Clinical Characteristics of Patients With Exercise-Induced ST-Segment Elevation Without Prior Myocardial Infarction

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Background Exercise-induced ST-segment elevation is a relatively uncommon problem and occurs more frequently in patients who have had a myocardial infarction. Data is limited on the characteristics of Taiwanese patients without prior myocardial infarction who develop exercise-induced ST-segment elevation.

Methods and Results Exercise-induced ST-segment elevation developed in 9 of 6,147 consecutive patients without myocardial infarction who underwent treadmill exercise testing at our institution over a 4-year period. The clinical and angiographic characteristics of these patients were studied. Angiographically normal coronary arteries with coronary vasospasm were found in 5 patients, hemodynamically significant coronary stenosis was found in 3 patients, and coexisting spasm in angiographically normal coronary arteries combined with hemodynamically significant coronary stenosis in the different vessel was found in 1 patient. During a median follow-up of 71 months, 2 patients with coronary vasospasm developed recurrent angina after self-discontinuation of calcium antagonists and 2 patients (1 with coronary vasospasm and 1 with hemodynamically significant coronary stenosis) died of cardiac causes before arrival at the emergency department.

Conclusion Coronary vasospasm was a more common underlying pathology of exercise-induced ST-segment elevation in this Taiwanese cohort. Coronary angiography ± intracoronary ergonovine provocation testing is necessary in these patients to identify the underlying pathology and appropriate treatment. (*Circ J* 2006; 70: 254–261)

Key Words: Coronary vasospasm; Exercise

Exercise-induced ST-segment elevation is a relatively uncommon problem in clinical practice; its prevalence depends upon the population tested, but occurs more frequently in patients who have had a myocardial infarction.^{1–3} The mechanisms have been suggested to result from left ventricular aneurysm, left ventricular wall motion abnormalities, coronary artery stenoses and, less commonly, from coronary vasospasm.^{4–13} It is important to identify the underlying factors causing the exercise-induced ST segment elevation as the appropriate choice of treatment varies accordingly. Data is limited on the characteristics of Taiwanese patients without prior myocardial infarction who develop exercise-induced ST-segment elevation. The purpose of the present study was to investigate the clinical and angiographic characteristics of these patients.

Methods

Patient Selection

To evaluate the clinical implications of exercise-induced

ST-segment elevation, we prospectively studied patients without prior myocardial infarction who underwent treadmill exercise testing (modified Bruce protocol), ± thallium-201 (²⁰¹Tl) tomography, as the cause of oppressive chest sensation at our outpatient clinic from June 1998 through May 2002. If ST-segment elevation was observed during exercise testing, coronary angiography was suggested to define the underlying pathology (fixed coronary stenosis or coronary vasospasm). The study inclusion criteria were ST-segment elevation ≥0.1 mV in 2 of the inferior or lateral leads or ≥0.2 mV in ≥2 contiguous precordial leads during an exercise testing. Exclusion criteria were as follows: 1) prior myocardial infarction as determined by medical history, available hospital records and appearance of Q waves ≥40 ms in serial electrocardiograms; 2) electrocardiographic evidence of left ventricular hypertrophy or left bundle branch block; 3) ST-segment depression during exercise testing; 4) lack of patient cooperation for any reason such as psychological or severe systemic illness; 5) refusal of patient to undergo cardiac catheterization; 6) severe anemia with hemoglobin <7.0 mg/dl; 7) severe symptomatic electrolyte imbalance; 8) severe peripheral vascular disease limiting vascular access; 9) decompensated congestive heart failure or acute pulmonary edema; 10) severe coagulopathy; 11) aortic valve endocarditis; 12) unexplained fever; and 13) severe uncontrolled hypertension.

Clinical Data

Patients were assessed for cardiac risk factors, including cigarette smoking, diabetes mellitus, hypercholesterolemia

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and hypertension. History of smoking was assessed by asking patients if they had ever smoked over 10 cigarettes per day for a year or more during their lifetime. Diabetes mellitus was defined based on receipt of dietary treatment and/or medical therapy. Hypercholesterolemia was defined as a serum total cholesterol >200 mg/dl and hypertension as receiving medical therapy or having a blood pressure of >140/90 mmHg.

