Transient Left Ventricular Apical Ballooning Syndrome: A Case Report

Che-Ming Chang1,2,3, Kou-Gi Shyu2,3,4

Transient left ventricular apical ballooning syndrome is a rare cause of acute chest pain mimicking acute myocardial infarction. Presentations include ischemic-like chest pain, ST-segment elevation, cardiac biomarker elevation, and characteristic midventricular and apical wall motion abnormalities in the absence of obstructive epicardial coronary disease. We describe a case of transient left ventricular apical ballooning syndrome in a 59-year-old man who presented with chest pain and ventricular tachyarrhythmia in our emergency department. Successful direct cardioversion was performed in the emergency department. Cardiac catheterization showed patent coronary arteries with midventricular and apical wall motion abnormalities and transient left ventricular apical ballooning syndrome was impressed. Transient left ventricular apical ballooning syndrome should be included in the differential diagnosis of acute chest pain.

Key words: ST-segment elevation myocardial infarction, tako-tsubo cardiomyopathy, transient left ventricular apical ballooning syndrome

Introduction

The first findings of transient left ventricular apical ballooning syndrome (TLVABS), also known as tako-tsubo-like cardiomyopathy, were described in Japan in the early 1990s(1). It can mimic ST-segment elevation myocardial infarction (STEMI), leading to emergency thrombolysis or coronary angiography in many patients. Patients presenting with this syndrome are usually post-menopausal women who present with ischemic-like chest pain, ST-segment elevation, low level cardiac biomarker elevation, and characteristic midventricular and apical wall motion abnormalities in the absence of obstructive epicardial coronary disease. The electrocardiographic changes in TLVBS are similar to those seen with anterior STEMI, including ST-segment elevation in the anterior precordial leads, QT-interval prolongation, and evolutionary T-wave inversions(2). Here, we report a case of TLVABS in a 59 year-old man who presented with chest tightness for two hours and ventricular tachycardia (VT).

Case Report

A 59-year-old man presented to the emergency department via the outpatient department (OPD) with chest tightness and cold sweating for two hours. His blood pressure was 80/60 mmHg in
the OPD. He had no episode of emotional stress before this episode. He had been treated with a cholesterol-lowering agent (Fluvastatin 80 mg 1#QD) for hypercholesterolemia for about 11 months and an angiotensin-converting enzyme (ACE) inhibitor (Ramipril 10 mg 1# QD) plus beta-blocker (Bisoprolol 5 mg 1#QD) for hypertension for nearly 4 years. He is a teacher. Ventricular tachycardia with a heart rate of 200 bpm was noted in the emergency department and cardioversion at 100 joules was done immediately (Fig. 1A).

After cardioversion, his heart rhythm reverted to normal sinus rhythm (HR 80 bpm) (Fig. 1B) and his blood pressure was 140/90 mmHg. Routine blood tests were done after cardioversion and showed only mildly elevated cardiac enzymes – troponin I 0.766 ng/ml (normal < 0.5 ng/ml), CPK 209 ng/ml (normal range 39-308 ng/ml), and CK-MB 42 ng/ml (normal range 7-25 ng/ml). Serum electrolytes were within normal limits. Electrocardiography (Figure 1B) showed normal sinus rhythm with first degree AV block, and an inverted T wave in V3-V6, and I, aVL, aVF and Lead II. Acute coronary syndrome was impressed and coronary angiography was performed after his condition became stable (the 2nd day after this episode). The coronary angiogram showed normal coronary arteries. His left ventriculogram showed hypokinesis of the left ventricular apex with ballooning during systole (Fig. 2), but the proximal parts of the left ventricle displayed exaggerated contraction. An echocardiogram performed one month later showed complete resolution of the hypokinesis of the left ventricular apical portion, disappearance of the apical ballooning and complete recovery of left ventricular function. Cardiac serology after cardiac catheterization was within normal limits. After cardioversion, the patient had an uneventful hospital course and was discharged on amiodarone (200mg 1#BID).

![Figure 1](image-url)  
**Fig. 1** Electrocardiograms: A. Ventricular tachycardia; B. After cardioversion, there is T-wave inversion in Leads I,II, aVF, aVL, and V3-V6.
Discussion

