

# A case of acute myocardial infarction mimicking Tako-Tsubo cardiomyopathy: the role of cardiac magnetic resonance imaging

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## SUMMARY

Tako-Tsubo cardiomyopathy is a unique reversible cardiomyopathy which is frequently precipitated by a stressful event and has a clinical presentation that is indistinguishable from a myocardial infarction. Clinical presentation of most patients including ECG and symptoms are equal to acute coronary syndromes. Cardiac magnetic resonance imaging may provide insight into potential pathophysiological mechanisms in Tako-Tsubo cardiomyopathy by assessment of myocardial viability. In this paper, we report a 49-year-old woman presenting with acute anterior myocardial infarction in whom apical ballooning was found and differential diagnosis was achieved by cardiac magnetic resonance imaging.

**Key words:** Acute myocardial infarction, magnetic resonance imaging, Tako-Tsubo cardiomyopathy

## ÖZET

**Tako-Tsubo kardiyomiyopatisini taklit eden bir akut miyokard infarktüsü olgusu: kardiyak manyetik rezonans görüntülemenin rolü**

Tako-Tsubo kardiyomiyopatisi, sıklıkla stresli bir olay ile başlayan, geri dönüşlü, nadir bir kardiyomiyopatidir ve miyokard infarktüsünden ayırt edilmesi zor olan bir kliniğe sahip olabilir. Birçok hastanın elektrokardiyografi ve semptomları içeren kliniği, akut koroner sendromlar ile benzerdir. Kardiyak manyetik rezonans görüntüleme, miyokardiyal canlılığı belirleyerek Tako-Tsubo kardiyomiyopatisinde potansiyel patofizyolojik mekanizmaları anlamamızı sağlar. Bu yayında, apikal balonlaşma tespit edilen ve kardiyak manyetik rezonans görüntüleme ile ayırıcı tanıya gidilen, akut anterior miyokard infarktüsü kliniği olan 49 yaşında bayan bir hasta sunulmuştur.

**Anahtar kelimeler:** Akut miyokard infarktüsü, manyetik rezonans görüntüleme, Tako-Tsubo kardiyomiyopatisi

## Introduction

The incidence of Tako-Tsubo cardiomyopathy (TTC) is approximately 1% to 2% of the patients presenting with a acute coronary syndrome (ACS) (1). TTC is a unique reversible cardiomyopathy which is frequently precipitated by a stressful event and has a clinical presentation that is indistinguishable from a myocardial infarction (MI). Up to now there has been no consensus on the diagnostic criteria for it. Clinical presentation of most patients including ECG and symptoms are equal with ACS. Cardiac magnetic resonance imaging (CMRI) may provide insight into potential pathophysiological mechanisms in TTC by assessment of myocardial viability. There are limited reports of CMR findings in patients with TTC (2).

Herein we report a 49-year-old woman presenting with acute anterior MI in whom apical ballooning was found and differential diagnosis was achieved by CMRI.

## Case Report

A 49-year-old woman with acute anterior MI was referred to our department for further evaluation after initiation of the first line therapy with fibrinolytics. After fibrinolytic infusion, she had been intubated due to ventricular tachycardia (VT) and cardiac arrest. She had been extubated 12 hours later and transferred to our hospital. On hospital admission 12-lead surface ECG demonstrated 2 mm ST segment elevation in leads V3-V6 accompanied with precordial poor R wave progression and diffuse symmetric T wave inversions. Her previous cardiac history was unrevealing. Except for a 2/6 grade apical systolic murmur, her cardiac examination was normal and lung fields were clear. Her blood pressure was measured as 110/60 mmHg with an aneroid sphygmomanometer and her pulse rate was 92 bpm. On bedside echocardiographic examination, severe hypokinesis of the anterior wall sparing basal segment with an ejection fraction of 40% and

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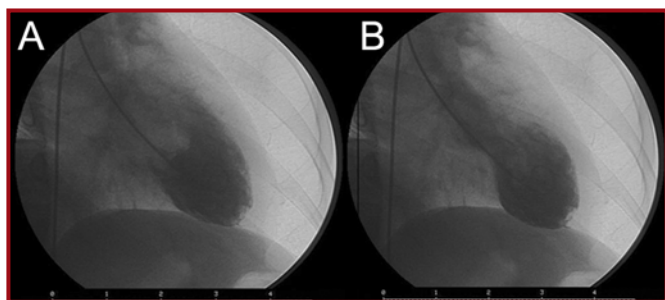
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thrombus formation on the apex were detected. On admission creatinine kinase (CK) was 425 U/L with an MB fraction of 122 U/L. Her initial examination revealed a left sided central facial paralysis and motor aphasia with National Institute of Health Stroke Scale score of 3 points was found on neurological examination. Cerebral magnetic resonance images revealed an acute cerebral ischemia in the right frontal cortex. As the patient was right handed, she had crossed aphasia. Subsequent diagnostic coronary angiography revealed tortuous and normal coronary artery anatomy. Left ventriculogram showed hyperdynamic basal contraction but hypokinesis of the midsegment and akinesis of the apical segments compatible with classic TTC (Figure 1). In CMRI late gadolinium enhancement demonstrated full thickness late myocardial enhancement in the apical area which was in consistent with infarcted myocardium with microvascular obstruction foci (black arrows) and thrombus (white arrow) in the left ventricular cavity (Figure 2). In-hospital course of the patient was uneventful and she was discharged from the hospital 5 days later with medical therapy including warfarin.

