

Coronary Spasm during the Dobutamine Stress Echocardiography

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Introduction

Stress echocardiography is an imaging method whose main objective is the diagnostic and prognostic evaluation of patients with known or suspected coronary artery disease. It is a safe method with few complications, even when used in large scale¹.

The echocardiographic diagnosis of myocardial ischemia is done by viewing the reduced myocardial contractility (thickening and movement) during pharmacological stress. The test has a sensitivity of up to 88% and specificity of up to 83% for the detection of coronary obstructions larger than 50% on angiography².

The drug most widely used in pharmacological stress is dobutamine. Its sympathomimetic, inotropic and positive chronotropic action increases myocardial oxygen consumption and may trigger ischemia in patients with significant obstruction of coronary arteries. This agent is well tolerated and has a short half-life (2 to 3 minutes). Therefore, its effect can be stopped relatively quickly and safely. Betablockers such as metoprolol are often used at the end of the test to reverse the effects of dobutamine.

During dobutamine stress echocardiography, ischemic changes in left ventricle segmental contractility may be associated with ST segment depression. ST-segment elevation occurs most frequently in patients with atherosclerotic disease. ST-segment elevation in patients without coronary obstruction is a rare event usually attributed to coronary spasm.

We present the case of a patient without significant obstructive coronary artery disease who had ST-segment elevation while recovering from dobutamine stress echocardiography.

Case Report

Male, 55-year-old patient, brown, obese, referred for diagnostic investigation with dobutamine stress echocardiography due to clinical suspicion of coronary artery disease with history of hypertension and diabetes mellitus and ECG with diffuse ST and T abnormalities (Figure 1A).

Keywords

Betablocker, Vasospasm, ST-segment elevation.

Initial transthoracic echocardiogram showed left ventricle with preserved systolic function and mild diastolic dysfunction, mild to moderate left atrial increase, no abnormalities in left ventricular segmental contractility, mild to moderate mitral regurgitation and pulmonary systolic pressure estimated at 36 mmHg. The patient underwent a standard protocol with progressive infusion of dobutamine at doses of 5, 10, 20 and 30 mcg/kg/min with 3-minute increments. At the peak of the test, global left ventricular contractility improved, without any apparent segmental deficit. The test was stopped without reaching the submaximal heart rate for the age with 30 mcg/kg/min with BP of 150/100 mmHg and 112 bpm due to ventricular arrhythmia (frequent, isolated, coupled and clustered ventricular extrasystoles). After discontinuation of dobutamine and use of intravenous betablocker (2 mg metoprolol), the patient developed mild and diffuse left ventricular hypocontractility and transient 2 mm ST-segment elevation in DII, DIII, aVF, V3, V4, V5 and V6 leads without associated chest pain (Figure 1B).

The test was considered positive because of extensive electrocardiographic abnormalities in recovery; the patient was referred for the emergency room and underwent coronary angiography. The right coronary artery was dominant and presented focal stenosis of 30% in the proximal third; the left main coronary artery was normal; the anterior descending artery presented segmental stenosis of 30% in the middle third; the first diagonal branch was thin and of a small anatomic importance, with 90% ostial stenosis; and the circumflex artery presented parietal irregularities (Figure 2). Ventriculography showed global preserved systolic function without segmental impairment. A conservative medical treatment was chosen.

During hospitalization, there was a mild elevation of troponin I up to 1 ng/mL (normal value: smaller than 0.04), without an increase in CK-MB. Evolutionary electrocardiogram maintained the pre-test pattern with no Q waves. The patient was discharged early and referred for outpatient treatment.

Discussion

We describe a case of a patient who developed ST-segment elevation and presented minor abnormalities in the coronary arteries on angiography. The patient had no history of myocardial infarction and presented no significant coronary stenosis that justified the ST-segment elevation in multiple ECG areas (anterior and inferior). The electrocardiographic event disproportionate to coronary obstructions and abnormal left ventricular motility can be explained by a phenomenon of coronary spasm. This is a little frequent cause of false-positive results of dobutamine stress echocardiography.³

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Manuscript received August 09, 2013.; revised manuscript October 17, 2013;
accepted October 17, 2013.

Case Report



Figure 1A - Resting electrocardiogram.

