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CASE REPORT

Significance of intravascular ultrasound and exercise stress echocardiography in diagnosis of exercise-induced vasospastic angina at the site of moderate stenosis

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Abstract Recently, exercise-induced spastic coronary artery occlusion at the site of moderate stenosis, which Prinzmetal's angina or cardiac syndrome X does not cover, was reported. Multi-modality imaging is important for the diagnosis of coronary artery disease with a complex ischemic mechanism. However, the previous report did not include findings from intracoronary imaging at the site of moderate coronary stenosis. We report a case of exercise-induced vasospastic angina at the site of moderate stenosis, where multi-modality imaging, including exercise stress echocardiography and intravascular ultrasound, was utilized to make a definitive diagnosis and investigate underlying causes.

Keywords Vasospastic angina · Intracoronary imaging · Stress echocardiography

Introduction

The role of coronary artery spasm is well established in variant angina, which occurs at rest, particularly at night, and in the early morning, and is associated with ST elevation on ECG. Specchia et al. [1] reported that exercise could trigger a coronary vasospasm, thus inducing angina pain and ST-segment elevation in their case series. Yilmaz et al. [2] recently reported a case of exercise-induced vasospasm at the site of coronary stenosis. However, their report did not include findings from intracoronary imaging. We report a case in which exercise-induced vasospastic angina at the site of moderate stenosis was diagnosed by multi-modality imaging including intravascular ultrasound (IVUS) and exercise stress echocardiography.

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Case

A 77-year-old woman was transferred to our hospital because of recurrent episodes of chest pain on exertion over 6 months. Holter ECG, recorded 3 days prior to the hospitalization, had demonstrated ST-segment elevation with atrioventricular blocks, while she was experiencing chest pain on exertion (Fig. 1). Her past medical history includes hypertension, for which she takes bisoprolol 5 mg daily and candesartan 4 mg daily, and mitral regurgitation, for which she underwent mitral valve repair at the age of 70 years. On admission, cardiac enzymes were not elevated and ECG revealed no ischemic changes without symptoms at rest. Transthoracic echocardiography revealed normal left ventricular wall motion with preserved ejection fraction (66%). Coronary angiography demonstrated 50% stenosis in the distal right coronary artery (Fig. 2a). IVUS of the lesion showed moderate stenosis with concentric fibrous plaque (plaque burden of 67%). There was no sign of thrombus or plaque rupture at the site of the stenosis (Fig. 2b). Because of a suspicion of coronary vasospasm as an underlying cause of the angina, pharmacological provocation with intracoronary ergonovine was performed. When 15 μg of ergonovine was infused into the right coronary artery, a coronary occlusion occurred at the site of the stenosis with typical angina and ST elevation in inferior leads (Fig. 2c). Because we considered that her chest pain was caused by coronary vasospasm associated with the

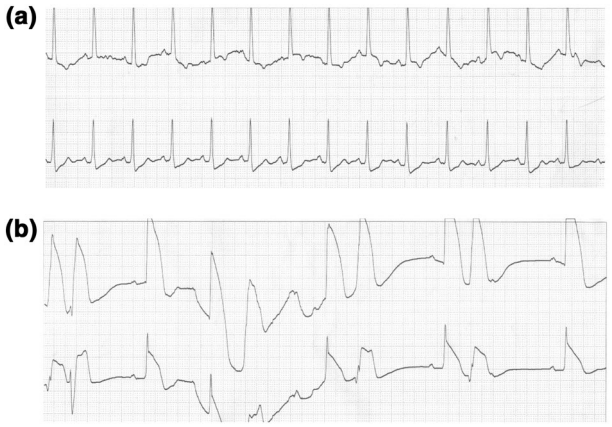

Fig. 1 Holter ECG demonstrating no ST changes at rest (**a**), but demonstrating ST elevation and complete atrioventricular block with chest pain during exercise (**b**)

