Large right-sided pneumothorax with Takotsubo cardiomyopathy

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ABSTRACT

Patients with left-sided pneumothorax presenting with electrocardiogram (ECG) changes resembling acute coronary syndrome (ACS) have been reported in the literature. Takotsubo cardiomyopathy (TCM) occurs predominantly in post-menopausal women with underlying intense emotional or physical stress. However, as this case report shows, it can complicate any acute illness leading to intense stress. We report a rare combination of ECG changes mimicking ACS in a man with a large right-sided pneumothorax and TCM.

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INTRODUCTION

Pneumothorax causing various electrocardiogram (ECG) changes, published in the early part of the 20th century, was described first in patients with artificial pneumothorax.[1] Subsequently, there have been many reports describing ECG changes in pneumothorax with patterns similar to other serious respiratory and cardiovascular conditions, such as pulmonary embolism and myocardial infarction.[2]

Similarly, there have been reports of tension pneumothorax associated with Takotsubo cardiomyopathy (TCM) causing ECG changes mimicking acute coronary syndrome (ACS). Takotsubo (meaning Japanese octopus fishing pot) cardiomyopathy was first described in Japan in 1990 by Sato *et al*.[3] It is also known as stress cardiomyopathy, broken heart syndrome or apical ballooning syndrome.[4]

We report a rare combination of right-sided large pneumothorax with TCM in a man associated with ECG changes mimicking ACS.

THE CASE

A 75-year-old man without any comorbid conditions or habits presented with complaints of worsening exertional breathlessness (NYHA II), retrosternal chest discomfort and cough with scanty expectoration for one week. On examination, his pulse rate was 120 beats/minute, blood pressure: 90/60 mmHg and respiratory rate: 33/minute with elevated jugular venous pulse (10 cm H_2O above sternal angle). His arterial oxygen saturation was 85% on room air. Respiratory system examination revealed that the trachea was pushed to the left side and there was a hyper-resonant note on percussion with decreased breath sounds on the right hemithorax. The patient had had pulmonary tuberculosis 15 years back, treated and declared cured with no further recurrence or reinfection. ECG showed sinus rhythm, ST coving in the anterior chest leads V1 to V4 with normal R-wave progression and terminal T inversions in lateral chest leads (V4–V6). There were no reciprocal ST-T changes in inferior leads (Fig. 1a). A provisional diagnosis of right pneumothorax was made with ACS as a differential diagnosis.

A chest X-ray (CXR) revealed a large pneumothorax in the right lung. High-resolution computed tomography (HRCT) of the thorax showed features of chronic obstructive pulmonary disease with emphysematous bullae in both the lungs with large tension pneumothorax due to a ruptured bulla in the right lung causing collapse of adjacent lung tissue (Figs 2a and b). Two-dimensional echocardiogram (2D-ECHO) revealed regional wall motion abnormality (RWMA) with severe hypokinesia in mid-apical segments and apical ballooning (Figs 3a and b) with normally contracting basal segments (features of TCM) and severely reduced left ventricular ejection fraction (LVEF; 25%). Cardiac biomarkers were elevated (troponin T 1.023 ng/ml and CK-MB 14 IU/L). Erythrocyte sedimentation rate was 40 mm/hour with raised C-reactive protein (6 mg/L) and B-type natriuretic peptide (9500 pg/ml). Three sputum samples and cartridge-based nucleic acid amplification test (CB-NAT) for tuberculosis and serology for human immune deficiency virus, hepatitis B and hepatitis C were negative. Other investigations such as complete blood count, renal, liver and thyroid function tests, serum electrolytes, calcium, magnesium, lipid profile and blood sugars were within normal limits.