Treadmill Exercise Testing

If the patient did not undergo exercise ^{201}Tl tomography, the treadmill exercise testing was performed in the afternoon. If the patient underwent exercise ^{201}Tl tomography, the treadmill exercise testing was performed in the mid-morning (09.00–10.00 h). Exercise testing was performed after the patients had fasted for ≥ 4 h. Beta-blocking agents were discontinued for 48 h before the testing, and nitrate agents were stopped for ≥ 4 h. The exercise stress tests were carried out according to the modified Bruce protocol using two 3-min warm-up stages (at 2,735 m/h 0% grade and 2,735 m/h and 5% grade) before the standard Bruce protocol, which starts at 2,735 m/h and 10% grade. Signals from a 12-lead electrocardiogram were displayed continuously during the observations and recorded at regular intervals and when chest pain occurred during the exercise or in the recovery phase. A positive exercise testing was defined as follows: 1) horizontal or down-sloping ST-segment depression of ≥ 1 mm below the PR isoelectric line or ≥ 1 mm further if there is baseline depression with a duration of ≥ 0.08 s; or 2) ST-segment elevation of ≥ 0.1 mV compared with the resting tracing with a duration of ≥ 0.04 s after the J point in 2 of the inferior or lateral leads or ≥ 0.2 mV in ≥ 2 contiguous precordial leads. Three consecutive complexes conforming to one of these criteria were required for a test to be accepted as abnormal. Angina pectoris was defined as development of exertional or postexertional chest discomfort that was relieved with rest or sublingual nitrates. The duration of each patient's chest pain and heart rate changes were also recorded. Each patient's blood pressure was monitored by indirect sphygmomanometry at 1-min intervals during the test and recovery phase. Exercise end points included physical exhaustion, claudication, angina pectoris, dyspnea, ST-segment depression ≥ 2 mm, ST-segment elevation ≥ 1 mm, sustained ventricular tachycardia and exertional hypotension (≥ 10 mmHg decrease in systolic blood pressure).

^{201}Tl Tomography

Three millicuries (111 MBq) of thallous chloride was administered intravenously approximately 1 min before stopping exercise. Thallium tomographic imaging was performed immediately after stopping exercise and 4 h later. The location of perfusion defects was correlated with the coronary artery vascular territories. Septal and anterior defects were assigned to the left anterior descending artery, inferior and posterior defects to the right coronary artery and lateral defects to the left circumflex artery. Isolated apical perfusion defects were not assigned to any particular coronary artery due to the known variation in vascular supply to the left ventricular apex.

Coronary Angiography and Intracoronary Methylergonovine Testing

After informed consent was obtained, coronary angiography was performed within 7 days after exercise testing

Table 1 Characteristics of Patients With Exercise-Induced ST-Segment Elevation

Pathology character	Patient no.	Age/sex	Angina	Risk factors	Exercise time (s)	HR baseline/maximum	PRP ($\times 10^{-3}$)	Exercise ECG leads with ST-segment elevation	Exercise-related thallium defect	Coronary angiography	LVEF (%)	Medications
Angiographically normal coronary arteries with vasospasm	1	65/M	Rest angina	Age, Gender, Smoking	180	57/95	12.0	II, III, aVF	Inferoposterior	Normal with RCA spasm	73	CA, Ni
	2	70/F	Rest and effort angina	Hypertension, Diabetes mellitus	286	66/128	18.9	III, aVF, V4-6	—	Normal with RCA & LAD spasm	72	CA, Di, Ni
	3	52/F	Rest angina	Smoking	313	68/155	14.6	V2-6	Anteroseptal	Normal with LAD spasm	76	CAs, Ni
	6	63/M	Rest angina	Age, Gender, Hypertension	330	53/107	18.4	V2-6	Anteroseptal	Normal with LAD spasm	65	CA
Hemodynamically significant stenosis	8	42/M	Rest angina	Gender, Smoking	379	51/142	20.7	V2-5	Anteroseptal	Normal with LAD spasm	62	CA
	4	61/M	Rest and Effort angina	Age, Hypercholesterolemia, Diabetes mellitus	332	84/142	20.5	II, III, aVF	Inferior	LM + triple vessel stenoses	81	ACEI, ASA, BB, CA, Di, Ni
	5	41/M	Effort angina	Hypertension, Smoking	657	58/142	23.7	V2-4	—	68% stenosis of mid-LAD	70	ASA, BB, Ni
	9	69/M	Effort angina	Age, Gender, Hypertension, Smoking	262	90/152	28.3	V1-4	—	LAD 83% segmental stenosis	66	ASA, CA, Ni
Coexisting vasospasm and hemodynamically significant stenosis in different arteries	7	41/F	Rest and effort angina	Hypertension	318	64/109	18.3	II, III, aVF, V2-6	—	Normal RCA with spasm, 70% stenosis of LAD orifice	72	ARB, ASA, CA, Ni

