Lesson of the month 1: Broken heart in the intensive care unit

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Takotsubo cardiomyopathy (TCM) is an unusual form of acute cardiomyopathy showing left ventricular apical ballooning. TCM can masquerade as ST elevation myocardial infarction (STEMI). TCM usually occurs following a variety of emotional stressors, but physical stressors can also trigger the condition, as highlighted by the present case. TCM can occur after an acute medical illness; therefore, physicians should be aware of this condition as a potential cause of inotrope-resistant hypotension. In patients with hypotension and moderate-to-severe left ventricular outflow tract (LVOT) obstruction, inotropic agents should be avoided, because they can worsen the degree of obstruction. Instead, beta-blockers are preferred, because they are capable of resolving the obstruction and consequently improve the haemodynamics.

KEYWORDS: Takotsubo cardiomyopathy, physical stress, ST segment elevation, intensive care unit

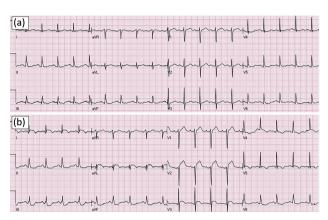


Fig 1. Electrocardiogram (ECG). (a) ECG on admission revealed sinus tachycardia with nonspecific ST-T changes. (b) ECG during the acute episode revealed new ST elevation in leads $V_1 - V_3$, I and AVL, with ST depression in the inferior leads.

Introduction

The presence of new ST-segment elevation in the anterior precordial leads in patients with chest pain usually indicates acute ST elevation myocardial infarction (STEMI) because of occlusion of the left anterior descending (LAD) coronary artery. However, patients can also present with similar findings in the absence of coronary artery disease (CAD). Takotsubo cardiomyopathy (TCM) can masquerade as ST elevation myocardial infarction. TCM usually occurs following a variety of emotional stressors, but physical stressors can also trigger the condition, as highlighted by the present case.

Lesson

A 64-year-old man was hospitalised with acute exacerbations of chronic obstructive pulmonary disease (COPD). At the

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time of initial presentation, he did not have chest pain and was haemodynamically stable. Electrocardiogram (ECG) on admission showed sinus tachycardia with non-specific ST-T changes (Fig 1a). His cardiac enzymes were negative.

Despite aggressive medical therapy for COPD, his respiratory status deteriorated and he consequently required intubation. After intubation, his status improved rapidly and he was extubated 48 h later. However, after extubation, he developed sudden-onset severe dyspnoea, chest pain and diaphoresis. On physical examination, he was afebrile but distressed; his blood pressure was 125/75 mmHg and his heart rate was 105 beats per minute and regular. \mathbf{S}_1 and \mathbf{S}_2 were detected upon heart examination, and diffuse bilateral wheezing was observed upon chest auscultation.

The 12-lead ECG showed new ST segment elevation in leads V_1 – V_3 , I and AVL, with reciprocal ST depression in inferior leads (Fig 1b). Emergent cardiac catheterisation revealed normal coronary arteries with thrombolysis in myocardial infarction (TIMI) grade III flow in all coronary arteries. Transthoracic echocardiography revealed normal contraction of the left ventricle (LV) basal segments, but hypokinetic-akinetic midapical segments with severe systolic dysfunction, consistent with takotsubo cardiomyopathy (Video S1 in the supplementary material). Angiotensin-converting enzyme inhibitor (ACEi) was started at the time of the diagnosis of TCM and beta-blockers (BB) were added on discharge once acute bronchospasm had

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resolved. Follow-up echocardiography at 8 weeks revealed complete resolution of the segmental wall motion abnormalities (Video S2 in the supplementary material).

Discussion

TCM, also known as apical ballooning syndrome, broken heart syndrome or stress-induced cardiomyopathy, is observed in 1.7–2.2% of patients presenting with acute coronary symptoms (ACS).^{1,2} It is characterised by transient systolic dysfunction of the apical and/or mid segments of the LV that mimics myocardial infarction, but occurs in the absence of obstructive CAD.^{1,3} Although the modified Mayo criteria are commonly used for diagnosing TCM, there is currently no consensus on the diagnostic criteria for TCM; therefore, its diagnosis can be difficult.²

TCM most commonly affects women aged 50 years or older, and generally starts abruptly and unpredictably.^{1,4} It is associated with symptoms of chest pain and frequently also with shortness of breath, usually triggered by an emotionally or physically stressful event.^{1,4} Other less common, but likely, triggers of TCM include cocaine and methamphetamine use, opiate withdrawal, excessive phenylephrine use, stress testing using dobutamine, phaeochromocytoma, acute brain injury, lightning strike, ergonovine injections and thyrotoxicosis.^{1,4} However, a triggering event is not always present.

