Takotsubo cardiomyopathy (left ventricular ballooning syndrome) induced during dobutamine stress echocardiography

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A 75-year-old woman presented with left ventricular apical ballooning syndrome mimicking acute anterior myocardial infarction. She had a long history of chest tightness and was undergoing a contrast stress echocardiogram because of frequent re-admissions with chest pain, all with normal ECGs and troponins.

A standard dobutamine/atropine protocol had been used. The patient developed central crushing chest pain, marked ST elevation, transient slowing of heart rate and left ventricular apical ballooning. She was admitted to the coronary care unit and received thrombolysis. Echocardiography showed no mitral regurgitation, normal pulmonary artery systolic pressure and no intraventricular gradient. Immediate troponin T was significantly raised and peaked at 6 h. The patient underwent coronary angiography the next day. This showed systolic left ventricular apical ballooning and no coronary lesions or vasospasm. A further contrast echo 2 weeks later showed complete resolution of the wall motion abnormalities.

Left ventricular apical ballooning or takotsubo syndrome can lead to cardiogenic shock but has a favourable prognosis compared to myocardial infarction. This patient was noted to be unusually anxious about having a stress echocardiogram and mental stress might be a precipitating factor, furthermore, this response during dobutamine stress echocardiography favours a catecholamine related disease mechanism.

KEYWORDS
Stress echo; Takotsubo; Cardiomyopathy; Catecholamines; Myocardial infarction

Introduction

Takotsubo cardiomyopathy is increasingly recognised as a syndrome, usually provoked by severe mental stress and associated with acute 'ballooning' or dyskinesis of the left ventricle.1 Although the aetiology of takotsubo syndrome remains obscure catecholamine release appears to be the principal trigger. This report describes a case where the combination of severe anxiety and exogenous stressing catecholamines resulted in acute takotsubo syndrome mimicking acute myocardial infarction.

Case

A 75-year-old woman attended for dobutamine stress echocardiography (DSE). She had a long history of atypical precordial chest pain and had undergone coronary angiography three years before which showed only minor plaque disease. Echocardiography had not been undertaken during previous admissions. DSE was performed because of frequent re-admissions with chest pain, all with normal ECGs and without troponin elevation. A standard dobutamine/atropine protocol was used with 10 mcg/kg/min dose increments at 3 min intervals. Her resting echocardiogram and blood pressure were normal. At 30 mcg/kg/min dobutamine and following 0.5 mg atropine (given for failure of initial rate response) the patient developed central crushing chest pain, marked ST elevation and transient slowing of heart rate.

Left ventricular opacification (LVO) with contrast provides better visualisation of the endocardial border, which translates into improved detection of wall motion abnormalities during stress. In this department, LVO is part of our protocol for stress echocardiography unless it is contraindicated.

The contrast enhanced four-chamber echocardiogram at this point showed marked systolic dyskinesis of the cardiac apex and surrounding region. (Figures 1 and 2). ECG showed 2 mm ST elevation in V3/4 (Figure 3). She was...
immediately admitted to the coronary care unit with suspected dobutamine induced myocardial infarction. The patient received thrombolysis after showing no response to intravenous nitrates. Echocardiography repeated immediately after thrombolysis showed no mitral regurgitation, normal pulmonary artery systolic pressure (20 mmHg) and no intraventricular cavity gradient. Troponin T was significantly raised and peaked at 6 h (1.03 ng/ml). The patient underwent coronary angiography on the following morning. This showed systolic left ventricular apical ballooning (Figure 4) but no significant coronary lesions, vasospasm or muscle bridging. A further contrast enhanced echocardiogram 2 weeks later showed complete resolution of the wall motion abnormalities (Figures 5 and 6). She remains well with no long term adverse sequelae.

Discussion

Left ventricular apical ballooning or syndrome is an increasingly recognised entity that closely mimics myocardial infarction. Current estimates suggest that around 1% of patients presenting with acute myocardial infarction may in fact be suffering from this syndrome. Although the outcome is usually benign cardiogenic shock may occur. Various aetiological factors have been proposed including microvascular dysfunction, mitral regurgitation, dynamic left ventricular outflow tract obstruction and coronary muscle bridging. The clearest finding is that it has a predilection for postmenopausal women and a tendency to occur at times of extreme emotional stress suggesting a catecholamine based aetiology. This case conforms to the typical patient profile and she was noted to be unusually anxious about having a stress echocardiogram; furthermore the clear relationship to high dose dobutamine which has also been noted elsewhere supports a catecholamine related disease mechanism. It is possible that the aetiology of the wall motion abnormalities and ECG changes could have been due to coronary spasm or acute plaque rupture with thrombosis. The findings of persistent wall motion abnormalities after 24 h in the face of normal arteries and
the observation that the subject’s echocardiogram and left ventricular function returned to normal within 7 days of the initial presentation, suggest that the diagnosis was in fact takotsubo syndrome.

While complications during DSE are rare myocardial infarction may be induced. This case illustrates how the use of catecholamine stressors in an already anxious patient induced this increasingly recognised cardiomyopathy. It is likely that other cases of DSE induced myocardial infarction are in fact due to takotsubo syndrome.

References