Follow-up Exercise Electrocardiography Can Confirm the Appropriateness of Treatment for Exercise-induced Myocardial Ischemia and Life-threatening Cardiac Arrhythmias due to Coronary Vasospasm

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To the Editor: Exercise-induced ST-segment elevation is an uncommon clinical scenario with a prevalence of 0.15%.[1] ST-segment elevation during exercise is a marker of both transient and severe ischemia. Appropriate treatment is crucial because coronary vasospasm can result in acute coronary syndrome, syncope, life-threatening cardiac arrhythmias, and even out-of-hospital cardiac arrest.[2] Here, we report on a patient with exercise-induced myocardial ischemia and life-threatening cardiac arrhythmias due to coronary vasospasm.

A 70-year-old man with a history of cigarette smoking, benign prostate hyperplasia, and hypertension presented to the cardiology outpatient clinic with a 2-month history of dyspnea on exertion. His hypertension had been well-controlled with bisoprolol 5 mg once daily for more than 2 years. The exertional dyspnea was associated with general weakness that was soon relieved after resting. Because the patient had multiple cardiovascular risk factors, he underwent treadmill exercise electrocardiography for possible coronary artery disease. Sinus arrest with junctional escape rhythm of 60 beats/min was noted at the beginning of the examination. No management was performed since there were no subjective symptoms and no hemodynamic changes. Sinus tachycardia with ST-segment depression in leads V_{3-6} was noted on peak exercise, reaching 76% of the maximal, age-predicted heart rate [Figure 1a]. Electrocardiographic examination revealed atrioventricular dissociation rhythm and ST-segment elevation in the inferior leads within minutes after reclining on the bed. The electrocardiogram then demonstrated complete atrioventricular block with a ventricular rate of 24 beats/min and persistent ST-segment elevation in the inferior leads [Figure 1b]. Sublingual nitroglycerin 0.6 mg resulted in the gradual resolution of ST-segment elevation in inferior leads; however, complete atrioventricular block persisted. After resting for 7 min, Wenckebach second-degree atrioventricular block with anterolateral wall T-wave inversion was noted on the electrocardiogram. At 9 min, the electrocardiogram showed first-degree atrioventricular block with resolving T-wave inversion in the anterolateral wall [Figure 1c]. Laboratory test results revealed normal findings with the exception of elevated serum creatinine (1.36 mg/dl; reference range: <1.27 mg/dl) and elevated high-sensitivity C-reactive protein (5.12 mg/L; reference range: <1.0 mg/L). Coronary angiography revealed no evidence of obstructive coronary artery disease in the right and left coronary arteries. Intracoronary methylergonovine provocation testing resulted in near-total spasm of the right coronary artery after administration of only 5 μg methylergonovine into the right coronary artery [Figure 1d]. The spasm was relieved, however, after intracoronary administration of 200 μg nitroglycerin [Figure 1e]. No inducible left coronary vasospasm was noted subsequently. Right coronary vasospasm complicated by exercise-induced variant angina and complete atrioventricular block was diagnosed. The patient was treated with nifedipine extended release 30 mg once daily at night and was advised to abstain from cigarette smoking. To determine whether the treatment was effective, treadmill exercise electrocardiography was performed at 1-year follow-up. At the beginning of the test, sinus rhythm with first-degree to Mobitz type II second-degree atrioventricular block was noted and no more inducible myocardial ischemia at the peak exercise that reached 85% of the maximal, age-predicted heart rate [Figure 1f]. Atrioventricular dissociation rhythm with a ventricular rate of 80–85 beats/min was noted on recovery without myocardial ischemic changes. The patient was doing well without any evidence of angina pectoris. In normal physiology, coronary arteries are dilated in response to exercise to supply adequate oxygen demand of myocardium. Exercise has been shown to induce constriction in minimal (<30%) and advanced (≥50% stenosis) atherosclerotic coronary arteries.[3] The exercise-induced imbalance in myocardial oxygen supply and demand, combined with varying degrees of altered vasomotor tone resulted in myocardial ischemia in our patient, indicating that the vasospastic coronary artery is not a normal coronary artery as

