

# Takotsubo Syndrome and Acute Coronary Syndrome: Case Report and Literature Review

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## Abstract

Takotsubo syndrome (TS) has been described as an acute cardiac condition precipitated by a stressful mental or physical condition. A majority of patients are presented with acute substernal chest pain. It is often difficult to differentiate TS from acute ST-elevation myocardial infarction (STEMI) on initial presentation. The electrocardiograph classically mimics STEMI and is generally accompanied by elevation of myocardial enzymes that warrants immediate cardiac catheterization. The present article reports a patient with TS referred to the emergency department because of symptoms suggestive of acute coronary syndrome. (*JAEM 2015; 14: 41-3*)

**Key words:** Takotsubo, cardiomyopathy, stress, acute coronary syndrome, emergency department

## Introduction

Takotsubo syndrome (TS) is also called stress-induced cardiomyopathy or the broken heart syndrome. The main characteristic feature of Takotsubo cardiomyopathy (also known as "apical ballooning syndrome") is a transient systolic dysfunction of apical and/or mid segments of the left ventricle in the absence of occlusive coronary artery disease (1). The entity is also noted to masquerade as myocardial infarction.

Takotsubo syndrome is an acute cardiac condition which is precipitated by a stressful mental or physical condition. It affects aging women generally with a favorable prognosis. A majority of patients are presented with acute substernal chest pain. TS may lead to heart failure, pulmonary edema, tachyarrhythmias (including ventricular tachycardia and ventricular fibrillation), bradyarrhythmias, mitral regurgitation, and cardiogenic shock (2). The present article reports a patient with TS referred to the emergency department because of symptoms suggestive of acute coronary syndrome (ACS).

## Case Presentation

A 43-year-old male presented with awakening substernal chest pain accompanied by sweating and nausea. The respiratory rate was 19 breaths per minute, body temperature was measured as 37.3°C,

blood pressure was 135/85 mmHg, pulse rate was 74 beats per minute, and the oxygen saturation was 100%. Physical examination revealed an apical grade 2/6 systolic murmur on auscultation. Jugular venous distention and pretibial edema were not remarkable, and auscultation revealed clear lungs. The initial electrocardiograph (ECG) revealed normal sinus rhythm with right bundle branch block (RBBB) and ST-segment elevations in leads DI-aVL (Figure 1).

Complete blood count, blood glucose, and values from kidney and liver function tests were found to be within the normal limits. Two sets of myocardial enzyme assays showed an insignificant increase in creatine phosphokinase from 84 U/L to 121 U/L (normal range <190 U/L), and in troponin T from 2.16 g/L to 2.79 g/L (normal range <1 g/L) over 1 h. There was no dynamic ECG change during the follow up.

Because of the ST elevation in leads DI and aVL with reciprocal changes in the ECG and typical chest pain, the patient was assumed to have an ACS and this syndrome was consulted with the cardiology department. Despite the coronary angiography with normal coronary vessels, ventriculography revealed apical ballooning and akinesis in left ventricle (Figure 2).

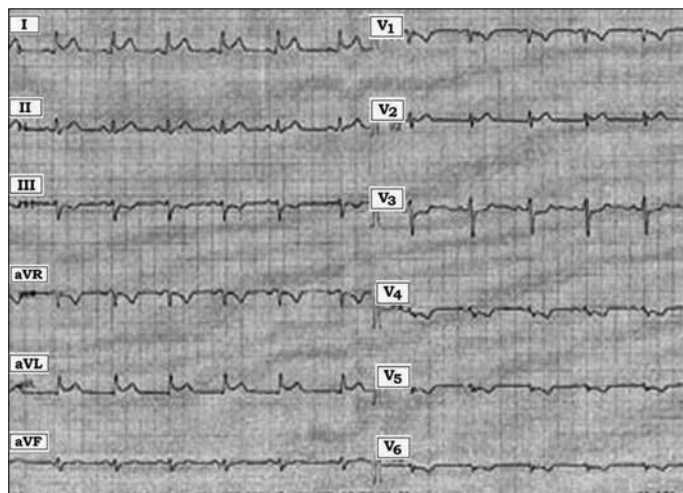
Transthoracic echocardiography revealed an akinesia of apico-mid-left ventricle (LV) free wall with LV dilatation (LV ejection fraction, 45%). The patient's clinical course was clinically and hemodynamically stable during his 4-day stay in the hospital.

