2015

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CASE REPORT

Stress-induced cardiomyopathy

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Accepted 13 March 2015

SUMMARY

A woman in her early 70s presented with chest pain, dyspnoea and diaphoresis 30 min after her husband expired in our hospital. Cardiac markers were elevated and there were acute changes in ECG suggestive for acute coronary syndrome. Echocardiogram showed apical akinesis, basal segment hyperkinesis with an ejection fraction of 30%. Cardiac catheterisation was performed showing non-obstructive coronary arteries, leading to the diagnosis of stress-induced cardiomyopathy. The patient improved with medical management. Repeat echocardiogram 2 months later showed resolution of heart failure with an ejection fraction of 65–70%.

BACKGROUND

Takotsubo cardiomyopathy, also known as stress-induced cardiomyopathy or apical ballooning syndrome, is a complex condition that has been increasingly reported over the past few years. It is characterised as a reversible cardiomyopathy that is often precipitated by a stressful or emotional event.[1 2] The presentation can be indistinguishable from an acute myocardial infarction and the absence of angiographically significant coronary artery disease must be confirmed prior to making this diagnosis.[2] Presentation can include chest pain, ST segment elevations, rise in cardiac markers, heart failure, and characteristic apical and mid-segment hypokinesis with compensatory basal segment hyperkinesis, producing an apical ballooning, which gives the appearance of a Japanese octopus trap, 'takotsubo' (figure 1).[3]

CASE PRESENTATION

A woman in her early 70s presented to our emergency room 30 min after her husband expired with 8/10, substernal chest pain with concurrent diaphoresis and dyspnoea. The patient's husband had an out-of-hospital cardiac arrest earlier that day and was brought to our emergency room and subsequently admitted to the coronary care unit. He had recurrent ventricular fibrillation and could not be successfully resuscitated.

The patient's medical history included hypertension and hypothyroidism. She denied having had a similar episode of pain. She had regular appointments with her primary care physician. Her medication included oral furosemide 20 mg and levothyroxine 100 µg, both once daily. She denied allergies.

On presentation, the patient was afebrile, with a blood pressure of 105/55, heart rate of 93 bpm, respiratory rate of 20/min and an oxygen saturation of 98% on room air. On physical examination, she was in significant distress due to pain. Her skin was warm and moist, with minimal crackles in bilateral bases, regular rate and rhythm, 2/6 systolic murmur of the apex without radiation, no rubs, elevated jugular vein distention of 8 cm, strong peripheral pulses and no peripheral oedema. The abdomen was soft, non-tender, non-distended, with bowel sounds, and the patient was alert and oriented without focal neurological deficits.

Figure 1 Octopus trap; in Japanese: 'Takotsubo'.

INVESTIGATIONS

The complete blood count, basic metabolic panel and coagulation tests were within normal limits. The cardiac enzymes were increased with a troponin I of 2.59 ng/mL (normal range 0.015–0.045 ng/mL), creatine phosphokinase of 84 U/L (normal range 26–192 U/L). ECG on admission revealed normal sinus rhythm with 2–3 mm ST segment elevations in leads V2–V3 and Q waves in leads V1–V3, suggestive of anterior wall infarct (figure 2).

An echocardiogram revealed left ventricular ejection fraction of 30–35% with mild concentric left ventricular hypertrophy, akinesis of the apex and 2/3 of the inferoseptum and apical inferior-anterior wall segments, suggesting ischaemic damage at the left anterior descending artery distribution versus takotsubo cardiomyopathy (figures 3 and 4).

The patient received aspirin 162 mg and prasugrel 60 mg, and was taken to the cardiac catheterisation laboratory. The angiogram showed non-obstructive coronary artery disease with luminal irregularities (figures 5 and 6). The left ventriculography showed an ejection fraction of 35% with akinesis of apical wall and hyperkinesis of basal segments (figures 7 and 8).

To cite: Lisung FG, Shah AB, Levitt HL, *et al*. *BMJ Case Rep* Published online: [*please include* Day Month Year] doi:10.1136/bcr-2014-208690

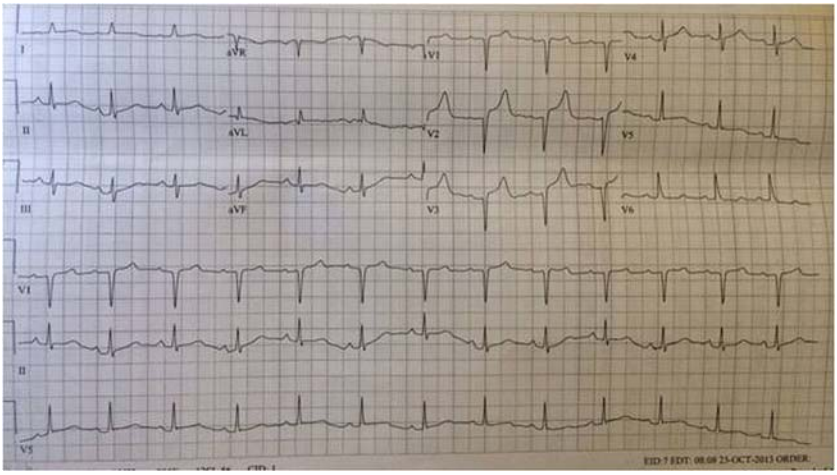

Figure 2 ECG showing ST segment elevation in anterior leads (V2–V3).