HR, heart rate; PRP, pressure-rate product; ECG, electrocardiographic; LVEF, left ventricular ejection fraction; RCA, right coronary artery; CA, calcium antagonist; Ni, nitrate; —, not done; LAD, left anterior descending coronary artery; Di, diuretic; LM, left main coronary artery; ACEI, angiotensin-converting enzyme inhibitor; ASA, aspirin; BB, -blocker; ARB, angiotensin-receptor blocker.

Fig 1. Baseline electrocardiogram (A); peak-exercise electrocardiogram (B); thallium-201 (^{201}Tl) tomograms during peak-exercise (C) and 4 h afterward (D); left coronary arteriogram after intracoronary methylergonovine administration (E); and left coronary arteriogram after intracoronary nitroglycerin administration (F) of patient 3. The patient developed exercise-induced anterior lead ST-segment elevation and anterior segment hypoperfusion as compared with ^{201}Tl image at 4 h after peak exercise. The left coronary arteriogram shows coronary vasospasm in the mid-portion of the left anterior descending artery (arrows).

Fig 2. (A) Baseline electrocardiogram at presentation of patient 8; (B) exercise-induced ST-segment elevation in leads V2-5; (C) thallium-201 (^{201}Tl) tomogram: left panel shows perfusion defect of the anterior wall at peak exercise and right panel shows redistribution of anterior wall on image at 4 h after peak exercise; (D) follow-up baseline electrocardiogram 2 years later; (E) no exercise-related ST-segment elevation on follow-up exercise electrocardiogram; (F) follow-up ^{201}Tl tomogram at peak exercise (Left panel) and 4 h after stopping exercise (Right panel) showing no evidence of perfusion defect; (G) angiographically normal left anterior descending artery (arrow); (H) diffuse 80–90% diameter reduction (arrow) after intracoronary methylergonovine administration.

Fig 3. Baseline electrocardiogram (A); peak-exercise electrocardiogram (B); thallium-201 (^{201}Tl) tomograms during peak exercise (C) and 4 h afterward (D); right coronary arteriogram (E); and left coronary arteriogram (F) of patient 4. This patient developed exercise-induced inferior leads ST-segment elevation and more severe inferior segment hypoperfusion as compared with the ^{201}Tl image at 4 h after stopping exercise. The coronary arteriograms show left main and triple vessel coronary artery stenoses with the most critical stenosis (85%) in the distal portion of right coronary artery (arrow).

using the standard Judkins technique via a femoral or a radial approach. Nitrates and calcium antagonists were withdrawn for ≥ 24 h prior to coronary angiography. Selective left and right coronary angiographies were done in multiple axial and hemiaxial projections. Hemodynamically significant coronary stenosis was defined as $\geq 50\%$ diameter reduction in lumen caliber after 100 mg intracoronary nitroglycerin administration. Intracoronary methylergonovine (Methergin®, Novartis, Basle, Switzerland) provocation testing was performed in succession if no hemodynamically significant coronary stenosis was found. The stepwise dose (1, 5, 10, 30 μg) administration of methylergonovine into the right coronary artery was performed first into the left coronary artery. Positive provocation testing for coronary vasospasm was considered to be positive when a reduction in lumen diameter $>70\%$ occurred during provocation testing and was associated with angina and/or ST depressive or elevated changes. After coronary vasospasm was diagnosed, the intracoronary methylergonovine administration was stopped and the reversal was achieved with 50–100 μg intracoronary nitroglycerin (Millisrol®, G. Pohl-Boskamp, Hohenlockstedt, Germany) administration. The observation of reversal of coronary artery diameter change further confirmed the diagnosis of coronary vasospasm. Measurements of the coronary artery lumen diameters before and after administration of methylergonovine and after administration of nitroglycerin were performed by at least 2 persons who were not aware of the exercise electrocardiographic changes.

