Case Report

Takotsubo cardiomyopathy occurring during the recovery period of dobutamine stress echocardiography

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Summary  An 83-year-old Caucasian female was referred for dobutamine stress echocardiography. At peak dobutamine stress, no chest pain was elicited, and there were no ischemic electrocardiographic changes or inducible wall motion abnormalities. During recovery, however, the patient experienced chest burning, and ST-elevations were seen on recovery electrocardiography. Moreover, distal inferior and apical akinesis developed during recovery echocardiography. The patient was referred for coronary angiography, revealing apical akinesis and no obstructive stenoses. A follow-up echocardiogram 2 weeks later showed recovery of the “ballooned” segments. To our knowledge, this report is the third case of Takotsubo cardiomyopathy occurring during the recovery phase of dobutamine stress echocardiography.

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Introduction

Dobutamine stress echocardiography is a procedure that is associated with a low risk of complications. Serious complications do, however, occur, with the majority taking place during the stress portion of the procedure. Herein, we report a case of Takotsubo cardiomyopathy occurring during the recovery portion of dobutamine stress echocardiography. To our knowledge, this account is the third case of this complication occurring after cessation of the dobutamine infusion.

Case report

An 83-year-old Caucasian female presented to our clinic with a variety of non-specific somatic complaints. She had noted transient lightheadedness, abdominal discomfort, and some lower extremity swelling. Her cardiovascular risk factors included hypertension, hyperlipidemia, a remote tobacco history, and a family history of heart disease, with her mother dying of a myocardial infarction at the age of 66.
She had undergone a previous evaluation for these non-specific atypical symptoms. Two years previously, a regadenoson myocardial perfusion study had revealed a small, mildly reversible inferoapical defect. One year prior, a coronary computed tomography angiogram had revealed moderate (50–60% in severity) disease of the proximal 1st obtuse marginal. Finally, rest echocardiography had revealed mild anterior mitral valve prolapse with mild mitral regurgitation.

In light of the low suspicion that these symptoms were a manifestation of obstructive heart disease, she was referred for dobutamine stress echocardiography. On presentation, she was afebrile and mildly hypertensive (157/86 mm Hg). Her physical examination revealed a Grade 1 holosystolic murmur at the apex with some radiation posteriorly. The rest of her physical exam was normal. Resting electrocardiography (ECG) demonstrated a normal sinus rhythm with a rate of 77 bpm, a normal axis, and no abnormal ST–T changes.

Baseline resting echocardiography demonstrated a normal ejection fraction with no segmental wall motion abnormalities. With a gradual increase in the dobutamine infusion to a peak rate of 40 μg/kg/min and adjunctive atropine 0.5 mg IV, an adequate peak stress heart rate of 138 bpm was attained. No chest pain was elicited, and the peak stress ECG showed ventricular bigeminy and no ischemic ST changes (Fig. 1). Moreover, peak stress echocardiography revealed augmented contractility and thickening of all segments (Fig. 2 A and B, Movie Clips 1 and 2).

During the recovery portion of the stress test, however, the patient started to experience chest burning. The accompanying ECG showed dramatic ST elevations in leads II, III, aVF, V3–5, with reciprocal changes in leads avL, V1, and V2 (Fig. 3). The associated recovery echocardiographic images revealed the interval onset of inferoapical and apical akinesia (Fig. 4A and B, Movie Clips 3 and 4).

She was given aspirin, nitrates, and metoprolol with resolution of the chest burning. She was also emergently transferred for invasive coronary angiography (Fig. 5). No significantly obstructive stenoses were, however, detected.
Figure 3  Recovery electrocardiography revealed dramatic ST elevations in leads II, III, avF, V3–5, with reciprocal changes in leads avL, V1, and V2.

Accompanying left ventriculography showed apical akinesis with preserved contractility of the basal and mid segments (Fig. 6, Movie Clip 5). Serial laboratory analysis revealed a peak creatine kinase (CK) 115 U/L (reference 20–168), a peak CK-MB 9.10 ng/ml (reference ≤ 6.60), and a peak troponin I 1.559 ng/ml (reference ≤ 0.036), all findings suggestive of minimal cardiac myonecrosis. A follow-up echocardiogram two weeks later revealed normalization of the distal inferoapical and apical akinesis.