OUTCOME AND FOLLOW-UP

The patient remained haemodynamically stable and was started on oral heart failure medications, including lisinopril 2.5 mg daily and metoprolol tartrate 12.5 mg two times a day.

Her pain and shortness of breath resolved and she was discharged home after 48 h of hospitalisation in stable condition. A repeat echocardiogram 2 months later showed resolution of her cardiomyopathy and an ejection fraction of 65–70%, with normal left ventricular motion.

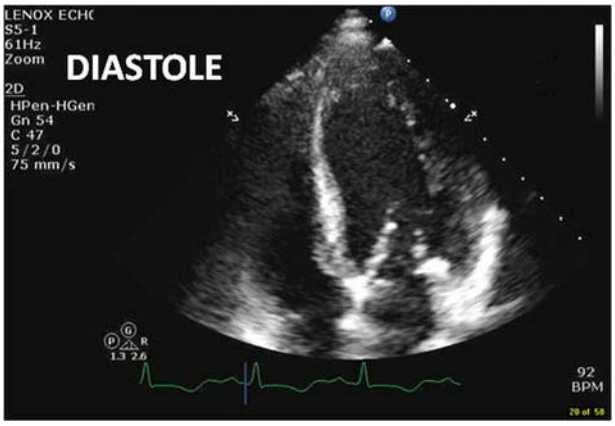

Figure 3 Echocardiogram showing the left ventricle during diastole with normal relaxation.

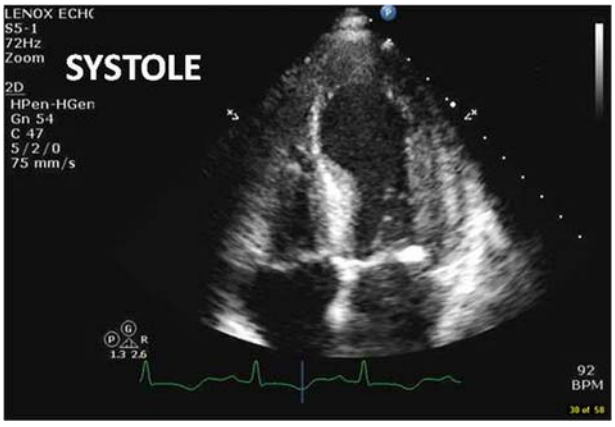

Figure 4 Echocardiogram showing the left ventricle during systole with akinesis of the apex and hyperactive basal segments, giving the appearance of an 'octopus trap'.

DISCUSSION

We describe a case of typical takotsubo cardiomyopathy. This syndrome is a unique, transient and reversible cardiomyopathy that mimics acute coronary syndrome. It should be included in the differential diagnosis of all patients with clinical presentation of acute coronary syndrome, including elevated cardiac enzymes and ST segment elevation, particularly when there is apical wall motion abnormality with compensatory basal segment hyperkinesis and the absence of angiographic signs of obstructive coronary artery stenosis.[2 4] The pathogenesis of takotsubo cardiomyopathy is not well understood, however, many hypothetic mechanisms have been proposed, such as coronary vasospasm, microvascular spasm and neurogenic stunned myocardium due to catecholamine excess.[2 5] One of the hallmarks of stress-induced cardiomyopathy is its preponderant occurrence in postmenopausal females who have been exposed to acute emotional or stressful situations.[1 2 5 6] Despite the severity of the acute onset, takotsubo cardiomyopathy,

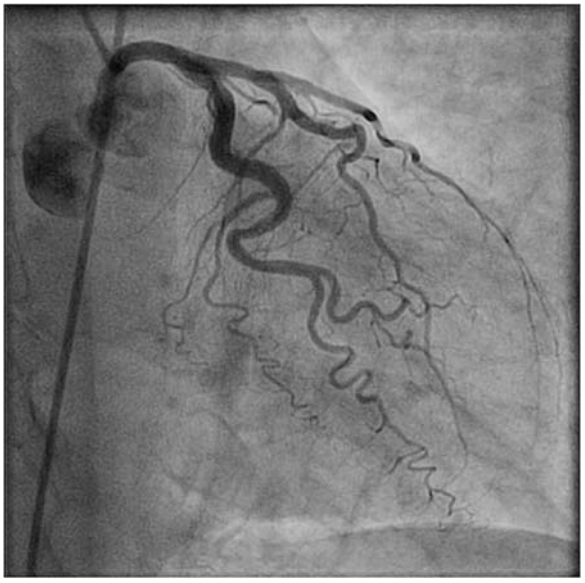

Figure 5 Right anterior oblique caudal view during cardiac catheterisation showing grossly luminal coronaries with less than 20% stenosis.