Patient Follow-up

Follow-up data for all patients were obtained from hospital records, personal communication with the patient's physicians, telephone interviews and the patient's visit to staff physicians at regular intervals in the outpatient clinic. Events were defined as death, non-fatal reinfarction and recurrent angina. Cause of death was further classified as cardiac or noncardiac.

Results

A total of 6,147 consecutive patients without prior myocardial infarction underwent modified Bruce protocol of treadmill exercise testing at our institution. Nine (0.15%) patients developed ST-segment elevation during the exercise testing. The characteristics of the patients are listed in Table 1. The age range of the patients was 41–70 years. Six patients were men and 5 of the 6 men were cigarette smokers, which was the most common cardiac risk factor in this series. All patients had attacks of angina that recurred more than 5 times per week at the time of study. Rest angina was the most common presentation in patients with coronary vasospasm.

All patients had chest pain and ST-segment elevation during the exercise testing, which was relieved after sublingual nitroglycerin or rest. Exercise testing was performed in the mid-morning in 4 patients (80%) with coronary vasospasm (patient 1, 3, 6, and 8) and in 1 patient with hemodynamically significant coronary stenosis (patient 4). The

Fig 4. Baseline electrocardiogram of patient 9 shows incomplete right bundle branch block and T-wave inversion in leads V2-4 (A). The patient developed exercise-induced ST-segment elevation in leads V1-4 (B). The corresponding coronary stenosis is 85% segmental stenosis in the mid-portion of the left anterior descending artery (C, arrowheads), which was not relieved after intracoronary nitroglycerin administration (D, arrowheads).

Fig 5. (A) Baseline electrocardiogram of patient 7; (B) exercise-induced ST-segment elevation in leads II, III, aVF and V2-6; (C) angiographically normal right coronary artery (arrow); (D) diffuse diameter reduction with 70% diameter reduction (arrow) in the mid-portion of the right coronary artery after intracoronary methylergonovine administration; (E) 70% eccentric discrete stenosis in the orifice of left the anterior descending artery (arrowhead); (F) left anterior descending orifice stenosis is still present (arrowhead) after intracoronary nitroglycerin administration.

median duration of exercise, indicating the time of ST-segment elevation, was 313 s (range, 180–279) in patients with coronary vasospasm, 332 s (range, 262–657) in patients with hemodynamically significant coronary stenosis, and 318 s in the patient who had coexisting spasm and stenosis

in coronary arteries. The location of ST-segment elevation was in the anterior leads in 5 patients, inferior leads in 2 and both anterior and inferior leads in 2. In 5 patients who had undergone exercise ^{201}Tl tomography, the transient perfusion defect was consistent with the leads showing ex-

ercise-induced ST-segment elevation. Coronary angiograms revealed that the stenotic vessel or spastic vessel corresponded to the findings of exercise-induced ST-segment elevation. Five patients had angiographically normal coronary arteries with corresponding coronary vasospasm as follows: patient 3 (Fig 1), 6 and 8 (Fig 2) had provocative spasm of the left anterior descending artery; patient 1 had spasm of the right coronary artery; and patient 2 had spasm of both the left anterior descending artery and the right coronary artery. Three patients (patient 4, 5 and 9) had hemodynamically significant coronary stenosis: patient 4 had left main and triple vessel stenoses (Fig 3); patient 9 had 83% segmental stenosis in the mid-portion of left anterior descending artery (Fig 4). Patient 7 had coexisting 70% stenosis in the orifice of the left anterior descending artery and spasm of the right coronary artery (Fig 5). All patients had adequate left ventricular contraction with no evidence of ventricular wall motion abnormality.

Calcium antagonists were prescribed to all patients except patient 5, who received β -blockers, aspirin, and nitrate. Patient 8 underwent follow-up treadmill exercise testing with exercise ^{201}Tl tomography 2 years after treatment, which revealed no evidence of myocardial ischemia on electrocardiogram and no perfusion defect on thallium study (Fig 2D,E and F). During a median follow-up of 71 months (range 38–83), 2 patients (patient 1 and 3) had recurrent angina that developed after self-discontinuation of medications (calcium antagonists) in both patients. The angina recurrences were soon relieved after resumption of previous medications. Patient 2 developed end-stage renal disease and required maintenance hemodialysis 2 years after the positive exercise testing. Patient 2 and 4 died on arrival at the emergency department 3.5 years after positive exercise testing, with cardiac death listed as the presumed cause.

