Tako-tsubo Cardiomyopathy (Transient Left Ventricular Apical Ballooning Syndrome): A Case Report

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Chest pain, elevated cardiac enzymes and dynamic ST-T change on an electrocardiogram (ECG) are always diagnosed as acute coronary syndrome. Tako-tsubo cardiomyopathy has similar clinical manifestations and is characterized by transient left ventricular dysfunction without significant coronary artery disease. We report on a 70-year-old woman with chest pain and dyspnea. She was diagnosed and managed as suffering from acute coronary syndrome initially, but Tako-tsubo cardiomyopathy was diagnosed after a series of examinations. She was discharged and maintained on medications, including aspirin, ACE inhibitor, nitrates and diuretics. At the outpatient department follow-up, her left ventricular dysfunction had recovered completely.

Key words: apical ballooning, Tako-tsubo, transient left ventricular dysfunction

Introduction

Tako-tsubo cardiomyopathy, also known as transient left ventricular apical ballooning syndrome, was first described in the Japanese literature in 1991 by Dote et al\(^1\). More current names include broken heart syndrome, stress-induced cardiomyopathy or stress cardiomyopathy. Tako-tsubo cardiomyopathy accounts for 1% of admissions for suspected acute myocardial infarction in Japan\(^2\) and it is increasingly recognized in the West. To our knowledge, only two cases have been reported in Taiwan\(^3,4\).

The syndrome is characterized by abrupt onset of angina-like chest pain, mild creatine kinase (CK) and troponin I elevations, electrocardiogram (ECG) changes that typically demonstrate anterior ST segment elevation on precordial leads V3-6 or deep T wave inversion with QT prolongation. The left ventriculogram demonstrates hypokinesis or akinesis from the mid portion of the anterior wall to the apex, and hyperkinesis of the basal area. The clinical presentation mimics acute myocardial infarction but without evidence of coronary artery stenosis on angiography, and the left ventricular dysfunction can completely regress.

We now describe such a case, treated as acute coronary syndrome initially, in which the diagnosis was made after coronary angiography was performed.

Case Report

A 70-year-old woman was brought to our emergency room because of acute substernal chest pain and dyspnea after a gastrointestinal (GI) upset. She had had a 4-year history of diabetes mellitus and hypertension without regular treatment. There
was no history of chest pain or smoking in the past. On physical examination, her blood pressure was 170/110 mmHg, pulse 140 beats per minute, respirations 26 per minute and temperature 36 °C. There were bilateral crackles in the lung fields. The heart rhythm was regular and a grade 2/6 systolic murmur was heard at the apex of the heart. A chest X-ray film showed mild cardiomegaly and increased diffuse pulmonary vascularity (Fig. 1A). The ECG showed sinus tachycardia with ST segment elevation in leads V2-5 (Fig. 1B). Her blood sugar was 418 mg/dl, CK and CK-MB were 160 and 20 IU/L, respectively, and troponin I was 0.67 ng/ml (normal range <0.5 ng/ml). Complete blood count and other blood biochemistry were normal. Under the impression of acute coronary syndrome with acute pulmonary edema, aspirin, clopidogrel and low molecular weight heparin were given, and furosemide and nitrates were administered parenterally. Her chest pain and dyspnea improved and a follow-up ECG showed resolution of ST elevation with biphasic T wave changes in V4-6 (Fig. 1C). She was immediately admitted to our medical intensive care unit (ICU). Twenty-four hours later, her peak CK and CK-MB were 674 and 75 IU/L, respectively, and troponin I was 7.64 ng/ml. An ECG revealed deep T-wave inversion in I, II, aVL, aVF and V2-6 leads, with QT prolongation (Fig. 2A). On physical examination, the patient looked well, her heart rhythm was regular and mild basal rales were heard on low lung fields. Her blood
pressure was 120/70 mmHg. Echocardiography showed hypokinesis over the mid portion to the apex of the left ventricle. Cardiac catheterization was performed on the third day of hospitalization and revealed no evidence of coronary artery stenosis (Figs. 3A, 3B). Contrast left ventriculography demonstrated hypokinesis from the mid portion of the anterior wall to the apex (Figs. 3C, 3D) and an ejection fraction of 40%. The patient was diagnosed with Tako-tsubo cardiomyopathy. She remained stable and was transferred to a ward on the fourth ICU day. She was discharged on the sixth day after admission, and maintained on a medication regimen of aspirin, ACE inhibitor, diuretics, nitrates and metformin. One month later, repeated echocardiography showed complete resolution of the left ventricular wall motion abnormalities. The ejection fraction had improved to 65%. The ECG still revealed deep T wave inversion. Four months later, she was admitted to our hospital again due to poor control of diabetes. At that time, the ECG revealed normal sinus rhythm without significant ST-T change (Fig. 2B). [Fig. 2B is referred to after Fig 3.]

**Discussion**

In recent years, more and more articles about this syndrome have been published, especially in Japan and the West. However, the causes are still not clearly understood. Several mechanisms for reversible cardiomyopathy have been proposed: possibilities include the sex hormone, epicardial coronary arterial spasm, microvascular spasm or dysfunction, catecholamine-induced myocardial stunning, ruptured plaque and myocarditis.