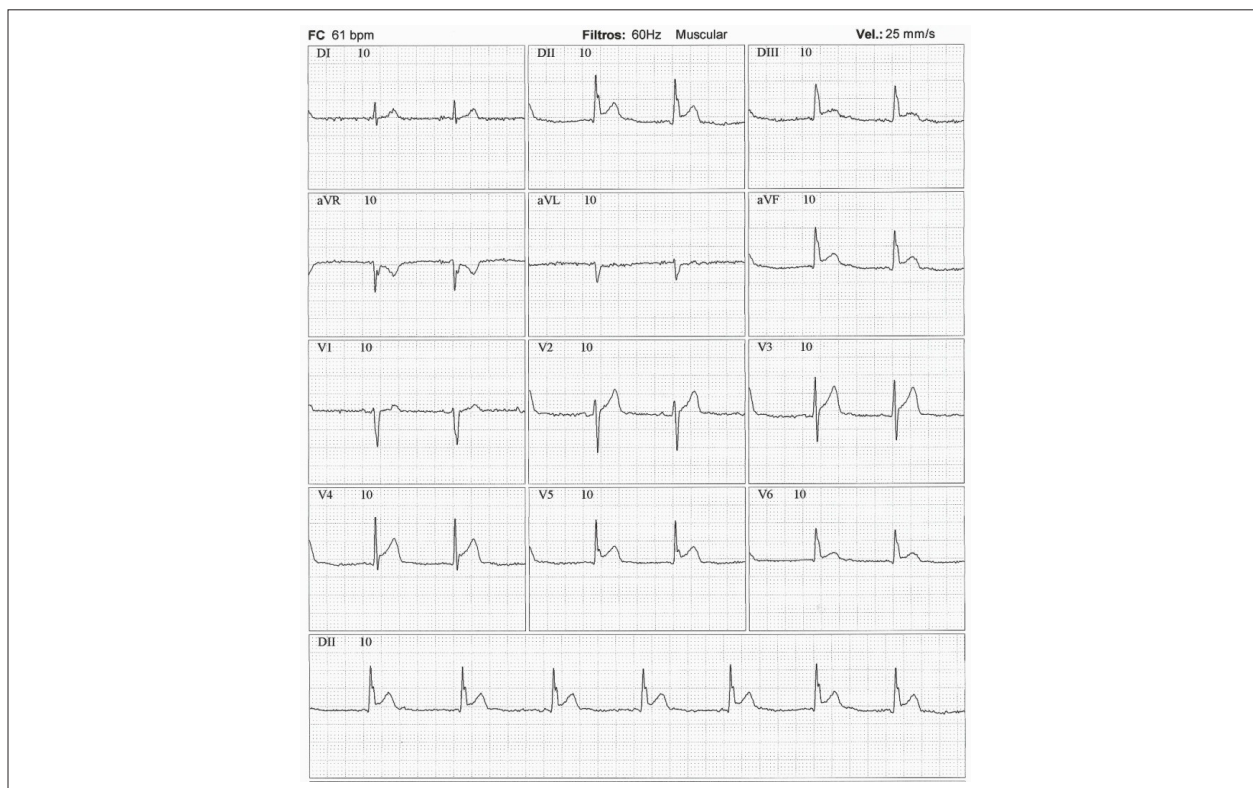


Figure 1B - Electrocardiogram with anterior and inferior ST-segment elevation.

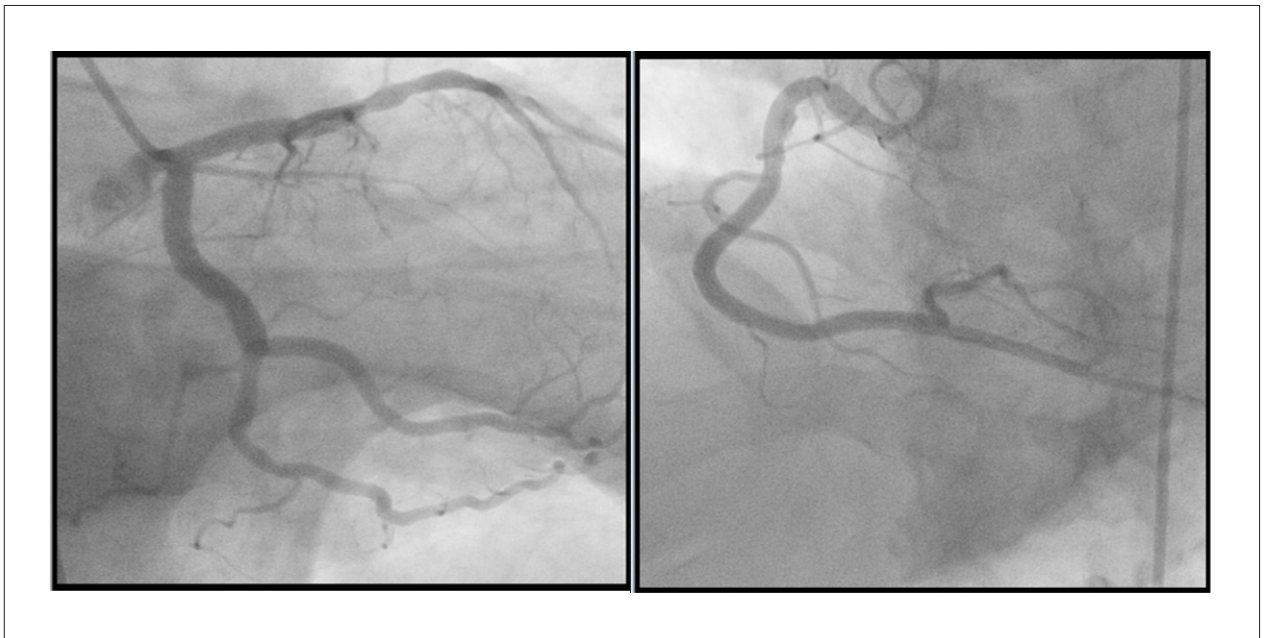


Figure 2 - Coronary angiography without significant obstructive lesions.