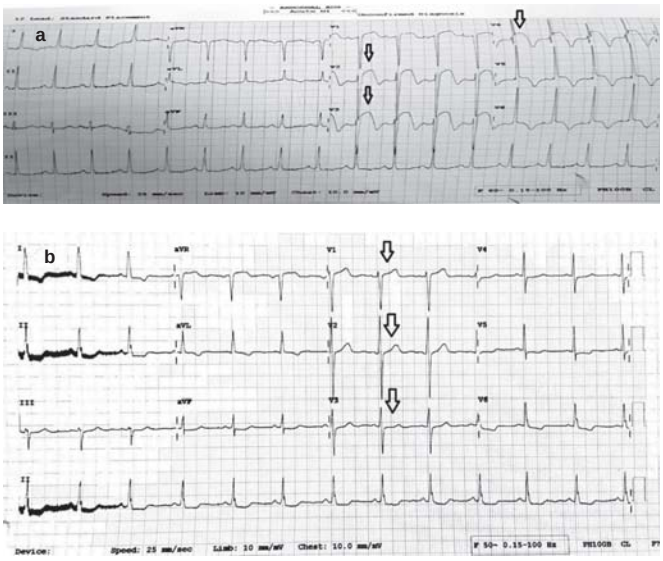

FIG 1. (a) Electrocardiogram at presentation: Downward arrows showing ST coving with J point elevation in V1 to V4 with terminal T inversions in V5–V6, I and aVL.
(b) On day 3, post-intercostal drain, as pneumothorax resolved, electrocardiogram also showed resolution of ST elevations or coving in V1 to V4 (downward arrows). V5–V6, I and aVL continued to show terminal T inversions.
II and aVF show subtle ST depression

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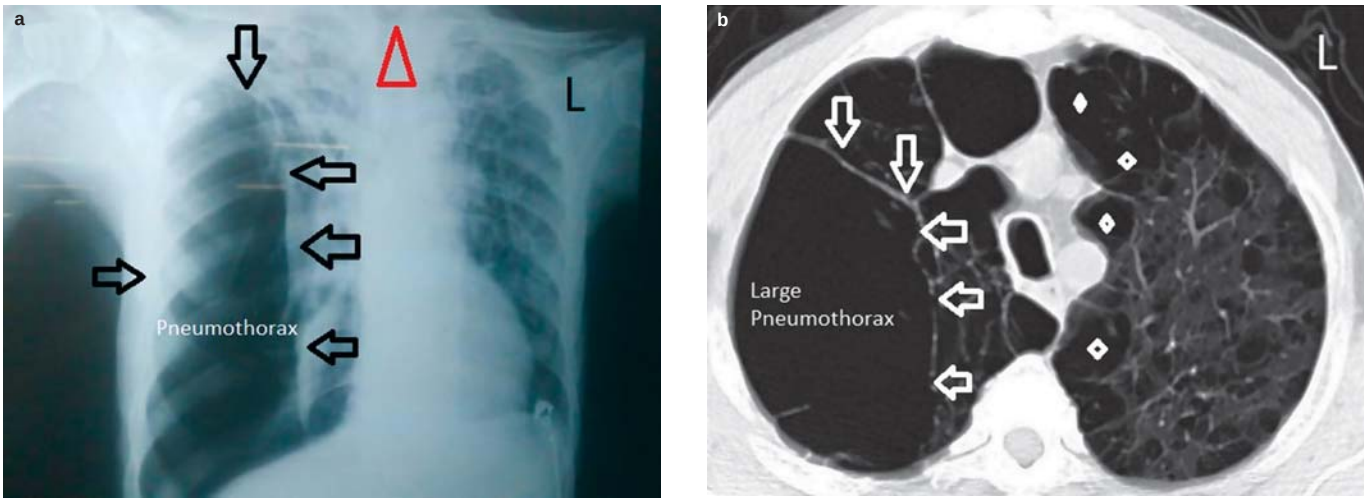

FIG 2. Chest X-ray and high-resolution computed tomography image showing right-sided pneumothorax: (a) black arrows pointing at large pneumothorax in the right lung; upward facing red arrowhead showing trachea pushed towards left; (b) white arrows pointing to pneumothorax and diamond white markings showing multiple bullae in the left lung

