CASE REPORT

Rare case of stress cardiomyopathy due to intramuscular epinephrine administration

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SUMMARY

We report a case of a 37-year-old woman who presented to our hospital with retrosternal chest pain following intramuscular administration of epinephrine due to presumed anaphylaxis. On arrival, she was found to have ST segment depression in the anterolateral leads on ECG and elevated cardiac troponins. She was diagnosed with stress cardiomyopathy based on left ventricle dysfunction and angiographically normal coronary arteries on cardiac catheterisation. To the best of our knowledge, this is the third reported case of takotsubo cardiomyopathy following appropriately dosed intramuscular administration of epinephrine for anaphylaxis. This case highlights the importance of considering stress cardiomyopathy in patients presenting with chest pain syndrome following systemic administration of epinephrine.

BACKGROUND

Stress cardiomyopathy, also known as takotsubo cardiomyopathy or broken heart syndrome, is a disorder of reversible systolic dysfunction of the left ventricle (LV). It is usually preceded by intense physical or emotional triggers and is most common in older postmenopausal women.¹ It presents as chest pain syndrome resembling acute coronary syndrome and is distinguished by the absence of angiographic evidence of obstructive coronary artery disease.² Catecholamines have been implicated to have a role in its pathogenesis.³ Cases of stress cardiomyopathy due to intramuscular use of epinephrine for anaphylaxis have rarely been reported in the literature. Hence it is crucial to recognise its presentation in this patient population, as long-term therapy is not needed in this condition in contrast to acute coronary syndrome.

CASE PRESENTATION

A 37-year-old woman with medical history of multiple environmental allergies presented to our hospital with chest pain that started 15 min prior to presentation. The patient was eating shrimp at a local restaurant, when she started to have mouth and tongue swelling with localised facial urticaria without any associated respiratory symptoms. She immediately went to a local urgent care centre where she was given epinephrine 0.3 mg intramuscularly. Following that, she developed new onset retrosternal chest pain and was brought to our emergency department. On arrival, she reported of chest pain associated with shortness of breath, nausea and dizziness. She reported that the mouth swelling and localised facial rash had already

improved significantly. On initial vitals, she was found to have a blood pressure of 180/112 mm Hg, heart rate of 108 bpm, respiratory rate of 18 breaths/min and oxygen saturation of 97% on room air. On physical examination, she appeared to be in distress, while the rest of the examination including a cardiovascular examination was within normal limits.

INVESTIGATIONS

The initial ECG showed new ST segment depression in anterolateral leads and a prolonged corrected QT interval of 469 ms. Laboratory testing identified troponin I of 1.2 ng/mL (reference range <0.06 ng/mL) at 2 hours, which peaked to 28.21 ng/mL 9 hours after presentation. Routine haematological and biochemical investigations were within normal limits, including a thyroid function test. Transthoracic echocardiogram (TTE) showed basal and midventricular akinesis with a hypercontractile apex, an ejection fraction of 40% and no valvular abnormalities (videos 1 and 2).

DIFFERENTIAL DIAGNOSIS

Stress-related cardiomyopathy, coronary artery spasm and acute coronary syndrome were considered based on the overall clinical picture. Left heart catheterisation with coronary angiography was performed. Left ventriculography identified akinetic basal and midventricular segments with a hypercontractile apex, an ejection fraction of 35–40% and elevated left ventricular end-diastolic pressure of 15 mm Hg (figure 1). The coronary angiography revealed normal coronary arteries. A diagnosis of stress-related cardiomyopathy was made based on the marked LV dysfunction and normal coronary arteries on cardiac catheterisation.

TREATMENT

The patient was started on aspirin, ACE inhibitor and β -blocker therapy. After the cardiac catheterisation revealed normal coronaries, aspirin was discontinued while the remaining medications were continued.

OUTCOME AND FOLLOW-UP

A repeat TTE was performed at 12 days after discharge, which showed a normal ejection fraction of 63% with no associated wall motion abnormalities. The patient had neither signs nor symptoms concerning for pheochromocytoma, and plasma fractionated metanephrines assessed after discharge were normal. Based on the clinical presentation, reversible ventricular dysfunction and normal

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Video 1 Transthoracic echocardiogram: apical two-chamber view.