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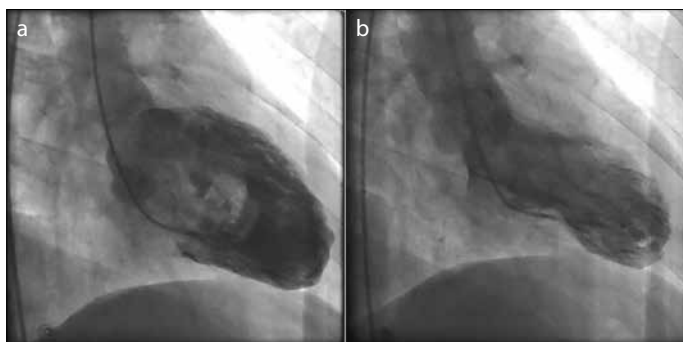
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**Figure 1.** Patient's ECG at admission  
ECG: electrocardiograph



**Figure 2. a, b.** Apical ballooning and akinesis in the left ventricle

The patient was discharged with medications including aspirin, ACE inhibitor,  $\beta$ -blocker, and a statin. The repeat echocardiogram performed 1 month later showed an ejection fraction of 50%-55% without any wall motional abnormalities.

## Discussion

The clinical symptoms and signs of TS resemble those of an ACS. Although the most common presenting symptom is acute chest pain, some patients may still present with dyspnea, syncope, shock, or ECG abnormalities. ST-segment elevation has been reported as 34%-56% in a systematic review (1). ST-segment elevation is commonly seen in anterior precordial leads. Deep T-wave inversion with QT interval prolongation, abnormal Q waves, and non-specific abnormalities are the other common abnormalities that may be seen in the ECG (2). Previously detected or new-onset left bundle branch block (LBBB) is associated with an adverse outcome in patients with acute myocardial infarction. LBBB was recorded on presentation in 9% of TS patients (3).

In TS, cardiac enzyme biomarkers are high; however, the elevations are typically mild, which may lead to the misdiagnosis of ACS. In a systematic review, it was reported to be elevated in 74%-86% of patients (4). In a subsequent series of 136 patients, troponin T levels ranged from 0.01 to 5.2 ng/mL (5).

Takotsubo syndrome patients are generally misdiagnosed as ACS; thus, they underwent primary percutaneous coronary interven-

tion (PCI). However, PCI is still mandatory to exclude ACS. Coronary angiography images reveal either normal vessels or mild-to-moderate coronary atherosclerosis. In risk population, obstructive coronary artery disease may be observed in its normal prevalence (6). The incidence of this reversible cardiomyopathy is estimated to be 1%-2% of patients presenting with an acute myocardial infarction (AMI). AMI with spontaneous clot lysis may mimic TS (7, 8).

Parodi et al. (9) noted that, nearly one-tenth of patients had at least one relevant ( $\geq 50\%$ ) coronary stenosis without supplying the dysfunctional myocardium, whereas nearly 90% patients had irrelevant stenosis or angiographically normal coronary arteries. Thus, when the stenotic artery does not supply the involved (dysfunctional) myocardium or when the extent of dysfunctional myocardium is larger than the area supplied by a single stenotic coronary artery, the presence of angiographically relevant coronary artery disease should be accepted as an exclusion criterion for TS.

Takotsubo syndrome patients showed significantly higher growth differentiation factor-15 (a stress responsive cytokine) values on admission compared with that of patients presenting with AMI (10). MicroRNAs (miRNAs) emerge as promising sensitive and specific biomarkers for cardiovascular disease. Jaguszewski et al. (11) have described four circulating miRNAs as a robust biomarker to distinguish TS from STEMI patients. They stated that the upregulation of the stress- and depression-related miRNAs is an indicator of the relation between TS and neuropsychiatric disorders.

Using the current guideline-endorsed viability criteria for semi-quantitative cardiac single photon emission computed tomography (SPECT) and fluorodeoxyglucose positron emission tomography (PET), these modalities failed to demonstrate the presence of viability in the acute state of TS (12).

With its superior soft tissue resolution and dynamic imaging capabilities, it is possible that cardiac magnetic resonance is currently the most useful imaging technique in diagnosing TS because the apical ballooning or medio-basal wall motion abnormalities, presence of wall edema, and late gadolinium enhancement are characteristic features in diagnosing this syndrome (13).

## Conclusion

Takotsubo syndrome is a potential life-threatening disease, which can hardly be clinically distinguished from ACS. There is no current established biomarker imaging techniques that are available for differentiating TS from AMI.

**Informed Consent:** Written informed consent was obtained from patient who participated in this case.

**Peer review:** Externally peer-reviewed.

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