CASE REPORT

Takotsubo cardiomyopathy secondary to spontaneous right-sided pneumothorax

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SUMMARY

Takotsubo cardiomyopathy (TCM) is a unique type of cardiomyopathy characterised by left ventricular systolic dysfunction in association with stressful conditions. Patients with this condition usually present with chest pain and dyspnoea, and the presentation can mimic acute coronary syndrome. We present a case of a woman aged 58 years who presented with progressive dyspnoea and cough. Her initial evaluation was suggestive of acute myocardial infarction with elevated serum troponin T and ST segment elevation. Her chest radiograph showed a large right-sided pneumothorax, which was treated with chest tube insertion. Coronary angiography and echocardiogram did not show any evidence of obstructive coronary artery disease but did show a large area of akinesis consistent with TCM. The patient was managed medically with supportive care. Her pneumothorax resolved, and her follow-up echocardiogram also showed improvement. The association between pneumothorax and TCM is rare, and only four other cases have been reported so far in the English literature.

BACKGROUND

Takotsubo cardiomyopathy (TCM) is a transient cardiomyopathy usually precipitated by a stressful event.^{1–10} It can mimic acute coronary syndrome (ACS); therefore, ruling out ACS is an extremely important step in the diagnostic evaluation of TCM.^{1–8} Catecholamine excess has been implicated in the pathogenesis of TCM.^{7 11 12} Although chest pain and dyspnoea are the most common presenting symptoms of TCM, cases of severe heart failure, cardiogenic shock or even sudden cardiac death have been reported as well.^{3–6} The Mayo Clinic criteria are used to diagnose TCM and include findings on echocardiogram and ECG in the absence of obstructive coronary artery disease, pheochromocytoma or myocarditis.^{3 7} Finally, the treatment of TCM is based on excluding ACS and then proceeding with supportive treatment, including heart failure management.⁷

CASE PRESENTATION

A female patient aged 58 years with a history of benign essential hypertension, chronic obstructive pulmonary disease and a 80 pack-year history of tobacco abuse presented to our hospital with progressive dyspnoea and non-productive cough for the past 3–4 days. She denied any chest pain, fever, chills, sputum production, haemoptysis, recent

upper respiratory tract infection, contact with sick patients or recent travel.

Initial clinical evaluation showed an oral temperature of 97.7 F, heart rate 118/min, respiratory rate 22/min, blood pressure 172/102 mm Hg and O₂ saturation 82% on room air. She was in mild to moderate distress. Cardiopulmonary examination revealed decreased air entry on the right side, normal S1 and S2 with tachycardia and no murmurs. Her peripheral pulses were palpable with no peripheral oedema. She was started on 2 L/min of supplemental O₂ via nasal cannula, and her O₂ saturation improved to the low 90 percentile range.

Her chest X-ray (CXR) (figure 1A) showed a large right-sided pneumothorax (~4.5–5 cm), and ECG (figure 1C) showed sinus tachycardia and ST segment elevation in leads V3, V4 and V5 suggestive of ischaemic injury and anterolateral myocardial infarction. Her laboratory workup revealed Troponin T 0.06 ng/mL (normal <0.03 ng/mL), pro-B-type natriuretic peptide 9818 pg/mL (normal for age and gender 10–168 pg/mL), Na⁺ 130 mmol/L (normal 135–145 mmol/L), serum creatinine 0.7 mg/dL (normal 0.9–1.3 mg/dL), white cell count (WCC) count 11 000/mm³ (normal 4000–11 000/mm³) and haematocrit 51.7% (normal 34–47%).