In patients with severe obstructive coronary artery disease, electrocardiographic and echocardiographic abnormalities during stress are due to reduced coronary reserve. These abnormalities appear progressively with increasing myocardial oxygen consumption during dobutamine infusion.

ST-segment elevation on the electrocardiogram during dobutamine stress echocardiography is uncommon (<3%) and mostly explained by dyskinesia of fibrotic area or transmural ischemia due to severe obstructive coronary artery disease. ST-segment elevation is very rare event in patients without significant obstructive coronary disease.⁴

There are studies showing that coronary spasm may appear with the same electrocardiographic abnormalities and these are rarely described during dobutamine echocardiography. In the study by Arruda et al., 4,240 patients underwent dobutamine stress echocardiography and only 134 (3%) had ST-segment elevation, of which only 1 was diagnosed as spasm (0.02%).⁴ Patients with coronary spasm present ST-segment elevation and worsened myocardial contractility during dobutamine infusion. Spasm triggered after addition of betablockers is still a controversial event.

The pharmacodynamic action of dobutamine in the coronary arteries is vasodilatation and increased coronary blood flow, which occurs by stimulation of β_2 -adrenergic receptors in the presence of normal-functioning vascular endothelium. This beta stimulation strongly predominates over the alpha stimulation, which in turn may cause vasoconstriction. Abnormal vasoconstrictor response in small and medium arteries may occur in the presence of atherosclerosis, and may be related to endothelial dysfunction.

Gordon et al. demonstrated that coronary artery segments with parietal irregularities respond to exercise and acetylcholine with spasm, indicating local endothelial dysfunction.⁵

The literature reported cases of coronary spasm during dobutamine use during stress echocardiography⁶⁻⁹ identified by ST-segment elevation on electrocardiogram. Only one case of coronary spasm was documented by dobutamine infusion during angiography¹¹. This effect was attributed to the alpha-adrenergic action of dobutamine on a substrate of endothelial dysfunction leading to transient vasoconstriction.

Apparently, a similar mechanism occurs for patients with variant angina¹². Roffi et al. evaluated 51 patients with clinical diagnosis of variant angina without obstructive coronary artery disease who underwent dobutamine stress echocardiography. Only seven patients presented segmental abnormalities on echocardiogram associated with ST-segment elevation on electrocardiogram, showing low sensitivity in the diagnosis of the disease.¹⁰

Coronary spasm has been rarely described after the infusion of dobutamine¹³ and administration of betablocker¹⁴. In our case, the electrocardiographic and echocardiographic abnormalities appeared in the test recovery period, which until then was considered negative for ischemia. The administration of betablockers is likely to have caused coronary spasm and myocardial ischemia, as a consequence. Metoprolol is a cardioselective betablocker that may rarely cause coronary spasm when it blocks beta stimulation and the consequent predominance of alpha stimulation. This mechanism has been demonstrated with the use of propranolol in patients with variant angina.¹⁵

Obviously, coronary spasm most commonly occurs in only one coronary artery and, in this case, the ST-segment elevation on electrocardiogram occurred in more than one coronary territory (anterior and inferior), and multiple spasms in right and left coronaries (LAD) would be unlikely

Case Report

even with discrete atherosclerotic lesions. Therefore, in this particular case, ischemia cannot be ruled out even with no significant catheterization abnormalities or the possibility of coronary spasm produced by the same dobutamine or by the betablocker within the average lifetime of both drugs.

Closing Remarks

ST-segment elevation during stress echocardiography is a rare event. In the absence of previous infarction or severe transmural ischemia by coronary atherosclerosis, coronary spasm should be remembered as a possible cause. During the test, coronary spasm may be due to the use of dobutamine during the procedure or the addition of betablockers in the recovery period.

In the case presented, the patient had no abnormalities on electrocardiogram or echocardiogram at rest suggesting

prior myocardial infarction. At the stress peak, there were no evident clinical, electrocardiographic or echocardiographic abnormalities typical of ischemia. The patient presented ST-segment elevation in multiple leads in the recovery period only after infusion of intravenous betablocker. Similar pictures are rare and potentially serious events, and in the absence of previous infarction, it is usually associated with severe coronary obstruction by atherosclerosis. However, in this particular case, coronary angiography presented minimal lesions that alone do not justify the exuberance of abnormalities.

For these reasons, despite the lack of tangible objective evidence, a coronary spasm associated with minimal coronary atherosclerosis is likely to have occurred. Besides possible, the spasm may have been caused by dobutamine or even by betablocker, as shown by studies and case reports described in the literature.

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