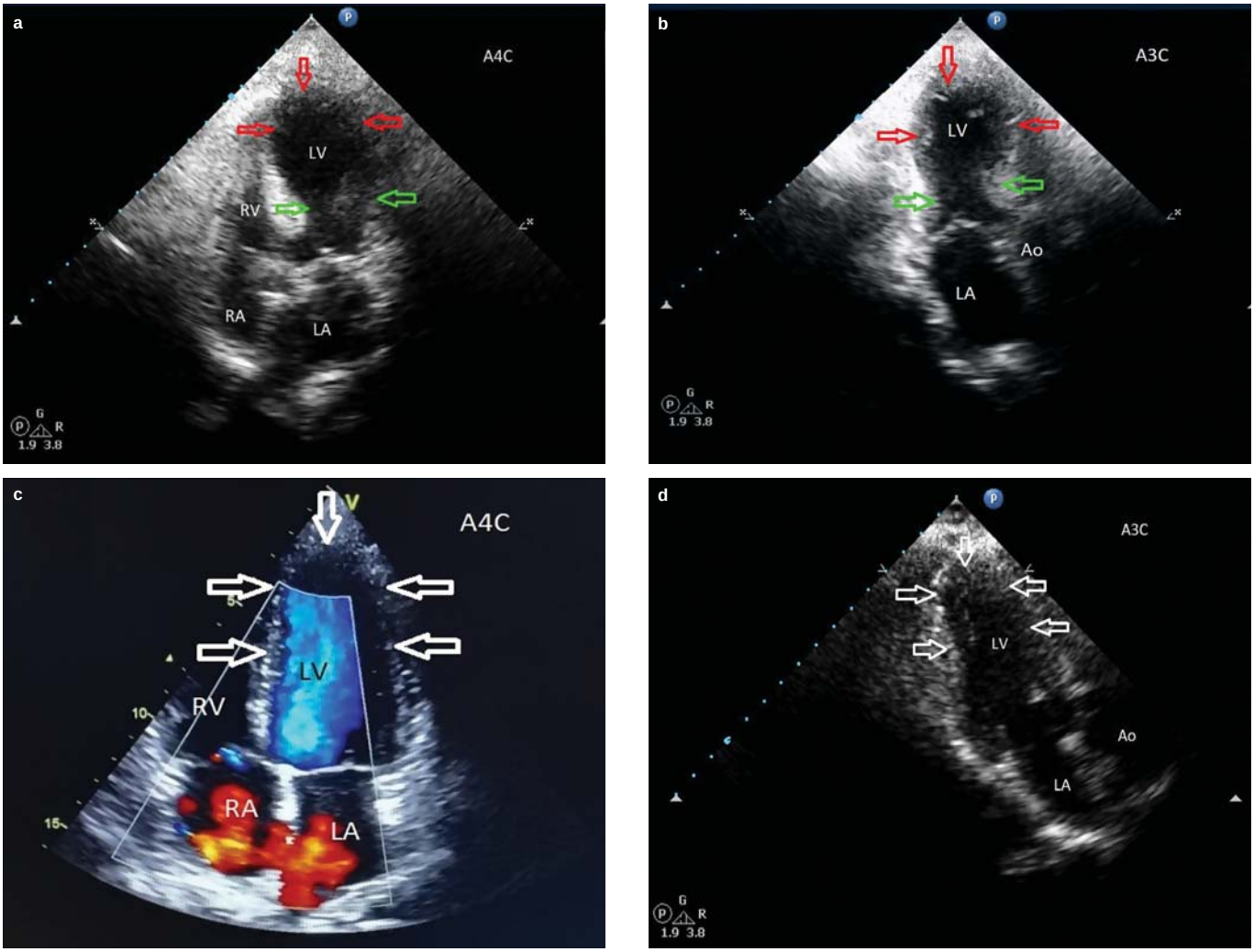

FIG 3. Echocardiogram showing apical ballooning (red arrows), green arrows depicting normal basal segments: (a) apical four-chamber; (b) apical three-chamber views at presentation; echocardiogram during 4 weeks of follow-up showing the disappearance of apical ballooning and normal left ventricular chamber morphology (white arrows); (c) apical four-chamber view with colour Doppler; and (d) apical three-chamber view

