groups. Median LV PST after exercise was statistically significantly greater in patients with severe coronary stenosis than in patients without coronary stenosis and with moderate coronary stenosis (27.4 [18.7; 34.7] ms versus 18.4 [10.8; 26.5] ms ($p=0.036$) and 20.9 [14.2; 29.5] ms ($p=0.036$), respectively).

Analysis of the diagnostic value of median LV PST after exercise in detecting coronary artery stenosis showed poor model quality (AUC was 0.57 ± 0.06). The assessment of median LV PSI after exercise in detecting severe coronary artery stenosis showed moderate model quality (AUC was 0.69 ± 0.06) with sensitivity of 75% and specificity of 65% for the value of 23.5 ms. The ROC curve of the evaluation of severe coronary stenosis using LV PST after exercise is shown in Figure 2.

Since the maximum values of AUC were obtained for median LV PSI and median LV PST after exercise, ROC analysis of the assessment of these indicators in combination with LVWMA index in detecting severe coronary artery stenosis was conducted (Figure 3). The combination of median LV PSI and LVWMA index in detecting severe coronary stenosis showed a very high model quality (AUC was 0.80 ± 0.06) with sensitivity of 71% and specificity of 79%. Assessment of the combination of median LV PST and LVWMA index in detecting severe coronary stenosis showed a high model quality (AUC was 0.78 ± 0.06) with sensitivity of 75% and specificity of 78%.

As shown in Table 4, statistically significant increases in median LV PSI after exercise were detected in all three patient groups. Decreases in median LV PST were statistically insignificant in all patient groups.

Discussion

In our study evaluating post-systolic LV shortening, the assessment of the functional significance of coronary artery

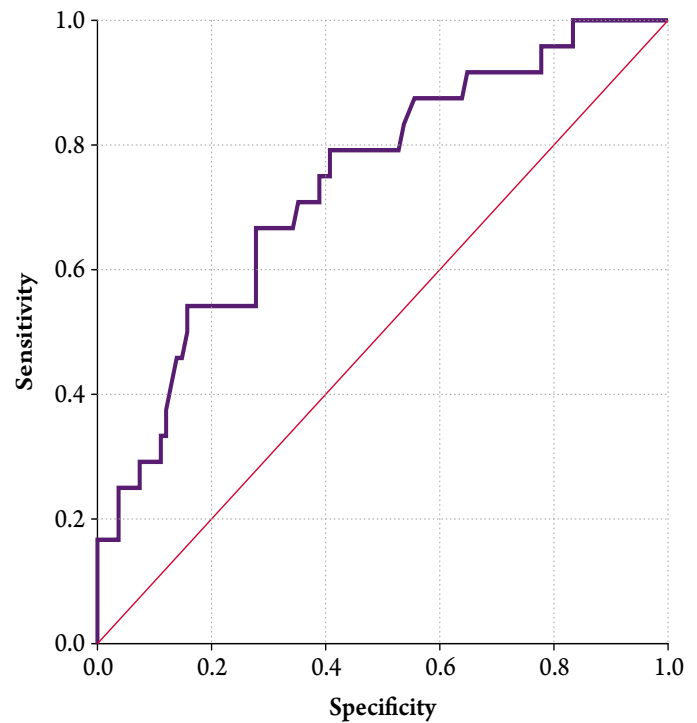
Table 3. Speckle-tracking stress echocardiography (n=132)