TLVABS is a unique disorder which was first described in Japan in the early 1990s\(^1\), and in the USA\(^2\) and Europe\(^3\) in the early 2000s as a transient cardiomyopathy that primarily affects post-menopausal women\(^4\). There have been reported cases in Taiwan\(^5,6\). The precise etiology of this syndrome is not clear but most likely it is a non-ischemic, metabolic syndrome caused by stress-induced activation of the cardiac adrenoceptors in the absence of ischemia and reperfusion\(^7\). Its clinical manifestations include acute onset of ischemic-like chest pain or dyspnea, characteristic transient apical and mid-ventricular regional wall-motion abnormality, minor elevations of cardiac enzyme and biomarker levels, and electrocardiographic ST-segment changes with QT-interval prolongation and evolutionary T-wave inversions. Diagnostic criteria proposed by the Mayo Clinic\(^2\) include (1) transient akinesis or dyskinesis of the left ventricular apical and mid-ventricular segments with regional wall motion abnormalities extending beyond a single epicardial vascular distribution; (2) absence of obstructive coronary disease or angiographic evidence of acute plaque rupture; (3) new electrocardiographic abnormalities (either ST-segment elevation or T-wave inversion) and (4) absence of recent significant head trauma, intracranial bleeding, pheochromocytoma, obstructive epicardial coronary artery disease, myocarditis, and hypertrophic cardiomyopathy. All four criteria must be met. The patient reported in this document manifested all of the diagnostic criteria for TLVABS. The overall prognosis seems to be favorable. The most common

Fig. 2 Left ventriculographic findings show the end systolic (A) and end diastolic phases (B). Apical ballooning is shown in (A) (arrow). Coronary angiograms of the (C) left coronary artery and (D) right coronary artery reveal no obvious stenosis.
reported clinical complication is left–sided heart failure, which may require aggressive diuresis, inotropic medication, and hemodynamic support. The case reported here is a rare case of TLVABS presenting with VT in a man.

**Gender**

As already mentioned, most cases occur in women (93.5%)\(^8\). The reason for female predominance is still not known. Estrogen may play a role, but the role of menopause and hormone replacement therapy is unknown in apical ballooning. There is no difference in average age for men and women\(^8\). Also, there is no difference between genders in regard to preceding stress triggering their cases\(^8\).

**Preceding stress**

Most cases of apical ballooning syndrome are preceded by a stressful event, either emotional or physical. In around 23% patients, there is no triggering event and the patients present spontaneously\(^8\).

**Causes**

Catecholamines seem to play a role in the syndrome. Patients presenting with this syndrome appear to have abnormalities of cardiac sympathetic innervation with evidence of sympathetic hyperactivity at the cardiac apex\(^9,10\). The distribution of apical wall motion abnormalities in transient left ventricular apical ballooning syndrome is similar to the distribution reported with catecholamine-induced cardiomyopathy\(^10\). Some investigators have suggested that transient left ventricular apical ballooning syndrome may be a result of catecholamine-associated stunning of the myocardium, which is provoked by emotional or physiologic stress\(^4,12,13\). So, it has also been called “the broken heart syndrome”. However, measurements of circulating catecholamine levels in patients with the syndrome have shown inconsistent results\(^14,15\). The cause of apical ballooning syndrome remains unknown. It is unknown if catecholamines cause the syndrome or increase as a result of the syndrome.

**Complications**

Overall, complications occurred in about 19% of patients in one study\(^8\). These complications included left-sided heart failure with and without pulmonary edema, cardiogenic shock, dynamic intraventricular obstruction with left ventricular intracavitary pressure gradient generation, mitral regurgitation resulting from chordal tethering as well as systolic anterior motion of the mitral valve apparatus, ventricular arrhythmia, left ventricular mural thrombus formation, left ventricular free wall rupture, and death. T-wave inversion and a physical stress trigger were the only two variables found to increase the likelihood of complications. Gender and race did not affect the complication rate. Those who died were on average older than those who survived\(^8\). Our patient came to our OPD department with VT. We don’t know whether the episode of VT in this patient was the preceding factor or a complication.

**References**


暫時性左心室心尖部氣球變形症候群—病例報告

張哲明1,2,3 徐國基2,3,4

暫時性左心室心尖部氣球變形症候群，是一種與急性心肌梗塞表現類似的少見症候群。它的表現包括類似缺血性胸痛、心電圖ST段升高、心肌酶素提升及特別的心室中間及心尖部收縮異常，但並無冠狀動脈狹窄。我們描述一位暫時性左心室心尖氣球變形症候群的59歲男性，因胸痛且心室頻脈而至急診。急診醫師以電擊成功治療之。隨後心導管檢查發現心室中間及心尖部收縮異常，但並無冠狀動脈狹窄的現象，因而診斷為暫時性左心室心尖氣球變形症候群。暫時性左心室心尖氣球變形症候群應該列入急性胸痛的鑑別診斷。

關鍵詞：ST段升高心肌梗塞，章魚變心肌變病，暫時性左心室心尖部氣球變形症候群

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1台北醫學大學醫學系 2新光吳火輝紀念醫院心臟內科 3輔仁大學醫學系 4台北醫學大學臨床醫學研究所
通訊及抽印本索取：徐國基醫師 台北市士林區文昌路95號 新光吳火輝紀念醫院心臟內科
電話：(02)28332211 傳真：(02)28123397
E-mail: liutp@ms1.mnh.org.tw