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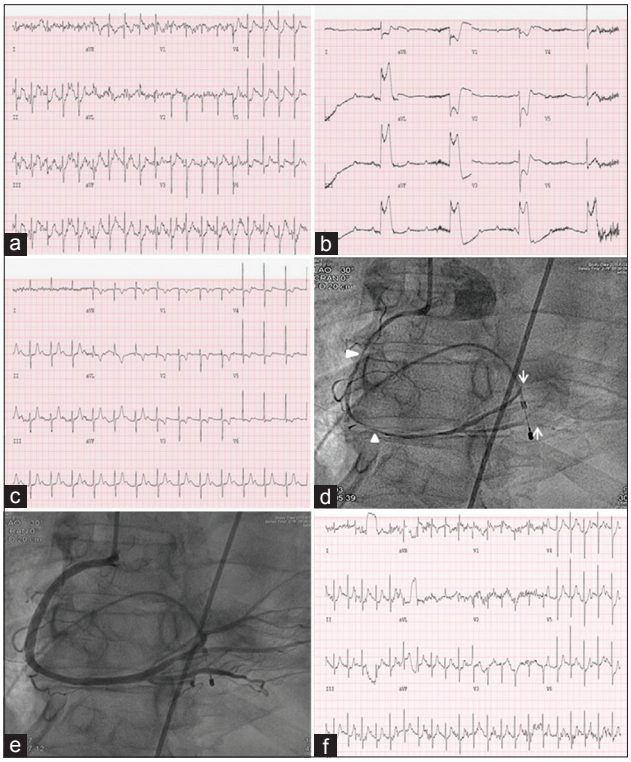

Figure 1: Baseline exercise electrocardiogram on peak exercise (a); complete atrioventricular block and ST-segment elevation in inferior leads (b); and first-degree atrioventricular block with resolving T-wave inversion in the anterolateral wall after resting for 9 min (c); Nonobstructive right coronary artery with provoked spasm (arrows and arrowheads, d); relief of the spasm (e); Follow-up exercise electrocardiogram on peak exercise (f).

reported previously.[4] However, the precise mechanisms responsible for exercise-induced coronary vasospasm remain elusive. In our case, the inadequate age-predicted heart rate at peak exercise suggests a strong basal parasympathetic tone. The ST-segment elevation and atrioventricular dissociation rhythm during the recovery phase suggest preexisting strong parasympathetic tone reactivation after strong sympathetic stimulation at peak exercise. An imbalance of the autonomic system may be responsible for the myocardial ischemia at peak exercise and total right coronary vasospasm causing ST-segment elevation in the inferior leads complicated by complete atrioventricular block during the recovery phase.

The results of exercise electrocardiography at 1-year follow-up confirmed the initial diagnosis of coronary vasospasm-induced myocardial ischemia. Calcium antagonists were prescribed for our patient. This main treatment abolished the coronary vasospasm-related myocardial ischemia and its complication as demonstrated by the same stress testing. Repeated stress testing at follow-up confirmed that the treatment chosen for this patient with exercise-induced myocardial ischemia due to coronary vasospasm was appropriate. Although vasospastic angina can usually be controlled by calcium antagonists and nitrates, sometimes vasospastic angina can be intractable with an incidence of 13.7% reported in Japan.[4] The Rho-kinase inhibitor, fasudil, has been demonstrated to be effective in patients with intractable vasospastic angina.[4] Percutaneous coronary intervention may be beneficial only for vasospastic angina with severe organic stenosis.[4] Because silent myocardial ischemia due to coronary vasospasm is not unusual, only clinical follow-up may underestimate the possible recurrence of vasospastic angina.[4] Our present findings further support that follow-up same stress testing could evaluate safely about the accuracy of initial diagnosis, beneficial effect of treatment, and the disease's complication during exercise.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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