Parameter	Group 1 (n=42)	Group 2 (n=63)	Group 3 (n=27)	P
LVGLS at rest, % (M±SD; 95% CI)	19.2±3.1 (18.2–20.2)	19.3±2.7 (18.6–20.0)	18.0±2.8 (16.9–19.1)	P ₁₋₂ =0.811 P ₁₋₃ =0.142 P ₂₋₃ =0.234
LVGLS after exercise, % (M±SD; 95% CI)	20.7±3.8 (19.5–21.9)	20.0±3.2 (19.1–20.7)	16.0±4.1 (14.4–17.7)	P ₁₋₂ =0.348 P ₁₋₃ <0.001* P ₂₋₃ <0.001*
Mean LV PSI at rest, % (Me [Q1; Q3])	2.0 [0.9; 4.1]	2.1 [1.3; 4.2]	2.7 [1.9; 5.2]	P ₁₋₂ =0.465 P ₁₋₃ =0.279 P ₂₋₃ =0.154
Mean LV PSI after exercise, % (Me [Q1; Q3])	3.8 [2.2; 6.8]	3.4 [2.2; 6.2]	8.9 [3.8; 10.7]	P ₁₋₂ =0.746 P ₁₋₃ =0.012* P ₂₋₃ =0.012*
Mean LV PST at rest, % (Me [Q1; Q3])	20.1 [11.6; 31.8]	22.2 [14.5; 30.2]	27.4 [21.3; 36.2]	P ₁₋₂ =0.578 P ₁₋₃ =0.147 P ₂₋₃ =0.102
Mean LV PST after exercise, % (Me [Q1; Q3])	18.4 [10.8; 26.5]	20.9 [14.2; 29.5]	27.4 [18.7; 34.7]	P ₁₋₂ =0.560 P ₁₋₃ =0.036* P ₂₋₃ =0.036*

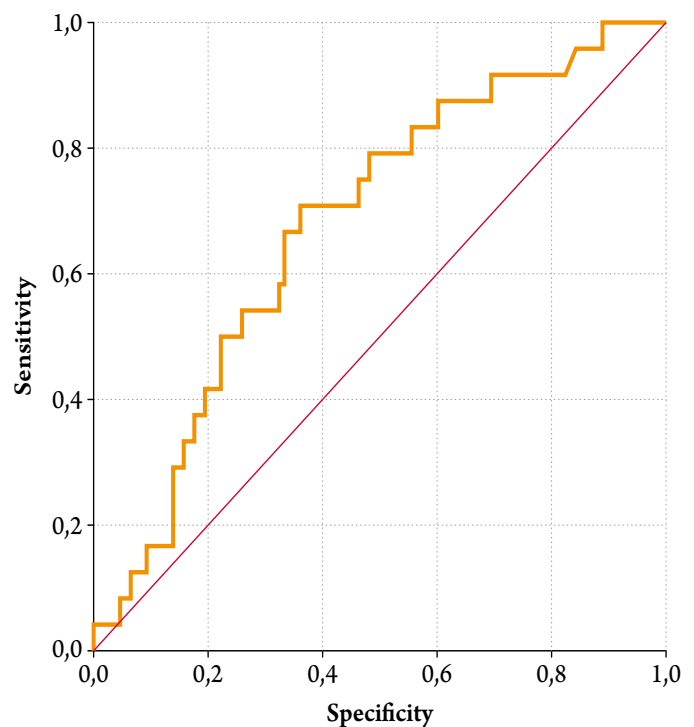
LVGLS – left ventricular global systolic strain; LV PSI – left ventricular post-systolic index; LV PST – left ventricular post-systolic time; CI – confidence interval. * p<0.05.

stenosis in patients with severe coronary artery stenosis demonstrated statistically significant higher median LV PSI and LV PST after exercise than in patients without stenosis and with moderate coronary stenosis. Our findings are consistent with the published data by Uusitalo et al. (2016), according to which higher median LV PSI after exercise was statistically significant in the group of patients with severe coronary artery stenosis than in the group without coronary stenosis [16].

Several studies have demonstrated high diagnostic significance of LV PSS during resting echocardiography and dobutamine stress echocardiography in patients with CAD [7, 17]. In our study, the evaluation of the diagnostic value of median LV PSI and median LV PST in the assessment of severe coronary artery stenosis demonstrated relatively lower sensitivity and specificity as compared to the study by Rumbinaite et al. (2020), in which regional LV PSI in dobutamine stress echocardiography after exercise detected severe single-vessel coronary stenosis with sensitivity of 87% and specificity of 92% [18]. In our study, the maximum sensitivity and specificity for median LV PSI after exercise in the diagnosis of severe coronary artery stenosis were 75% and 61%, respectively. Lower sensitivity and specificity may be due to the fact that Rumbinaite et al. evaluated regional LV PSI, while our study investigated median LV PSI for

Figure 1. ROC curve showing the potential of median LV PSI after exercise in evaluating severe coronary artery stenosis (AUC=0.74±0.06; 95% CI 0.63–0.85; p<0.001)


LV PSI – left ventricular post-systolic Index;
CI – confidence interval.