She was given aspirin 325 mg orally, ticagrelor 180 mg orally and unfractionated heparin intravenously 3500 units as initial bolus dose followed by 700 units/hour continuous infusion. She was also given atorvastatin 80 mg orally and metoprolol tartrate 12.5 mg orally. She was admitted to the intensive care unit (ICU) with concern for myocardial infarction and spontaneous secondary pneumothorax. Cardiology and thoracic surgery were consulted urgently. A chest tube was inserted. On further evaluation of her ECG, the cardiology team noted that she had an established Q wave. The initial decision was to continue medical treatment and observation, as she was pain-free, the ECG did not show dynamic changes suggestive of ongoing ischaemia and her repeat troponin-T was 0.08 ng/mL (previous was 0.06 ng/mL).

On hospital day 2, an echocardiogram (figure 2A, B) showed reduced left ventricular systolic function with a hyperdynamic base and a calculated ejection fraction (EF) of 39%, large area of akinesis (including mid and distal septal (anteroseptal and inferoseptal), apical, mid and distal anterior, mid and distal inferior, and mid and distal lateral) and a small anterior pericardial effusion that measured 1.1 cm in greatest dimension without any underlying tamponade physiology. Cardiac

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Figure 1 (A) Chest X-ray (CXR) on presentation showing large right-sided pneumothorax. (B) CXR prior to discharge showing resolution of pneumothorax. (C) ECG on presentation showing sinus tachycardia with ST segment elevation in V2–V4 suggesting acute anterior infarct.

Figure 2 (A, B) Echocardiogram with apical two-chamber view (A) and apical four-chamber view (B) showing cardiac wall motion abnormality in the left anterior descending (LAD) distribution. The estimated ejection fraction (EF) was 39%. (C, D) A follow-up echocardiogram with apical two-chamber view (C) and apical four-chamber view (D) showing normal left ventricular systolic function with an estimated EF of 60% and no wall motion abnormality.

catheterisation was performed (figure 3) which showed no significant coronary artery disease; decreased left ventricular EF of 37% and anterior, apical and inferior severe hypokinesia to akinesia with apical ballooning were noted. These findings with her clinical presentation were consistent with TCM.

TREATMENT

Aspirin 81 mg daily and atorvastatin 20 mg daily were continued. The β blocker was switched to metoprolol succinate 25 mg daily, and she was started on lisinopril 5 mg daily, with attention paid to avoiding diuresis, dehydration and preload reduction.

Figure 3 (A, B) Coronary angiography showing normal left anterior descending (A) and right coronary artery (B). (C) Left ventriculogram showing hyperdynamic basal left ventricular segment with apical left ventricular ballooning.

OUTCOME AND FOLLOW-UP

The patient showed continual improvement subjectively. Her follow-up CXR (figure 1B) showed resolution of the right-sided pneumothorax, and her chest tube was pulled out on hospital day 4. She was discharged on aspirin 81 mg daily, atorvastatin 20 mg daily, metoprolol succinate 25 mg daily and lisinopril 2.5 mg daily in addition to her inhalers. She followed-up in our Cardiology and Pulmonary clinics. A follow-up echocardiogram (figure 2C, D) showed normal left ventricular systolic function with an estimated EF of 60% and no wall motion abnormalities. Serum metanephrine was checked as a follow-up test in the outpatient setting and was 0.2 nmol/L (<0.50 nmol/L).

DISCUSSION

TCM is a transient cardiomyopathy and regional systolic dysfunction of the left ventricle, usually precipitated by a stressful event that can mimic myocardial infarction.^{1–10} Other names for this syndrome include apical ballooning syndrome, broken heart syndrome, stress cardiomyopathy and stress-induced cardiomyopathy. It was first described by Sato *et al* in 1990.^{1 2}