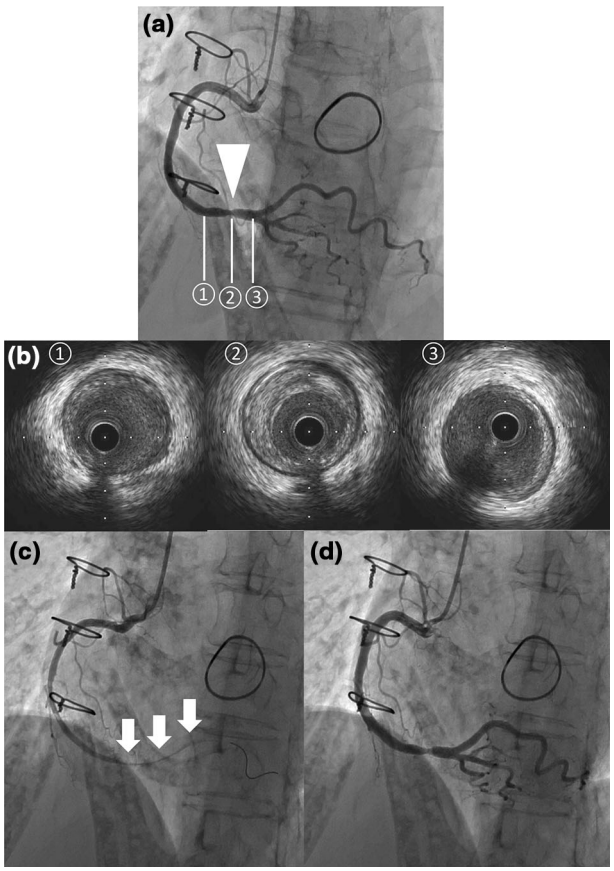

Fig. 2 Angiography revealed 50% stenosis in the mid portion of the right coronary artery (*white arrow head*) (**a**). Intravascular ultrasound (IVUS) showed fibrous plaque with no evidence of thrombus or plaque rupture (**b**). When 15 μg of ergonovine was infused into the right coronary artery, a coronary occlusion occurred at the same site of the stenosis (*white arrow*) with recurrent angina and ST elevation in leads II, III, and aVF (**c**). After intracoronary nitroglycerin injection, the RCA occlusion disappeared (**d**)

effect of beta blockade, bisoprolol 5 mg was discontinued. Three days later, she underwent treadmill exercise stress echocardiography to see whether the exercise-induced chest pain was reproducible. At 4 METs of stress exposure, she complained of chest pain with ST-segment elevation in the inferior leads and akinesis of a left ventricular inferior wall (Fig. 3b). Therefore, we considered exercise-induced coronary vasospasm at the site of moderate stenosis as an underlying cause of her symptom. Diltiazem 100 mg twice daily and benidipine 12 mg once daily were added. Five days later, repeated exercise stress echocardiography revealed improved exercise tolerance with no episodes of chest pain, ST-segment elevation, or wall motion abnormalities at 10 METs (Fig. 3d). Repeated Holter ECG as an outpatient also demonstrated no ischemic events.

Discussion

Yilmaz et al. [2] reported a case of exercise-induced vasospasm at the site of coronary stenosis, which Prinzmetal's angina or cardiac syndrome X does not cover. However, their report did not include findings from intracoronary imaging, such as IVUS and optical coherence tomography. It is important for an understanding of a complex ischemic mechanism with ST-segment elevation to evaluate pathological features of coronary arteries with advanced intracoronary imaging modality. To the best of our knowledge, our report is the first to include IVUS images in a case with exercise-induced vasospasm at the site of moderate stenosis diagnosed by multi-modality imaging including exercise stress echocardiography.

Beta blockers reduce myocardial oxygen demand and have been widely used in the treatment of exertional angina. On the other hand, the use of beta blockers in vasospastic angina might worsen spasms by converting the effect of a sympathetic stimulus into a pure alpha-adrenergic vaso-constrictor response [3]. Yasue et al. [4] reported that treadmill exercise-induced angina attacks in 8 of 13 patients (61.5%) with vasospastic angina were aggravated using propranolol, a beta-adrenergic blocking agent. However, diltiazem, a calcium antagonistic coronary vasodilator, was effective in suppressing the attacks in 100% of the same 13 patients [4]. According to their report, a calcium antagonistic coronary vasodilator, which dilates large coronary arteries by blocking the entry of calcium ions into the coronary vascular smooth muscle cells and which is effective in the treatment of Prinzmetal's variant angina, suppressed the attacks in the patients with exercise-induced vasospastic angina. In the present case, the ineffectiveness of a beta blocker and the effectiveness of a calcium antagonistic coronary vasodilator were consistent with the previous reports.