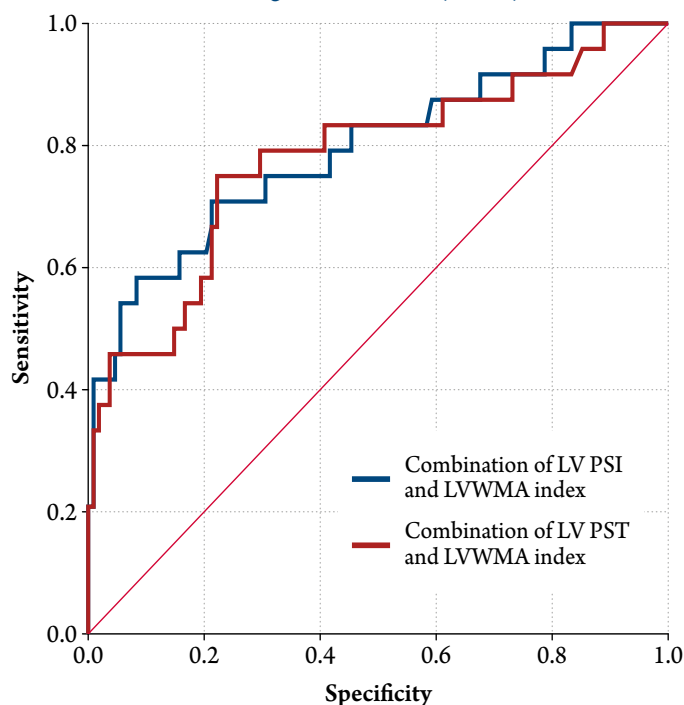
Figure 2. ROC curve showing the potential of median LV PST after exercise in evaluating severe coronary artery stenosis (AUC=0.69±0.06; 95% CI 0.58–0.80; p=0.004)


LV PST, left ventricular post-systolic time; CI, confidence interval.

Table 4. Changes in LV PSI and LV PST (n=132)

Parameter	Patient group	Before exercise (1)	After exercise (2)	Δ	P ₁₋₂
LV PSI, % (Me [Q1; Q3])	Group 1 (n=42)	2,0 [0,9; 4,1]	3,8 [2,2; 6,8]	1,2 [0,2; 3,0]	0,042*
	Group 2 (n=63)	2,1 [1,3; 4,2]	3,4 [2,2; 6,2]	1,1 [0,4; 3,3]	0,006*
	Group 3 (n=27)	2,7 [1,9; 5,2]	8,9 [3,8; 10,7]	3,5 [1,1; 7,0]	0,007*
LV PST, ms (Me [Q1; Q3])	Group 1 (n=42)	20,1 [11,6; 31,8]	18,4 [10,8; 26,5]	2,4 [-5,5; 12,0]	0,261
	Group 2 (n=63)	22,2 [14,5; 30,2]	20,9 [14,2; 29,5]	0,4 [-2,1; 5,9]	0,561
	Group 3 (n=27)	27,4 [21,3; 36,2]	27,4 [18,7; 34,7]	5,4 [-10,9; 15,1]	0,864

LV PSI – left ventricular post-systolic index; LV PST – left ventricular post-systolic time. *p<0.05.

Figure 3. ROC curve showing the potential of the combination of LV PSS and LVWMA index after exercise in evaluating severe coronary artery stenosis

The blue ROC curve demonstrates the diagnostic potential of the combined assessment of median LV PSI and LVWMA index after exercise in detecting severe coronary artery stenosis (AUC = 0.80±0.06; 95% CI 0.69–0.91; p<0.001); the red ROC curve represents the potential of the combined assessment of median LV PST and LVWMA index after exercise in assessing severe coronary artery stenosis (AUC=0.78±0.06; 95% CI 0.67–0.90; p<0.001). LV PSS – left ventricular post-systolic shortening; LVWMA – left ventricular wall motion abnormality; LV PSI – left ventricular post-systolic index; LV PST – left ventricular post-systolic time; CI – confidence interval.

all LV segments [18]. Moreover, the lower sensitivity and specificity in our study may be due to using the treadmill test, specifically recording echocardiography data following exercise unlike in the dobutamine stress test, in which echocardiography data are registered during stress.