## Discussion

TTC (apical ballooning syndrome, stress induced cardiomyopathy, ampulla cardiomyopathy and broken heart syndrome) was first described by Sato and coworkers in 1990 (1,3,4). TTC generally occurs in postmenopausal women and is indistinguishable from a MI. Chest pain and dyspnea are usually initial symptoms resembling acute MI, but syncope or cardiac arrest are also encountered (1,3). Approximately two thirds of the patients with TTC have an emotional or physical stressful event (1). Concordantly our patient, who was a prison guard, experienced sudden severe chest pain developing after a prisoner assault. ST-segment elevation, especially on precordial leads, is the most common abnormality on the ECG obtained from those patients. ST elevation in inferior or lateral leads, nonspecific T-wave abnormality, new bundle-branch block,

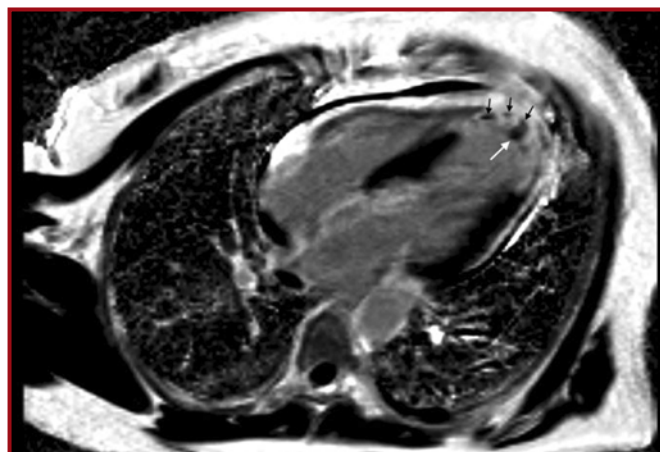


**Figure 1.** Left ventriculogram showing end-diastolic phase (A) and, end-systolic phases (B) compatible with classic Tako-Tsubo cardiomyopathy

and normal ECG may be seen on admission. The ECG findings are insufficient for differentiating TTC from ST elevation MI. However the magnitude of ST shift is usually less in TTC than that seen in a ST elevation MI (1,2,4). Resolution of the ST-segment elevation, development of diffuse and often deep T-wave inversion, and prolongation of the corrected QT interval may commonly occur over 3 days (1).

TTC syndrome is underrecognized and often misdiagnosed. It is an important differential diagnosis of an acute MI. The diagnostic criteria of the Mayo Clinic is as follow (1): (a) transient hypokinesis, akinesis, or dyskinesis in the left ventricular mid segments with or without apical involvement; regional wall motion abnormalities that extend beyond a single epicardial vascular distribution; and frequently, but not always, a stressful trigger; (b) the absence of obstructive coronary disease or angiographic evidence of acute plaque rupture; (c) new ECG abnormalities (ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin; and (d) the absence of pheochromocytoma and myocarditis. Japanese investigators have recently presented diagnostic guidelines; however, the modified Mayo criteria are commonly used. It is necessary to establish worldwide consensus on diagnostic criteria for TTC.

Tako-Tsubo contraction pattern may be encountered in patients with acute MI (1,4,6). Chao et al. reported that 14% of the patients with anterior ST elevation MI had a classic contraction pattern of TTC (4). Coronary angiography is the common tool to diagnose this condition, but angiography reveals only prominent atherosclerotic plaques and may not detect lipid-rich plaques that have undergone positive remodeling. On the other hand transient thrombosis



**Figure 2.** Late gadolinium enhancement image in four chamber view demonstrates full thickness late myocardial enhancement in the apical area which is in consistent with infarcted myocardium with microvascular obstruction foci (black arrows) and thrombus (white arrow) in the left ventricular cavity

or spasm of these invisible plaques in LAD may cause regional wall motion abnormalities as TTC (1,4).

Although the wall motion abnormalities could be detected by echocardiography, CMR reveals subendocardial or transmural MI via myocardial delayed enhancement even if coronary angiography does not detect an obstructive lesion. It was reported that CMR may be very helpful to differentiate TTC from MI (6). In addition, CMR is more helpful to show the involvement of the right ventricular dysfunction.

In conclusion, CMR appears to be a useful imaging modality for documenting the extent of the regional wall motion abnormality and differentiating TTC from MI and myocarditis in which delayed hyperenhancement is present. While TTC can mimic acute MI, the converse is also true, that acute MI can mimic TTC. Cardiac magnetic resonance may be very helpful to rule out TTC in this setting. The diagnosis of TTC can be easily excluded if CMR demonstrates myocardial late enhancement in consistent with myocardial necrosis in a pattern and distribution in consistent with acute MI.

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