Discussion

The present study had 2 main findings: first, coronary vasospasm was the most common underlying pathology of exercise-induced ST-segment elevation in Taiwanese patients without prior myocardial infarction, followed by hemodynamically significant coronary stenosis. Second, coronary angiography $\pm$ intracoronary ergonovine provocation testing was necessary to identify the underlying pathology in these patients because of the variation in possible underlying causes of coronary vasospasm, hemodynamically significant coronary stenosis and coexisting spasm in an angiographically normal coronary artery with hemodynamically significant stenosis of the remaining vessels.

Previous Studies

In postinfarction patients, exercise-induced ST-segment elevation usually occurs in the electrocardiographic leads with pathologic Q waves and is considered to be secondary to left ventricular dyssynergy or aneurysm rather than ischemia.^{1–9} Contrary to previous studies of patients with myocardial infarction, our data suggest that in those patients without infarction, ST-segment elevation during exercise is a marker for transient and severe ischemia. Waters et al studied 11 patients without previous myocardial infarction who had a large, reversible, exercise-induced perfusion defect on ^{201}Tl scintigraphy in a vascular territory corresponding to the electrocardiographic leads with ST-segment elevation.⁶ In 10 of their 11 patients, vari-

ant angina had been documented before exercise testing. Coronary angiography without ergonovine provocation testing revealed angiographically normal coronary arteries in 3 patients. On repeat exercise testing in 4 variant angina patients after treatment with nifedipine, no angina, ST-segment elevation or perfusion defects occurred. Lahiri et al studied 5 patients without prior infarction who had severe ($\geq 90\%$) coronary artery stenoses and ST-segment elevation after exercise.¹⁰ ^{201}Tl tomography showed reversible perfusion defects in areas corresponding to the leads of exercise-induced ST-segment elevation in all patients. Gallik et al studied 12 patients without previous myocardial infarction who also had average reversible perfusion defects of $33.5 \pm 13\%$ on ^{201}Tl tomography.¹¹ They found that only 2 patients had probable coronary vasospasm and insignificant coronary stenosis without confirmation by ergonovine provocation testing. The electrocardiographic changes and tomographic perfusion defects had resolved in these patients after medical therapy with calcium antagonists and nitrates. The role of coronary vasospasm in exercise-induced ST-segment elevation has been documented directly using coronary angiography during exercise.^{12,13}

Present Study

In the present study, the patients without suspected variant angina underwent routine exercise testing, which led to the incidental finding of exercise-induced ST-segment elevation. This method of diagnosis is unlike some previous studies in which patients had suspected or documented variant angina prior to exercise testing.^{6,12,13} The prevalence of exercise-induced ST-segment elevation in patients with variant angina is difficult to ascertain because patients with variant angina made no attempt to exercise routinely in most studies. Waters et al showed that in patients with variant angina, the exercise-induced ST-segment elevation was noted in 25 of 82 (30%) patients with variant angina.¹⁴ They found that: 1) ST-segment elevation occurred in the same electrocardiographic leads as during spontaneous attacks at rest; and 2) exercise-induced ST-segment elevation in patients with variant angina correlated well with the degree of disease activity but not with coronary anatomy. The incidence (6 in 9 (67%) patients, including patient 7) of angiographically normal coronary arteries with vasospasm in patients with exercise-induced ST-segment elevation in the present study was higher than in previous studies (0–31%).^{4–7,10–12} The racial heterogeneity in coronary artery vasomotor reactivity as found in our previous studies^{15,16} and by other investigators might be an explanation.^{17–20} Interestingly, 1 patient in this study who had coexisting right coronary vasospasm and a 70% hemodynamically significant stenosis in the orifice of the left anterior descending artery developed exercise-induced ST-segment elevation in both the inferior and anterior leads. To our knowledge, the combination of coexisting spasm in angiographically normal coronary artery and hemodynamically significant stenosis in the remaining vessel resulting in exercise-induced ST-segment elevations has not been previously reported. The coexistence of these conditions would not have been identified without coronary angiography. The failure to identify the role of coronary vasospasm in the presenting symptom of exercise-induced ST-segment elevation would then result in ineffective therapy.

