



# Takotsubo cardiomyopathy in a patient diagnosed with postresuscitation syndrome

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### Abstract

Takotsubo cardiomyopathy (TC) is a reversible cardiomyopathy that has been known for approximately 35 years and is characterized by apical ballooning and has morphological features specific to the left ventricle. Because of its similarity to myocardial infarction, Takotsubo cardiomyopathy requires careful diagnosis and treatment for the best possible outcome. When we look at the pathophysiology of this disease, it is known that the increase in catecholamines that occurs with physical or emotional stress plays an important role. Patients in intensive care unit are at high emotional and physical risk due to intensive diagnosis and treatment protocols. Especially in patients admitted to intensive care after cardiopulmonary resuscitation, an increase in sympathetic stimulation and intravenous catecholamines due to resuscitation may cause TC disease. In this case, we aimed to describe the characteristics of TC that developed in a patient treated in intensive care after cardiac arrest, and to make its differential diagnosis and literature review.



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## Introduction

Takotsubo cardiomyopathy (TC), also known as transient apical ballooning syndrome or broken heart syndrome, is a rare pathology and is a cardiovascular syndrome characterized by left ventricular dysfunction without any coronary artery lesion [1]. The distinguishing feature of TC is the apical ballooning of the left ventricle, similar in appearance to the 'Takotsubo', a vessel used for octopus hunting in Japan. When we look at the pathophysiology of this disease, catecholamine increase plays an important role, therefore it is also known as stress cardiomyopathy [2].

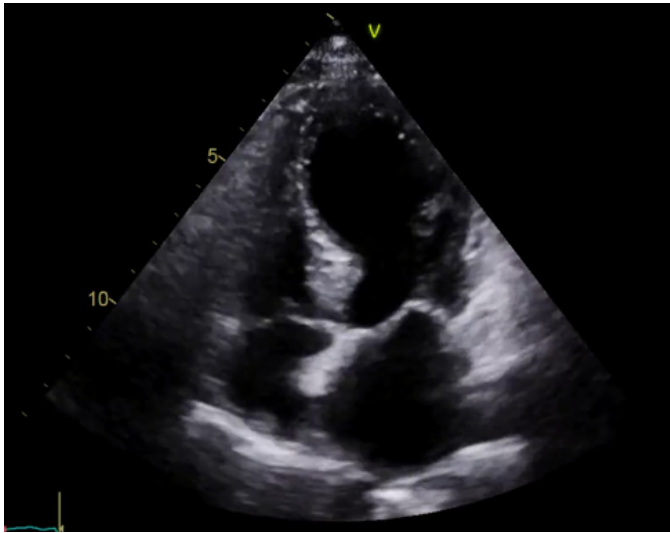
Alternatively, in patients diagnosed with postresuscitation syndrome; TC may occur after cardiopulmonary resuscitation (CPR) due to an increase in sympathetic stimulation and intravenous catecholamines. In our case, the management of TC, which was detected on the 3<sup>rd</sup> day of the patient's admission to the intensive care unit after cardiac arrest, is presented.

## Case Report

A 48-year-old male patient was brought to the emergency department of our hospital by 112 medical teams due to cardiac arrest at home. Cardiopulmonary resuscitation was applied for 60 minutes in the emergency department. The patient's heart rhythm was obtained as a result of CPR, and coronary angiography was performed with the preliminary diagnosis of acute coronary syndrome. During the catheterization procedure, no significant stenosis was detected in the coronary arteries. In echocardiography, ejection fraction (EF) was evaluated as 65% and left ventricular systolic functions and diameters were normal. After angiography, the patient was taken to the reanimation intensive care unit with the diagnosis of post-resuscitation syndrome. On the 3<sup>rd</sup> day of the patient's admission to intensive care unit, it was observed that the pro-BNP level increased from 7950 ng/L to 32500 ng/L. Following this significant increase, echocardiography was planned for the patient to investigate cardiac pathologies. Echocardiography revealed segmental wall motion abnormality, 30% ejection fraction, and anteroapical ballooning, which is a pathognomonic finding for TC (Figure 1). In addition to this determination, the patient was diagnosed with TC due to the presence of stress factors in the patient, an increase

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**Figure 1.** Antero-apical ballooning image in the LV on echocardiography.

in cardiac biomarkers, no stenosis or rupture detected in the angiography, and no evidence of myocarditis in the echocardiography.

The patient was given dopamine and levosimendan to treat the symptoms caused by left ventricular dysfunction. The patient underwent regular echocardiography and it was determined that cardiac functions improved daily. The patient's pro-BNP levels also decreased significantly day by day (14900ng/L-3270ng/L-2600ng/L). In the echocardiographic evaluation performed on the 6<sup>th</sup> day of the patient's admission to the intensive care unit, EF was evaluated as 60-65% and left ventricular systolic functions and diameters were normal. The patient was extubated on the 13<sup>th</sup> day of hospitalization after post-cardiac arrest care and continued to be monitored for spontaneous respiration. A signed consent form was obtained from the patient on 10/01/2023.

## Discussion

Throughout history, it has been thought that there is an interaction between stress and heart, and it has been believed that stress is effective in the development of heart diseases. With studies conducted in recent centuries, stress has been associated with heart diseases [3]. When we look at the risk factors of TC, we see that there are emotional or physical stress and genetic factors. As a result of stress, the response of the sympathetic system increases and this is associated with the emergence of TC [2].