Video 2 Transthoracic echocardiogram: apical four-chamber view.

coronary arteries, a diagnosis of stress cardiomyopathy likely due to intramuscular epinephrine use was made.

DISCUSSION

Takotsubo (stress) cardiomyopathy was first described in Japan in 1991, when authors found that the shape of an affected LV is

similar to a takotsubo, a Japanese pot for fishing octopus.⁴ It mimics acute coronary syndrome and is characterised by sudden onset of acute chest symptoms, ECG changes consistent with myocardial ischaemia, elevated cardiac troponins, temporary LV dysfunction and normal coronary arteries on coronary angiography.⁵ Coronary artery vasospasm due to epinephrine was another important rare differential that was considered in our case. However, in these cases, patients usually have a normal LV function as opposed to in our case.^{6 7}

Physical triggers are more common than emotional triggers and sometimes an attack can occur without any evident preceding trigger.² Not every patient with stress cardiomyopathy presents with ST segment elevation on ECG or apical left ventricular involvement. In the international takotsubo registry,² it was found that 8.3% of patients had ST segment depression, 47% had prolonged corrected QT interval, 2.2% had basal LV involvement and 93% had an increased left ventricular end-diastolic pressure (>11 mm Hg). All these features were present in our reported case. It is also suggested that drugs used to prolong the QT interval should be avoided in the acute phase of stress cardiomyopathy.⁸

A stress-induced surge in catecholamines leading to myocardial stunning due to microvascular dysfunction has been proposed in the pathogenesis of stress cardiomyopathy.^{9 10} Takotsubo cardiomyopathy has been associated with elevated plasma catecholamines and, specifically, elevated epinephrine levels.¹¹ Cases of stress cardiomyopathy due to exogenous epinephrine administration through intramuscular, subcutaneous, submucosal and intravenous routes have been reported in the literature.¹² Zubrinich *et al*¹³ reported a case of a 76-year-old woman who developed stress cardiomyopathy following intramuscular administration of 0.3 mg epinephrine for anaphylaxis. However, the index patient was on oxprenolol 20 mg daily, a drug with intrinsic sympathomimetic activity that, with its partial agonist effects, may have perpetuated the catecholamine effects. Magri *et al*¹⁴ also reported the case of a healthy 26-year-old woman who developed stress cardiomyopathy following intramuscular administration of 0.5 mg epinephrine for anaphylaxis. Both these patients had uneventful recoveries.

LV dysfunction usually recovers within 7–37 days with conservative management including ACE inhibitor, β -blocker and diuretics, if associated with pulmonary oedema.^{15 16} The use of ACE inhibitors or angiotensin receptor blockers is associated with survival benefit at 1 year, whereas no survival benefit was found for β -blockers at 1 year, in the international takotsubo

Figure 1 Left ventriculogram: (A) diastole and (B) systole show basal and midventricular akinesis with apical hyperkinesis.

registry.² The prognosis is generally favourable, with complete recovery in most cases and rare recurrence rate.¹⁵

Systemic administration of epinephrine is considered lifesaving treatment for anaphylaxis. Patients and healthcare personnel should be educated about the indications and proper use of epinephrine for anaphylaxis. It is possible that the physical and psychological stress associated with the underlying allergic reaction were potential contributors in the development of stress cardiomyopathy in our patient. However, the resolution of swelling and facial hives on initial presentation, the continuously rising troponin that peaked at 9 hours after epinephrine administration and the proposed role of catecholamines in the pathogenesis, suggest that epinephrine use was the most likely cause of stress cardiomyopathy in our patient.

Learning points

- ▶ Takotsubo cardiomyopathy should be considered in patients presenting with chest pain after intramuscular epinephrine administration.
- ▶ It mimics acute coronary syndrome and diagnosis requires coronary angiography to exclude coronary artery disease.
- ▶ It most commonly presents with ST segment elevation on ECG but can also present with ST segment depression in 7.6% of cases.
- ▶ Prognosis is generally favourable, with eventual recovery in 1–2 months.

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