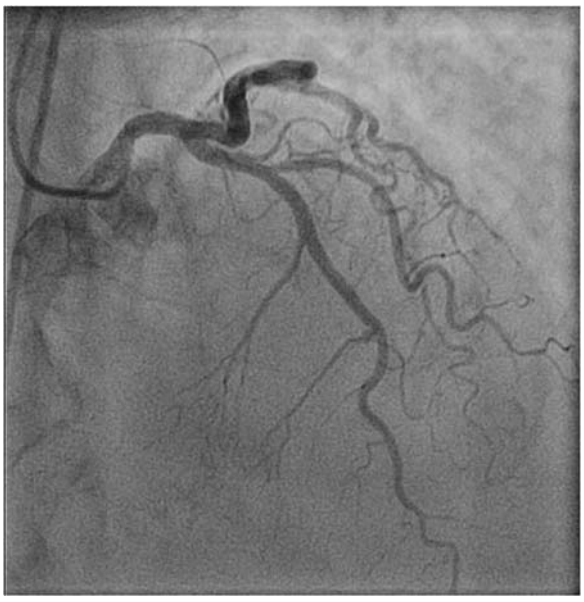

Figure 6 Right anterior oblique cranial view during cardiac catheterisation showing grossly luminal coronaries with less than 20% stenosis.

also known as broken heart syndrome, is generally a transient disorder and normalisation of left ventricular ejection fraction usually occurs within 1–4 weeks with minimal recurrence seen.[7] Management is supportive and must be tailored to each patient's haemodynamics and volume status (hypotension, shock, left ventricular outflow obstruction, heart failure, etc). If haemodynamically stable and left ventricular outflow obstruction is not seen, the 2014 AHA/ACC guidelines suggest the use of conventional treatments, including ACE inhibitors, β-blockers and diuretics, as otherwise indicated (class I, level of evidence C).[7–9] These guidelines are based on the treatment of patients with heart failure with reduced ejection fraction. Evidence-based guidelines specifically for treatment of takotsubo cardiomyopathy do not exist. In addition, for haemodynamically stable patients, the current guidelines

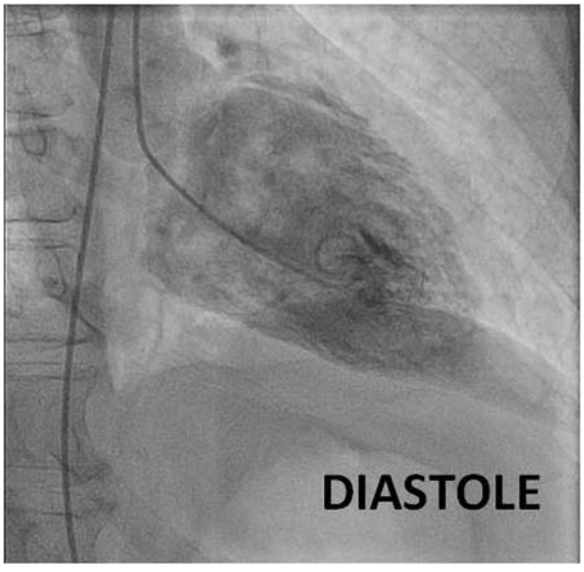

Figure 7 Left ventriculography during diastole showing normal relaxation of left ventricle.

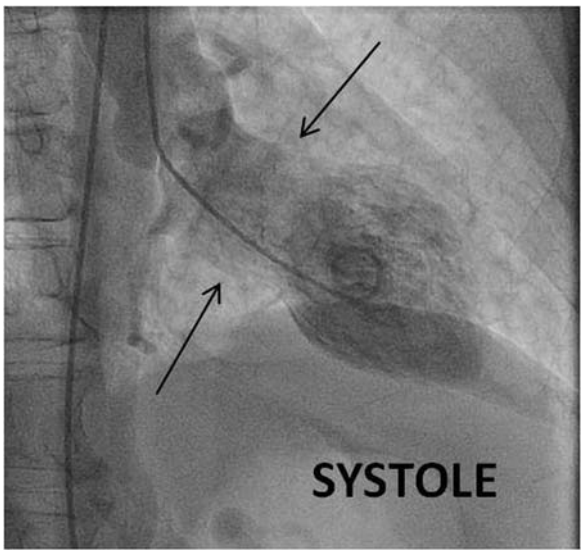

Figure 8 Left ventriculography during systole showing apical akinesis and contraction of basal segments. Arrows point to basal hyperkinesis.

do not specify whether conventional heart failure therapy should be initiated in the acute phase or if a short trial period should be given to see if the ejection normalises without therapy.

Learning points

- Stress-induced cardiomyopathy is an acute, transient, reversible and non-ischaemic cardiomyopathy.
- Consider stress-induced cardiomyopathy in the differential diagnosis of acute coronary syndrome.
- Treatment is tailored to each patient's haemodynamics and volume status.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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