The ECG findings of TCM are often confused with those found during an acute anterior wall STEMI. Most ST elevations (95%) involve the precordial leads and are usually maximal in leads V₂–V₃ However, the amplitude of ST-segment elevations is substantially lower in patients with TCM compared with patients with STEMI caused by LAD occlusion.^{5,6} Initially, normal or nonspecific ECG findings are observed in approximately 15% of patients with TCM.^{1,2,4} Furthermore, cardiac markers are usually elevated in patients with TCM. However, these levels tend to be lower, and normalise sooner compared with ACS.^{1,2} Apical ballooning (typical variant) and/or midventricular hypokinesis are usually seen on echocardiography. In a subset of cases, the transient left ventricular hypokinesis is restricted to the midventricular segments ('atypical variant' or 'apical sparing variant') without involvement of the apex.^{3,4} Perhaps most importantly, these wall motion abnormalities extend beyond the distribution of any single coronary artery. Angiography is required for diagnosis, because there is no accurate method to distinguish TCM from ACS reliably using ECG or cardiac markers. 1,2,4

There are currently no controlled data on the optimal treatment regimen for TCM, but it is reasonable to treat these patients with standard medication for LV systolic dysfunction, including ACEi, BBs and diuretics. ^{1,2,4} The benefits of other standard outpatient post-STEMI medications, such as statins, aspirin, and clopidogrel, are currently unknown. The prognosis of TCM is excellent, with

Supplemental material.

Please find the following videos online at: www.clinmed.rcpjournal.org.

Video 1. Baseline transthoracic echocardiogram (apical three-chamber view) during the acute episode showed normal contraction of left ventricular basal segments, whereas the midapical segments were hypokinetic-akinetic.

Video 2. Transthoracic echocardiogram (apical three-chamber view) at the 8-week follow up showed normalisation of the wall motion abnormalities.

nearly 95% of patients experiencing complete recovery within 4–8 weeks; however, a small subset of patients experience potentially life-threatening complications during the initial presentation, including left ventricular outflow tract (LVOT) obstruction. ^{1,4} LVOT obstruction has been described in 10–25% of all patients with TCM. As highlighted by the present case, TCM can occur after an acute medical illness; therefore, physicians should be aware of this condition as a potential cause of inotrope-resistant hypotension. In patients with hypotension and moderate-to-severe LVOT obstruction, inotropic agents should be avoided, because they can worsen the degree of obstruction. Instead, BBs are preferred, because they are capable of resolving the obstruction and consequently improve the hemodynamics. ^{1,4}

References

- Bybee KA, Prasad A. Stress-related cardiomyopathy syndromes. Circulation 2008;118:397.
- 2 Hurst RT, Prasad A, Askew JW. Takotsubo cardiomyopathy: a unique cardiomyopathy with variable ventricular morphology. J Am Coll Cardiol 2010;3:641–649.
- 3 Eitel I, von Knobelsdorff-Brenkenhoff F, Bernhardt P et al. Clinical characteristics and cardiovascular magnetic resonance findings in stress (takotsubo) cardiomyopathy. JAMA 2011;306:277.
- 4 Sharkey SW, Windenburg DC, Lesser JR et al. Natural history and expansive clinical profile of stress (tako-tsubo) cardiomyopathy. J Am Coll Cardiol 2010;55:333–41.
- 5 Inoue M, Shimizu M, Ino H et al. Differentiation between patients with Takotsubo cardiomyopathy and those with anterior acute myocardial infarction. Circ J 2005;69:89–94.
- 6 Masami Kosuge M, Ebina T, Hibi K et al. Simple and accurate electrocardiographic criteria to differentiate Takotsubo cardiomyopathy from anterior acute myocardial infarction. J Am Coll Cardiol 2010;55:2514–6.

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