Several studies showed that the evaluation of longitudinal LV strain in combination with LVWMA index provides increased sensitivity and specificity of stress STE for detecting severe coronary artery stenosis [19–21].

Moreover, the combined evaluation of these indicators can potentially reduce the number of false positive results of stress echocardiography [19, 20]. The assessment of LV PSI and LV PST in combination with LVWMA index after exercise allowed us to obtain higher sensitivity in detecting severe coronary artery stenosis as compared to the isolated assessment of LVWMA index.

It should be noted that LV PSI in the group without coronary stenosis at rest in our study was comparable with the values obtained by Brainin et al. (2019), who studied LV PSI in healthy individuals: 2 [0.9; 4.1]% compared to 2 [0.7; 4.8]%, respectively [22]. These data allow determining approximate values of LV PSI at rest in patients without coronary artery stenosis.

When registering LV PSS in healthy individuals, it is much more difficult to interpret its indicators. Thus, studying changes in LV PSS is of particular interest in patients with CAD. The trends for median LV PSI have already been described in patients with severe coronary artery stenosis and those without stenosis using dobutamine stress STE [16]. Median LV PSI increased in both groups with the administration of low-dose dobutamine and at the maximum stress, decreasing after stress in the group without severe coronary artery stenosis and increasing in the group with severe stenosis [16]. In our study, although a statistically significant increase in median LV PSI was registered after exercise in all patient groups, this increase was more significant in the group with severe coronary artery disease. Thus, when assessing changes of LV PSI in detecting functional significance of coronary stenosis degree, a focus should be put on an increase in median LV PSI. During the assessment of LV PST, a statistically insignificant decrease in median LV PST was observed in all groups except for the group with severe coronary artery stenosis. Interpretation of median LV PST is more difficult due to it being a time index, which decreases as HR grows regardless of the presence of coronary artery stenosis. We found that median LV PST decreased less in patients with severe coronary artery stenosis than in other patient groups.

Since our study demonstrated the diagnostic capabilities of LV PSS in assessing severe coronary artery stenosis,

they could be potentially significant in patients with CAD. However, since the median values have to be calculated manually, their assessment is time-consuming, which limits their application in clinical practice.

Conclusions

1. The median post-systolic left ventricular index increases in response to stress in all patients. The increase in median left ventricular post-systolic index is greater in patients with severe coronary stenosis (from 2.7 [1.9; 5.2] at baseline to 8.9 [3.8; 10.7]; $p=0.007$) than in patients without coronary stenosis (2.0 [0.9; 4.1] at baseline to 3.8 [2.2; 6.8]; $p=0.042$).
2. Assessing the median left ventricular post-systolic index is difficult in stress echocardiography because it is a time index, which decreases when the heart rate increases regardless of the presence of coronary stenosis.
3. A median left ventricular post-systolic index higher than 4.9% following exercise suggests severe coronary artery stenosis with sensitivity of 75% and specificity of 61% ($AUC=0.74\pm0.06$; 95% confidence interval 0.63–0.85; $p<0.001$).
4. Combined assessments of median left ventricular post-systolic index and regional left ventricular wall motion

abnormality index and median left ventricular post-systolic time and regional left ventricular wall motion abnormality index increases the sensitivity of stress echocardiography in detecting severe coronary stenosis.

Limitations

The main limitation of our study was the inclusion of patients with suspected or verified CAD in the control group rather than healthy volunteers. While this is the approach used in most studies, it can produce lower strain values due to risk factors for CAD and concomitant diseases in those patients. For example, the study included patients with hypertensive heart disease, which can affect strain indices.

The second limitation of the study was a relatively small size of the group of patients with severe coronary artery stenosis compared to the other two patient groups.

The third limitation of the study was that fractional flow reserve was not evaluated at CAG. The anatomical extent of coronary artery stenosis was assessed visually by the interventional surgeon.

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