VIA MEDICA

CASE REPORT

Cardiology Journal
2012, Vol. 19, No. 1, pp. 79–80
10.5603/CJ.2012.0012

ISSN 1897–5593

Progressive bradycardia with increasing doses of dobutamine leading to stress echo interruption

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Abstract

Dobutamine stress testing is an important non-invasive method for evaluating patients with known or suspected coronary artery disease who are unable to adequately exercise. We present a case of a paradoxical, progressive bradycardia occurring with increasing doses of dobutamine that resulted in stress test interruption. (Cardiol J 2012; 19, 1: 79–80)

Key words: echocardiography, dobutamine, bradycardia

Pharmacological stress testing is an important non-invasive method for evaluating patients with known or suspected coronary artery disease who are unable to adequately exercise [1]. In combination with echocardiography, pharmacological stressors such as dobutamine and dipyridamole are used [1]. The safety of the stress test is a major determinant in the choice of the agent [1]. With dobutamine stress testing, side effects such as ventricular tachycardia, torsade de pointes, fatal ventricular fibrillation, cardiogenic shock, free wall rupture or transient ischemic attack have all been reported [2].

We present a case of an unusual chronotropic response to increasing doses of dobutamine that occurred on stress echocardiography, i.e. progressive bradycardia leading to test interruption.

A 44 year-old man was referred to our department due to suspected in-stent restenosis six months after acute myocardial infarction that was treated with stent-assisted primary angioplasty of the left anterior descending artery. The patient was sent to our echo lab for the assessment of left ventricular contractility on dobutamine stress echo. The baseline echocardiogram revealed apical akinesis and hypokinesis of the anterior wall, with an overall ejection fraction of 50%. The baseline heart rate (HR) was 63 bpm, blood pressure (BP) was 115/70 mm Hg. The beta-adrenolytic agent (bisoprolol 5 mg) was stopped on the day before the test. Dobutamine was infused at a dose commencing at 5 μg/kg/min and increasing incrementally by 5 μg/kg/min every three minutes. The values of HR and BP during the test are shown in Table 1.

During the test, in spite of a normal BP response and a lack of chest pain, the patient complained of progressive weakness, which made us interrupt the test at 13 min. Electrocardiography (ECG) revealed a progressive sinus bradycardia (Table 1) while no atrioventricular conduction block was observed. No new wall motion abnormalities

Table 1. Parameters during dobutamine stress.

Time	Dosage [μg/kg/min]	Heart rate [bpm]	Blood pressure [mm Hg]
Baseline	–	63	115/70
3 min	5	63	115/70
6 min	10	57	140/75
9 min	15	47	160/80
12 min	20	45	170/80

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Received: 08.09.2010 Accepted: 29.09.2010

were observed. Several minutes after the test interruption, the symptoms of weakness resolved, and HR and BP returned to baseline. Coronary angiography was performed on the following day. It revealed a widely patent stent and no progression of atherosclerosis.

Isolated, progressive sinus rhythm deceleration during dobutamine stress echocardiography has not been thus far reported in humans. However, dobutamine-induced bradycardia has been reported in dogs and horses [3, 4] although its mechanism was unclear. In humans, a paradoxical sudden sinus deceleration has been reported only with peak dobutamine doses (i.e., a sudden rather than a progressive effect) and it has always been accompanied by arterial hypotension. In a series of 58 patients subjected to dobutamine perfusion scintigraphy, Hopfenspringer et al. [5] reported such an effect in 17% of cases; this was seen usually in patients with an inferior wall perfusion defect. The exact mechanism responsible for a sudden bradycardia during dobutamine stress echo is unclear. A sudden sinus rhythm deceleration may be the result of Bezold-Jarisch reflex, an intracardiac parasympathetic nervous reflex responsible for bradycardia, hypotension and vasodilatation. The Bezold-Jarisch reflex may be stimulated by vigorous myocardial contractions occurring when ventricular filling is reduced, as may occur during dobutamine infusion [6].

In contrast to prior reports, the sinus bradycardia in our patient was progressive and it occurred in spite of a normal BP response (Table 1). It occurred 6 min after the beginning of dobutamine infusion (dobutanime dose 10 μg/kg/min). In contrast to previous reports, the bradycardia was accompanied by an increase in BP (max. 170/80 mm Hg). Such an effect is unlikely to be caused by the Bezold-Jarisch reflex. Similarly, it is unlikely to be caused by the baroreflex response, as the BP increase was mild (Table 1).

In this patient, the right and circumflex coronary arteries were free of atherosclerosis and the stent in the left anterior descending artery was widely patent.

Six months after the test, the patient was free of symptoms, and physical examination and 24 hour ECG monitoring were normal.

We report that a paradoxical, progressive bradycardia can occur with increasing doses of dobutamine and it may bring about stress test interruption. Such a paradoxical effect, accompanied by a normal BP response, has not been reported previously. Physicians performing stress echo should be aware of such a side effect.

Conflict of interest: none declared

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