Management

After initial evaluation, a diagnosis of right-sided large tension pneumothorax with TCM was made and he was started on low-dose aspirin and statin, inotropes and nebulization along with other measures for heart failure with the insertion of a negative pressure intercostal drain (ICD) on the right side. Serial CXRs showed gradual expansion of the right lung with ECG showing resolution of ST coving in anterior chest leads (Fig. 1b). ICD was removed on day 5. Repeat ECHO showed improvement in LVEF to 40% with RWMA disappearing and LV showing mild global hypokinesia. Apical ballooning had disappeared. After stabilization, on day 6, he underwent a coronary angiogram which revealed normal epicardial coronaries. He was discharged on day 7 with nebivolol, antibiotics, metered dose inhalers for chronic obstructive airway disease and oral paracetamol for pain. The patient recovered his LV function completely at 4 weeks of follow-up (Figs 3c and d) and has been asymptomatic for the past 2 years.

DISCUSSION

In many patients with left-sided pneumothorax, transient, ischaemic type ECG changes have been reported leading to both diagnostic and therapeutic challenges.[5,6] Most published data on ECG changes in pneumothorax are based on few summaries in small groups of patients with no large data available.[1] Various ECG changes have been reported with spontaneous pneumothorax of both left[1,5,6] and right sides.[7,8] The spectrum of such ECG findings includes a change in axis, ST-segment elevation, diminished R-wave amplitude, poor progression across chest leads and inverted T-waves.[5,8] Most often, these changes revert to normal with decompression of the pneumothorax. Although few ECG changes are more pronounced and frequent in left-sided pneumothorax, such as ST deviations, abnormal axis, T-inversions and decreased precordial mean QRS amplitude[5,6] compared to right-sided pneumothorax, no particular ECG sign or signs are pathognomonic of any one side. Possible mechanisms for these ECG changes include cardiac rotation, right ventricular dilatation, cardiac displacement and the insulating effect of air accumulated between the heart and chest wall.[2,9] Littman proposed that if the currents generated in the living heart produce a given curve when obtained from two electrodes on the surface of the body, this curve will alter if these currents are forced to complete their circuit by a different pathway, and suggested that air could act as an insulator by changing electrical conductance through the chest wall.[9]

Tension pneumothorax is known to be associated with true ischaemic changes on ECG, accompanied by wall motion abnormalities, which can be demonstrated by 2D-ECHO due to impaired coronary perfusion. Stress cardiomyopathy or TCM is an acute cardiac condition characterized by transient systolic dysfunction of the LV apex and mid-ventricle with depressed LV function mimicking ACS and recovers within a few weeks.[10,11] TCM occurring, especially in post-menopausal women, during acute illnesses or during a procedure is well-documented. The condition is diagnosed based on modified Mayo Clinic criteria,[12] which include transient RWMA not limited to single epicardial vascular distribution, ECG changes or elevated cardiac biomarkers, no angiographic evidence of culprit coronary artery disease and absence of myocarditis or pheochromocytoma.[12] Potential ECG findings of TCM include ST-segment elevation, ST-segment depression, new-onset left bundle branch block or prolonged QT interval.[13] Often, these findings overlap with those caused by pneumothorax leading to a diagnostic dilemma. However, we maintain that ECG changes in our patient could be due to pneumothorax rather than TCM, as the insertion of the chest tube and lung expansion led to the resolution of ECG changes.

Although the exact mechanism of TCM is not known, multivessel coronary spasm or catecholamine cardiotoxicity have been suggested as possible causes.[14] Many clinical conditions have been reported to trigger TCM such as severe emotional or physical stress including central nervous system pathology, severe illness, drug use or withdrawal and severe pain.[14,15] Studies have shown TCM to be affecting predominantly women, and the percentage of men experiencing it ranged from 4.4% to 12.7%.[16] Although affected men have increased morbidity and mortality, the reason for the preponderance of woman with TCM is not clear.[16]

Conclusion

Though less common, important ECG changes can occur with right-sided pneumothorax. When TCM occurs in the setting of pneumothorax, the resulting ECG changes can pose diagnostic and therapeutic challenges. TCM should be considered as a differential diagnosis even among men with ACS mimics.

Conflicts of interest. None declared

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