In the present study, there were no patients with left circumflex coronary vasospasm. It is plausible that left circumflex coronary vasospasm is uncommon. However,

ST-segment depression as reciprocal change is observed in precordial leads (especially in leads V₂₋₄) but ST-segment elevation is not observed in any leads in a 1-lead electrocardiogram (ST-segment elevation is observed in only V₇₋₉) if spasm of the left circumflex coronary artery, which supplies only the posterior wall, is induced by exercise. There is some possibility of not detecting spasm of the left circumflex coronary artery, which supplies only the posterior wall.

Possible Mechanisms of Exercise-Induced Coronary Vasospasm

Exercise has been shown to induce constriction in minimal (<30% stenosis) and advanced (≥50% stenosis) disease of atherosclerotic coronary arteries.²¹ The exercise-induced imbalance in myocardial oxygen supply and demand, combined with varying degrees of altered vasomotor tone, caused ischemia in our patients with and without hemodynamically significant stenosis, which supports that the vasospastic coronary artery is not a normal coronary artery as was found in previous reports.^{22,23} The mechanisms responsible for exercise-induced coronary artery spasm, however, remain elusive. Yasue et al found that coronary vasospasm can be induced by an intracoronary injection of acetylcholine and prevented by premedication with atropine, which suggests that the parasympathetic nervous system might play a role in the pathogenesis of coronary vasospasm.²⁴ Tsuchiya et al noted that in patients with multivessel coronary vasospasm, as compared with patients with single vessel coronary artery spasm, parasympathetic activity decreased and sympathetic activity increased.²⁵ We also reported that the isoproterenol head-up tilt test could provoke coronary artery spasm, and we speculated that both increased basal parasympathetic tone and strong sympathetic stimulation are important in causing coronary vasospasms.²⁶ In our patients, the poor response of pressure-rate product during exercise suggested a strong basal parasympathetic tone. The sympathetic discharge is maximal and parasympathetic stimulation is withdrawn during strenuous exercise. The maximal sympathetic discharge during exercise and preexisting strong parasympathetic tone withdrawal in our patients is likely to have resulted in exercise-induced coronary vasospasm.

Possible Mechanisms of Coronary Vasospasm

Previous studies involving histological evaluation of coronary plaques or arteries in patients with variant angina have reported evidence of intimal injury, such as neointimal hyperplasia with infiltration by inflammatory cells.^{22,27} These findings indicate that early inflammatory changes might play a part in initiating atherosclerotic lesion formation in spastic coronary arteries and, as suggested also by other investigators, in its progression.^{28,29} Previous studies have shown elevated coronary levels of soluble intercellular adhesion molecule-1 or serum lipoprotein(a) in patients with coronary spastic angina.^{30,31} The results of our recent investigations also suggest elevated C-reactive protein level in patients with coronary vasospastic angina pectoris who do not have hemodynamically significant coronary artery disease, implying the presence of endothelial dysfunction with atherosclerotic changes.^{32,33} Recently, Soejima et al, reported a significant increase in activated T cells in patients with both unstable angina and coronary spastic angina.³⁴ Further, these investigators found that coronary spastic angina is a frequent complication in cases involving

connective tissue disease, and that the inflammatory condition is associated with coronary spastic angina and unstable angina in these patients.³⁵ They also found higher serum C-reactive protein levels in patients with coronary spastic angina relative to analogs with non-ischemia and stable exertional angina. Further, the serum C-reactive protein level was lower in vasospasm than that observed in acute coronary syndrome. As a result, therefore, it appears reasonable to speculate that coronary vasospasm is an early inflammatory coronary artery condition, given the presence of endothelial dysfunction with resulting diffuse intimal thickening and impaired nitric oxide production. Nevertheless, the endothelial dysfunction and impaired nitric oxide production cannot result in coronary vasospasm without vascular smooth muscle contraction. Accumulating evidence indicates that Rho-kinase is substantially involved in the pathogenesis of coronary vasospasm.³⁶⁻³⁸ On the other hand, the autonomic system innervating the coronary arteries also plays an important role in the process of coronary vasospasm, as suggested by previous investigators.^{23-25,39}

Conclusions

The limited experience of the present study suggests that the coronary angiography ± intracoronary ergonovine provocation testing should be performed in all patients with exercise-induced ST-segment elevation in order to identify underlying pathology, as this is the only way to find a definitive indication for the choice of treatment. Our findings also support that exercise-induced myocardial ischemia can be abolished using correct medical therapy in patients with coronary vasospasm and no hemodynamically significant coronary stenosis.

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