Figure 4  (A) Recovery end-systolic stress echocardiographic imaging of the apical 4-chamber view revealed apical akinesis. (B) Recovery end-systolic stress echocardiographic imaging of the apical 2-chamber view revealed distal inferior and apical akinesis.

Figure 5  Coronary angiography of the left coronary artery revealed no significantly obstructive disease.
Discussion

Takotsubo cardiomyopathy, also known as the apical ballooning syndrome, stress cardiomyopathy, and the "broken heart" syndrome, is a form of transient left ventricular dysfunction first described in 1991 in Japan [1]. This syndrome is characterized by transient regional systolic dysfunction, most commonly affecting the apex with hyperkinesis of the basal segments. Other less common variants of this syndrome include "ballooning" of the midventricular [2] or basal segments [3].

As was seen in this case report, this syndrome frequently affects post-menopausal women. Upon presentation, symptoms are variable but generally include chest pain or dyspnea. The ECG manifestations often mimic those of an acute myocardial infarction, with marked ST elevations in the distribution of one of the coronary arteries. Kosuge et al. [4] recently described various electrocardiographic criteria that could distinguish Takotsubo cardiomyopathy from an acute anterior myocardial infarction. Specifically, the absence of abnormal Q-waves, the absence of reciprocal changes, the presence of ST-depression in lead aVR, and the absence of ST-elevation in lead V1 identified Takotsubo cardiomyopathy with modest accuracy. The combination of ST-depression in lead aVR and the absence of ST-elevation in lead V1 were particularly accurate, with a reported 95% predictive accuracy. In the current case report, these criteria would have properly identified this case as Takotsubo cardiomyopathy. The ECG in this case appropriately exhibited ST-depressions in lead aVR, no significant Q-waves, and no ST-elevation in lead V1. The ECG in this case did, however, show reciprocal changes.

Frequently mistaken as an acute myocardial infarction, these cases are commonly referred for urgent coronary angiography, with these procedures generally unrevealing for any significantly obstructive stenoses. The prognosis of affected patients is generally good, with recovery of the "ballooned" segments occurring within weeks with medical support. Complications are rare but can include congestive heart failure, thromboembolism, dynamic left ventricular outflow tract obstruction, mitral regurgitation, and arrhythmias [5].

The etiology of Takotsubo cardiomyopathy is unknown. Initially, multivessel coronary artery spasm was hypothesized as a cause of this entity. However, as with this current report, coronary artery spasm is not generally visualized, although ST-elevations are present during coronary angiography. In addition, thrombosis superimposed upon an eroded or "vulnerable" plaque has not been described.

The most promising hypothesis regarding the etiology of Takotsubo cardiomyopathy involves the phenomenon of catecholamine-mediated stunning. This mechanism was suggested by its frequent association with and provocation by extreme emotional stress. In line with this hypothesis, 123I metaiodobenzylguanidine imaging has revealed impaired sympathetic innervation in the "ballooned" apices of affected individuals [6]. Finally, plasma catecholamine levels in those individuals are markedly elevated, even when compared with levels in patients with Killip class III myocardial infarctions [7].

Also in line with this hypothesis of catecholamine-mediated stunning, there have been case reports of Takotsubo cardiomyopathy occurring with dobutamine stress echocardiography. Previtali et al. first reported in 2005 a case of dobutamine-induced apical ballooning complicated by mid-ventricular obstruction and mitral regurgitation [8]. Since this first report, there have been 6 case reports [9–14] and 1 small case series [15] of 3 of this syndrome. In most of these cases, the "ballooning" phenomenon appeared to develop during the dobutamine infusion of the procedure. In the present case, however, the apical ballooning took place during the recovery period. To our knowledge, the present case is the third report of this "ballooning" phenomenon occurring during dobutamine recovery [9,14]. In light of this potential complication, close monitoring of patients undergoing dobutamine stress echocardiography is recommended, even during the recovery period.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.jccase.2012.03.003.

References

Takotsubo cardiomyopathy after dobutamine stress echocardiography


