

Coronary Artery Spasm During Dobutamine Stress Echocardiography: A Case Report

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ABSTRACT

Dobutamine stress echocardiography (DSE) has been widely used as a diagnostic and prognostic modality in the management of stress. DSE is associated with limited complications and adverse effects on the health of patients. In this case report, we described a 42-year-old female patient with dobutamine-induced coronary artery spasm with history of exertional dyspnea, which had deteriorated recently. No risk factors of coronary artery disease were observed in the patient, and she had previous non-diagnostic exercise tolerance test. DSE was performed on the patient, and at the end of the infusion rate of 30 mcg/min, retrosternal pain was detected. Standard 12-lead electrocardiogram was indicative of ST segment elevation in inferior leads. Moreover, echocardiographic imaging of the patient revealed concomitant akinesia in the right coronary artery. On the other hand, subsequent coronary angiograms showed only mild coronary atherosclerosis.

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Introduction

Dobutamine stress echocardiography (DSE) is a non-invasive diagnostic and prognostic modality used in the stress management of patients with coronary artery obstructive disease. Dobutamine infusion increases myocardial demand due to its positive inotropic and chronotropic effects. In the presence of significant coronary artery disease, dobutamine infusion leads to new or worsening left ventricular regional wall motion abnormalities. Normally, these myocardial changes are not associated with electrocardiographic (ECG) ST-segment depression or elevation. ST-segment elevation with concomitant wall motion abnormality occurs not only in the presence of severe coronary obstruction, but also as a

consequence of arterial wall shear and coronary spasm, even in patients with no significant coronary obstruction (1).

Case presentation

In this study, we describe the case of a 42-year-old female patient with exertional dyspnea during the past year, which had deteriorated within the last few weeks. The patient presented with nonconventional risk factors of coronary artery disease, history of systemic disorders, and alcohol consumption or drug abuse.

In her physical examination, blood pressure was 120/80mmHg and heart rate was 72 beat per minute. Cardiac auscultation revealed normal S1

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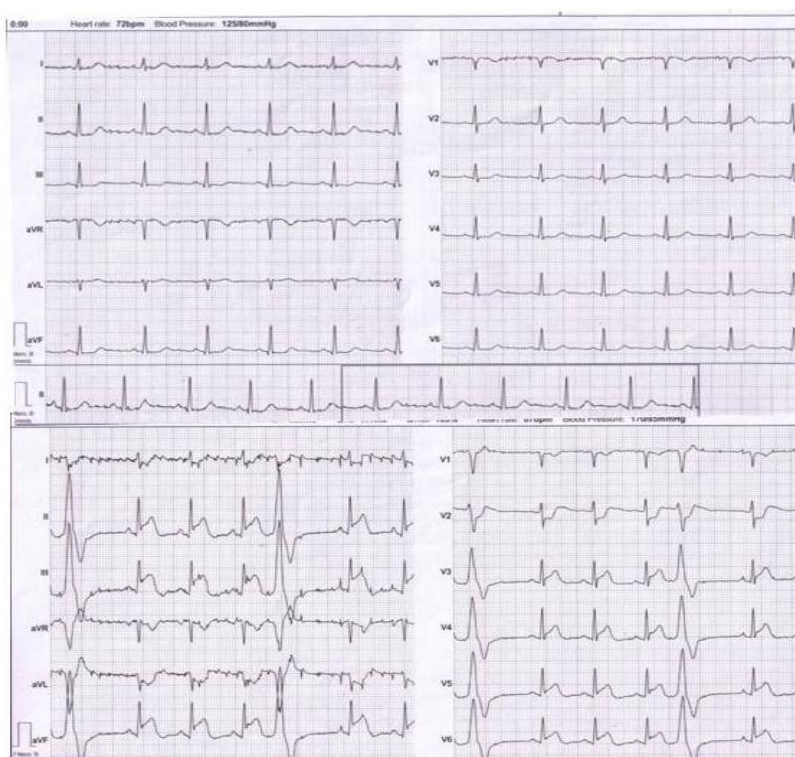


Figure 1. Results of resting 12-lead electrocardiogram showing the absence of ST-T changes (a); ST-segment elevation in V5, V6, and inferior leads with concomitant reciprocal changes in leads I, aVL, V1, and V2 with frequent premature ventricular complexes during dobutamine infusion period of 30 mcg/min (b)

