

## **Takotsubo Cardiomyopathy Secondary to Acute Asthma**

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Takotsubo Cardiomyopathy (TCM) is an acute reversible cardiomyopathy induced by physical or emotional stress, first described in Japan<sup>1</sup>. It is characterised by chest pain, mildly elevated cardiac biomarkers, electrocardiogram (ECG) changes and left ventricular dysfunction with reduced ejection fraction (EF) and morphological changes on echocardiography (ECHO). Acute asthma is a rare precipitant with few cases reported.<sup>2,3</sup>

A seventy-four-year-old woman was admitted with an acute asthma exacerbation due to respiratory syncytial virus (RSV) bronchiolitis. She presented with dyspnoea, wheeze and denied chest pain. Diffuse expiratory wheeze was evident on auscultation. Blood pressure on admission was 150/65, heart rate 123, respiratory rate 22, SpO<sub>2</sub> 98% on three litres of oxygen, and temperature 36.6. Arterial blood gas (ABG) showed respiratory acidosis with pH 7.1, pCO<sub>2</sub> 8.11 and pO<sub>2</sub> 14. Chest radiograph was clear. She responded to intravenous (IV) antibiotics, steroids and nebulised bronchodilators and maintained 95% SpO<sub>2</sub> on room air.

Subsequently she developed increasing respiratory distress and desaturated to 70% SpO<sub>2</sub> on room air, warranting intubation and ICU admission. Repeat examination revealed bilateral inspiratory crackles up to mid zones. ECG showed sinus tachycardia and old left bundle branch block (LBBB). Cardiac troponin rose from 31 to 111. Repeat chest radiography was consistent with acute pulmonary oedema. Bedside ECHO showed ballooning of the left ventricular apex with no regional wall motion abnormality. IV hydrocortisone, furosemide, dobutamine infusion and empiric antibiotic cover lead to rapid improvement in respiratory and haemodynamic status. Serial portable chest radiographs showed improvement in bilateral airspace opacification and pleural effusions. She was extubated and transferred to the ward for rehabilitation. Departmental ECHO revealed left ventricular septal hypokinesis and apical septal akinesis with normal chamber and wall dimensions. She was discharged at functional baseline with Cardiology follow-up for coronary angiography.

Almost ninety percent of TCM patients are female over the age of 50<sup>4</sup>. Presentations include chest pain, dyspnoea, syncope, arrhythmias, cardiogenic shock, cardiorespiratory arrest or sudden death.<sup>4</sup> ECG shows LBBB, ST elevation, T wave inversion or pathological Q waves.<sup>4</sup> Other signs include bilateral infiltrates on chest radiography and left ventricular dysfunction on ECHO with mean EF of 20-49%<sup>4</sup>. Cardiac biomarkers are mildly elevated and normalise sooner than in acute coronary syndrome (ACS).<sup>4</sup> Evidence of no coronary obstructive lesion on coronary angiography definitively differentiates TCM from ACS.<sup>4</sup>

There is no definitive consensus on the mechanism of TCM. It tends to be preceded by stress, which induces endogenous catecholamine release. A systematic review found elevated noradrenaline levels in almost 75% of patients.<sup>4</sup>

Treatment in the acute phase is supportive and depends on the ensuing complications, most commonly heart failure with or without acute pulmonary oedema<sup>4</sup>. This includes upright posture, oxygen, diuresis, ventilation, intra-aortic balloon pump, vasopressor and inotropic support. TCM usually resolves within weeks to months with full recovery and rarely recurrence<sup>4</sup>. In-hospital mortality is estimated at 1-3%.<sup>4</sup> Left ventricular function may determine the prognosis.<sup>1</sup>

This case demonstrates acute respiratory failure driving acute heart failure. It highlights the importance of early suspicion of TCM and recognition of asthma as a potential driver.

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