Stress factors include the death of a relative, traffic accidents, disasters, war, anxiety, divorce, unexpected funerals, surgery and illnesses [3]. In our case, the patient's cardiac arrest, subsequent CPR application for 60 minutes, angiography with the diagnosis of acute coronary syndrome, and the subsequent intensive care period each constitute a risk factor for stress on their own.

When we look at the literature; it has been reported that there are cases of TC that developed after cesarean section, rhinoplasty and nasal fracture surgeries, and patients diagnosed with TC along with Sheehan syndrome, chronic

**Table 1.** Mayo clinic diagnostic criteria for Takotsubo cardiomyopathy (2008).

- |   |
|---|
| 1. Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always, present. |
| 2. Absence of obstructive coronary artery disease or angiographic evidence of acute plaque rupture.   |
| 3. New ECG abnormalities (either ST elevation and/or T wave inversion) or modest elevation in cardiac troponin.   |
| 4. Absence of other precipitants eg pheochromocytoma, myocarditis.  |

anxiety, postpartum depression, myasthenia gravis, pancreatitis and Addison's diseases [4]. In our case, we think that the patient was diagnosed with TC due to the stress that occurred after cardiac arrest.

TC diagnostic criteria are from the Mayo Clinic and consist of four components [2] (Table 1).

In our case, it was determined that the patient we presented was in compliance with the Mayo Clinic diagnostic criteria, as there was a stress factor, apical ballooning was seen in the echocardiography, there was an increase in cardiac biomarkers, no stenosis or rupture was detected in the angiography, there was no evidence of myocarditis in the echocardiography, and our patient was far from the pheochromocytoma clinic.

Although the cause, pathogenesis and pathophysiology of TC are still not known with certainty, many hypotheses have been associated with the emergence of this disease. Recently, the most accepted theories are catecholamine-induced cardiotoxicity and microvascular dysfunction [5].

TC shares common features with acute coronary syndrome in terms of presenting symptoms, ECG abnormalities, and elevated cardiac biomarkers [6]. Therefore, the diagnostic approach must be made meticulously in patients with these clinical findings. At this point, Brain Natriuretic Peptide (BNP) levels play an important role in distinguishing between ACS and TC. It has been found that B-type natriuretic peptide (BNP) and N-terminal pro-BNP (NT-proBNP) are frequently 3-4 times higher in patients diagnosed with TC compared to patients diagnosed with ACS [7]. Although our patient's high troponin and creatinine phosphokinase-MB levels are common with ACS, the fact that our patient had higher pro-BNP levels than ACS patients guided us in diagnosing TC.

Coronary angiography may also play a critical role in differentiating TC from ACS. Coronary angiography can more accurately prove normal coronary artery or minor atherosclerosis. Performing angiography before our patient was admitted to the intensive care unit and determining that he had normal coronary arteries also helped us in the TC diagnosis process. The pathognomonic finding of TC on echocardiography is apical ballooning in the left ventricle. This unique morphology has been reported to occur in 75% of patients [8]. In our case, anteroapical ballooning was detected in the echocardiography performed on the 3rd day of admission to the intensive care unit.

In addition to echocardiography examination, magnetic

resonance imaging (MRI) is an important investigation to provide more meaningful evidence of TC. MRI can show certain imaging features, such as right ventricular involvement, and distinguish it from other cardiomyopathies [9]. Since our patient was in the post-cardiac arrest care process, the patient was not left in the intensive care unit for MRI and the diagnosis process continued with the pathognomonic finding detected in echocardiography.

When we look at the treatment of TC, the main aim is to regress cardiac symptoms and correct left ventricular dysfunction [10]. For this purpose, dopamine and levosimendan were administered to our patient. And afterwards, it was observed that cardiac functions returned to normal.

In addition, anticoagulation therapy should be continued even after the diagnosis of TC is confirmed. This treatment is useful to prevent LV apical thrombosis and possible embolic events [11]. In our patient, anticoagulation treatment was continued for the risk of thromboembolism.

However, hemodynamically unstable patients may need cardiopulmonary support, continuous venovenous hemofiltration and intra-aortic balloon pump [12]. Since dramatic improvement was observed in our patient, these treatments were not needed.

When we look at the mortality of TC, the in-hospital mortality rate varies between 0-8% and the recurrence rate varies between 0-15% [13]. The prognosis of patients diagnosed with TC is excellent; the recovery rate is 96% [14]. Left ventricular function may begin to improve within a few days and may fully recover in 3-4 weeks [14]. In our patient, it was determined in the echocardiography evaluation that the left ventricular functions returned to normal on the 4<sup>th</sup> day.

However, although the wall motion defect is thought to be benign due to its temporary nature, it should be kept in mind that the mortality rate increases in the event of cardiogenic shock [15].

## Conclusion

TC is a temporary and reversible cardiomyopathy with a good prognosis. Due to its characteristics similar to acute coronary syndrome, careful diagnosis and treatment should be applied. The main treatment is supportive treatment.

Although the syndrome was described 35 years ago and is now diagnosed more frequently, it remains a mystery as its exact pathophysiology is unknown and there are no randomized controlled studies on it.

Using combinations of diagnostic methods such as ECG, cardiac biomarkers, echocardiography, coronary angiography and cardiac magnetic resonance imaging helps to make a more accurate diagnosis of TC.

In order not to miss the diagnosis, in patients who are urgently taken to the catheterization laboratory with a diagnosis of acute coronary syndrome and no serious stenosis is detected; Ventriculography or echocardiographic examination should be performed as soon as possible to rule out rare pathological conditions that may develop later in case of deterioration of cardiac functions.

## Conflict of interests

The authors declare that there is no conflict of interest in the study.

## Financial disclosure

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