TCM accounts for 1–2% of patients who present with positive troponins and concern for ACS or ST-elevation myocardial infarction.^{8–10} The onset of TCM is usually triggered by severe emotional or physical stress.^{3–7 9} However, the incidence of TCM in individuals exposed to stress is not known. The exact pathogenesis of TCM is not well understood. The most common explanation includes catecholamine excess, which induces coronary artery spasm, microvascular dysfunction and myocardial toxicity.^{7 11–13} Several findings support the catecholamine excess hypothesis: first, the association between TCM and a stressful trigger in up to 70% of cases;^{3–7 9} second, plasma levels of catecholamine are more elevated in TCM cases than similar cases with plaque-rupture myocardial infarction;² and third, the demonstration of mononuclear infiltration and contraction band necrosis in endomyocardial biopsies of patients with TCM, findings that are usually seen in states of catecholamine excess. In general, TCM is more common in postmenopausal women.^{3–6 8} Oestrogens may have an important protective role against catecholamine-induced myocardial injury or at least, may influence the myocardial response to various stressful triggers.^{14–16}

Table 1 Reported cases of takotsubo cardiomyopathy associated with pneumothorax

Case	Age (years)	Gender	Presenting symptoms	Pneumothorax	ECG	Echo/left ventriculography	Cardiac catheterisation	Initial serum cardiac troponin-T (ng/mL)
Akashi <i>et al</i> ¹⁹	83	Female	Chest pain, progressive dyspnoea and cough	Left-sided	Sinus tachycardia and ST elevations in V2–V5	Apical akinesis and basal hyperkinesis	Normal coronary arteries	NA
Kumar <i>et al</i> ²⁰	64	Female	Chest pain	Right-sided	Sinus tachycardia, ST elevations and T inversions in anterior and inferior leads	Global hypokinesis with apical ballooning, and basilar hyperkinesis, EF 15%	Non-obstructive coronary artery disease	0.76
Gale <i>et al</i> ²¹	78	Female	Severe dyspnoea	Left-sided (Tension)	Sinus tachycardia, ST elevations and T wave changes in lateral leads	Global hypokinesis sparing the basal segments, apical ballooning, EF 13%	Normal coronary arteries	16.5
Mittal <i>et al</i> ²²	58	Female	Chest discomfort, cough and fever followed by progressive dyspnoea and hypotension	Right-sided (Tension)	ST elevation	Anterior wall hypokinesis with decreased EF	Normal coronary arteries	NA
Our case 2015	58	Female	Progressive dyspnoea and cough	Right-sided	Sinus tachycardia and ST elevation in V3–V5	Global akinesis sparing the base, EF 39%, small anterior pericardial effusion without an underlying tamponade physiology	Non-obstructive coronary artery disease	0.06

Animal models show that oestrogen-deficient states predispose patients to endothelial dysfunction, which increases the risk of stress-induced microcirculation dysfunction.¹⁵

The most important step in the evaluation of suspected TCM is to rule out ACS and myocardial infarction. The Mayo Clinic diagnostic criteria can be used to diagnose TCM. All four of these criteria are required for the diagnosis:^{3 7} first: transient left ventricular systolic dysfunction with wall motion abnormalities that extend beyond the distribution of a single coronary artery,⁷ second: absence of obstructive coronary artery disease or acute plaque rupture, third: new ECG abnormalities or elevated cardiac troponins, and^{6 17 18} fourth: absence of pheochromocytoma or myocarditis.

It is worth mentioning that pneumothorax could induce some ECG changes that result in ST segment elevation, but co-occurrence of pneumothorax and TCM is very uncommon. One possible explanation for the association between these conditions is that stress and hypoxaemia may induce catecholamine release, which can in turn contribute to cardiac dysfunction. To the best of our knowledge, there are only four other cases of TCM associated with pneumothorax in the English literature, two cases of spontaneous pneumothorax and two cases of tension pneumothorax^{19–22} (table 1).

Learning points

- ▶ Takotsubo cardiomyopathy (TCM) is characterised by a transient cardiomyopathy and regional systolic dysfunction of the left ventricle, usually precipitated by a stressful event.
- ▶ TCM usually presents with chest pain and dyspnoea and mimics myocardial infarction.
- ▶ Pneumothorax could induce some ECG changes that result in ST segment elevation.
- ▶ The association between pneumothorax and TCM is very uncommon even though it is reported.

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