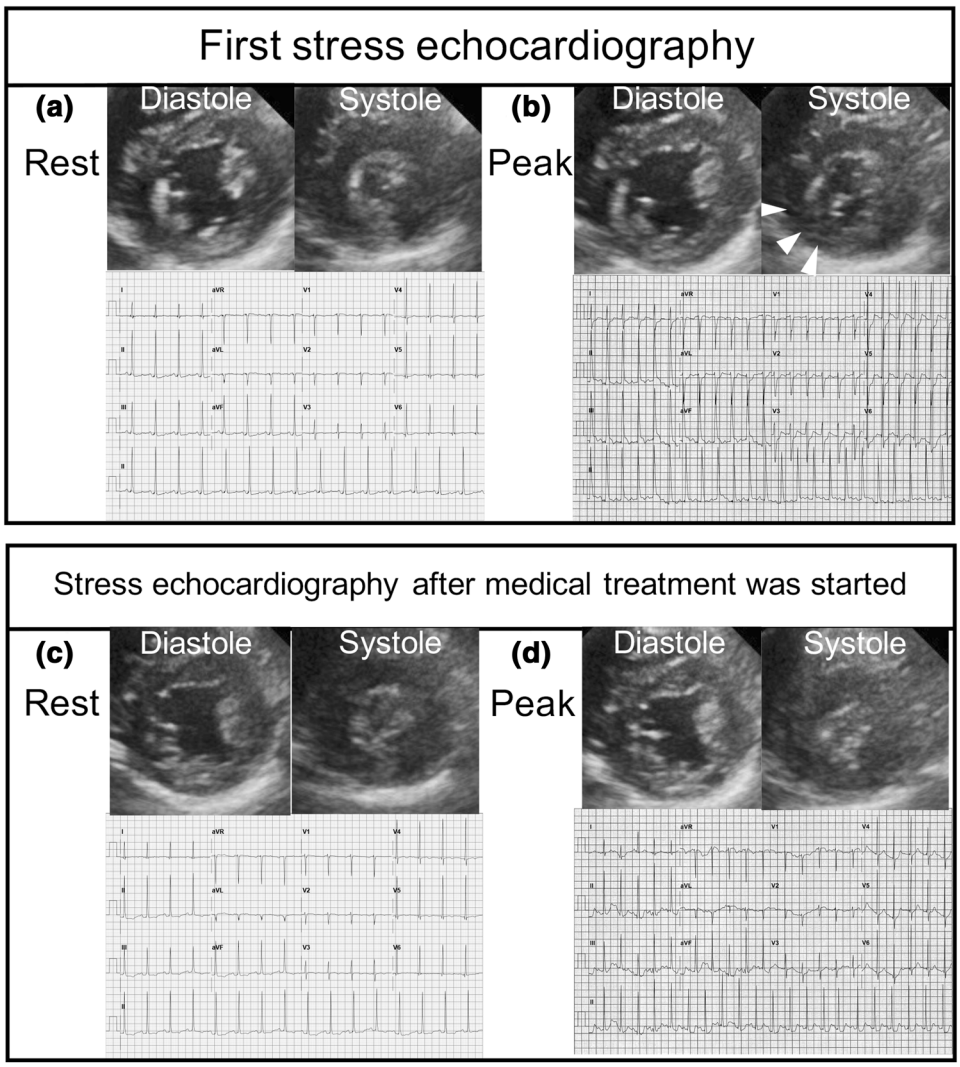

Fig. 3 Initial stress echocardiography with ECG recording (**a**, **b**). At rest, ECG was normal, and echocardiography demonstrated normal wall motion (**a**). At 4 METs of exercise, ST-segment elevation in leads II, III, and aVF was observed and akinesis of the left ventricular inferior wall was demonstrated (*white arrow head*) (**b**). Repeated stress echocardiography (**c**, **d**) demonstrated normal wall motion with no chest pain at 10 METs (**d**)

Conclusion

The combination of anatomical and functional assessments, including multi-modality imaging, is very helpful for evaluating the mechanism of ischemia and diagnosing exercise-induced coronary vasospasm at the site of moderate stenosis.

Compliance with ethical standards

Conflict of interest The authors declare that they have no competing interests.

Ethical statements Additional informed consent was obtained from all patients for which identifying information is included in this article.

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