As most patients affected by this syndrome are women, it has been attributed to the sex hormone, which exerts important influences on the sympathetic neurohormonal axis and coronary vasoreactivity\(^5\). High levels of catecholamine can induce myocardial stunning and transient left ventricular dysfunction. The same phenomenon has been reported in female patients who have suffered from a subarachnoid hemorrhage\(^6\), but the possible impact of this phenomenon has not been investigated in detail. Epicardial coronary arterial spasm may be a potential cause for this syndrome; a high percentage of coronary spasm in response to a provocative test was demonstrated in several studies\(^2,7\) but the results were conflicting\(^8,9\). In our patient, her chest pain and dyspnea improved soon after nitrates infusion, and resolution of ST segment elevation also occurred. Therefore, coronary vasospasm cannot be excluded, although a provocative test was not done. Microvascular spasm or dysfunction may be a possible cause of stress cardiomyopathy\(^7,10-12\). Reduced coronary flow reserve in Doppler flow wire measurements and higher thrombolysis in myocardial infarction (TIMI) frame counts were found in these patients. It has been reported that psychosocial stress increases the risk of myocardial infarction\(^13,14\). It has also been found that stressful events or aggravation of underlying disorders might trigger Tako-tsubo cardiomyopathy\(^2,9,15\). Wittstein et al. demonstrated that plasma catecholamine levels were markedly higher among patients with stress cardiomyopathy than among those with Killip class III myocardial infarction\(^8\). Their conclusions pointed toward an exaggerated sympathetic response with an increased release of catecholamines as a probable cause. We did not find a definitive trigger event for our patient but such a trigger was not observed in one third of these patients either. Chronic underlying disease with GI upset might have been a stressor to her. Other hypotheses are plaque rupture or myocarditis. Sharkey et al. did not demonstrate fixed atherosclerotic plaque and failed to detect regional T2 enhancement in cardiac magnetic resonance imaging (MRI) study, so plaque rupture or myocarditis did not play a role in these cardiac events\(^9\).

The treatment of Tako-tsubo cardiomyopathy
Fig. 2A  Twelve-lead electrocardiogram performed 24 hours later demonstrating deep T wave inversion with QT prolongation

Fig. 2B  Twelve-lead electrocardiogram performed 4 months later demonstrating no significant ST-T change

Figs. 3A, 3B  Coronary angiography (A: left coronary artery, B: right coronary artery). No coronary artery stenosis is seen

Figs. 3C, 3D  Left ventriculogram showing hypokinesis of the mid and apical portions of the left ventricle (C: End-diastole, D: End-systole)
is supportive care for congestive heart failure with diuretics and vasodilators. As a massive catecholamine release has been found in this syndrome, beta blockers could be used. Nitrate or calcium channel block have some benefits if coronary spasm is suspected, such as in our present case. Vasopressors or intra-aortic balloon counterpulsation are indicated if a hemodynamic compromise develops\(^8\,16\). Anti-coagulant may be added if thrombus is found. Although the overall prognosis is good, the transient left ventricular dysfunction can result in congestive heart failure with pulmonary edema, transient conduction abnormalities and other arrhythmia. Cardiogenic shock and mortality have also been noted, so aggressive medical treatment with adequate hemodynamic support is very important\(^7\,9\,16\).

Chest pain, elevated cardiac enzymes, and dynamic ST and T wave changes on an ECG are always diagnosed as acute coronary syndrome. Tako-tsubo cardiomyopathy’s clinical presentation mimics acute coronary syndrome but the management and prognosis are very different. Differentiating a patient who presents with a suspected acute coronary syndrome or ST-segment elevation myocardial infarction is important for cardiologists and emergency department physicians. To distinguish the two conditions using standard 12 lead ECG is difficult. The absence of reciprocal changes and abnormal Q waves, and significantly higher ratios of ST segment elevation in leads V4-6 to V1-3 have been reported in patients with this syndrome\(^17\). Our patient showed the above ECG findings. However, ST-segment elevation was less common in series reports from the United States\(^8\,9\). Tako-tsubo cardiomyopathy should be considered especially when the extent of left ventricular wall motion abnormalities exceeds the biomarker evidence for myocardial necrosis, and coronary angiography confirms no coronary artery stenosis. As complete recovery of systolic function and restoration of normal functional capacity can be expected for these patients, aggressive treatment with pharmacological agents and/or mechanical circulatory support are indicated.

**References**


Tako-tsubo心肌病變
(暫時性左心室心尖球狀突出症候群)一病例報告

陳政康  陳澄黎

胸痛，心肌酶升高以及心電圖ST節的變化，通常會被診斷為急性冠心症。Tako-tsubo心肌病變有類似的臨床表徵。暫時性左心室功能不良合併正常的冠狀動脈是其特徵。我們報告一位70歲女性病例，起初被當作急性冠心症來治療，最後診斷為Tako-tsubo心肌病變。

關鍵詞：心尖球狀突出，Tako-tsubo，暫時性左心室功能不良