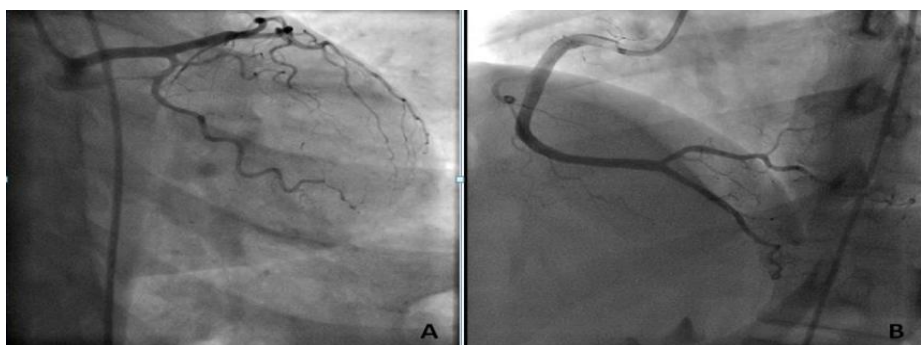


Figure 2. Selective coronary angiograms of left (a) and right (b) coronary arteries showing irregular luminal border of right coronary artery

and S2 without murmur. Lung was clear. Laboratory examinations revealed no signs of cardiovascular abnormalities. Moreover, the patient had normal resting 12-lead ECG and non-diagnostic exercise tolerance test.

DSE was performed on the patient at the initial infusion rate of 5 mcg/min, increasing stepwise to 10, 15, 20, and 30 mcg/min at three-minute intervals. At the end of the infusion rate of 30 mcg/min, the patient experienced severe chest pain. At this stage, ECG of the patient was indicative of ST-segment elevation in V5, V6, and inferior leads, as well as the associated reciprocal changes in leads I, aVL, V1, and V2. In addition, echocardiographic imaging showed akinesia in the inferior left ventricular wall and right ventricular free wall. At this point, heart rate of the patient was 90 beats/min, and blood pressure

was 170/85 mmHg. Therefore, dobutamine infusion was discontinued immediately. After the administration of sublingual nitroglycerin, the chest pain subsided rapidly, and ST segments returned to baseline. Furthermore, the wall motion abnormalities resolved completely after two minutes.

The patient was scheduled for coronary angiography immediately after dobutamine stress test, which were performed on the same day. Since Mild coronary atherosclerosis (Figure 2), the patient was discharged on the following day and prescribed with oral calcium channel blocker.

Discussion

As a diagnostic and prognostic modality, DSE has been widely used for patients with known or

suspected coronary artery disease in order to assess myocardial viability in cases presenting with left ventricular dysfunction. New or worsening regional wall motion abnormalities could be indicative of significant coronary artery disease. Although DSE is a safe modality in stress management, it has been associated with various complications, including cardiac rupture, myocardial infarction, cerebrovascular accidents, cardiac asystole, ventricular fibrillation, sustained ventricular tachycardia, supra-ventricular arrhythmias, atrioventricular block, coronary spasm, hypotension and hypertension, atropine intoxication, and hypersensitivity to dobutamine (2).

The exact incidence rate of dobutamine-induced coronary artery spasm remains unknown, while in one report, it was estimated at 0.4% (3).

Primarily, dobutamine exerts a vasodilatory effect on the normal endothelial function of coronary artery through β_2 -adrenergic receptor stimulation. On the other hand, coronary artery spasm during dobutamine infusion could result from α_1 -receptor-mediated constriction, particularly in patients with endothelial dysfunction associated with smoking, hypertension, and diabetes mellitus. Furthermore, patients with coronary artery endothelial dysfunction may present with deficient nitric oxide activity, which could lead to abnormal vasoconstrictive response after dobutamine infusion (4).

High-dose dobutamine infusion has been reported to cause anxiety and hyperventilation in as much as 6% of patients leading to respiratory alkalosis, which enhances Na/H exchange followed by Na/Ca²⁺ exchange. This process has been shown to increase intracellular calcium concentrations (5).

According to the literature, dobutamine has sympathomimetic activity and the ability to release vasoactive substances from platelets, which may potentially contribute to the occurrence of coronary artery spasm (6).

In the absence of intraluminal atherosclerotic narrowing, myocardial bridging is considered to be another mechanism involved in myocardial ischemia (7). In such cases, intimal irregularities might be observed in the coronary angiogram without significant stenosis.

In the patient described in the current study, coronary artery spasm during DSE was suspected to be the cause of wall motion abnormalities and the associated ST-segment elevation. Due to the unavailability, provocative pharmacologic testing could not be performed on our patient. Moreover, no coronary constriction was detected in the patient despite the presence of hyperventilation.

Combination of ST-segment elevation and regional wall motion abnormalities during DSE with

the relatively rapid resolution of the symptoms, as well as the presence of echocardiographic and ECG changes, raised the suspicion of coronary spasm. Therefore, provocative tests with ergonovine or acetylcholine were required (8). In such cases, the most effective approaches are infusion cessation and sublingual nitroglycerin administration. Furthermore, prescription of calcium channel blockers and modulation of risk factors should be considered for the long-term management of the patients.

Conclusion

According to the results of this study, DSE is associated with limited complications and adverse effects on the health of patients; therefore, it is considered a safe method for the diagnosis of coronary artery disease. One of the consequences of DSE is coronary artery spasm, the occurrence of which could be attributed to alpha-adrenergic stimulation. In such cases, dobutamine cessation and nitroglycerin administration could be effective.

Conflict of Interest

The authors declare no conflict of interest.

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