ARCHIVES OF THE TURKISH SOCIETY OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

# Takotsubo Cardiomyopathy Mimicking Obstructive Hypertrophic Cardiomyopathy

## Obstrüktif Hipertrofik Kardiyomiyopatiyi Taklit Eden Takotsubo Kardiyomiyopatisi

**ABSTRACT**

Takotsubo cardiomyopathy (TCM) is characterized by transient left ventricular dysfunction, diagnosed via echocardiography or left ventriculography. In most cases, TCM involves an emotional, physical, or combined trigger. Acute coronary syndrome is one of the most frequent misdiagnoses in TCM patients due to electrocardiogram (ECG) abnormalities and elevated cardiac biomarkers. Typically, coronary angiography reveals no stenosis or occlusion of the coronary arteries. Hypertrophic cardiomyopathy (HCM) is a distinct pathology characterized by a hypertrophied left ventricle with various phenotypes. However, some reports have described TCM cases mimicking obstructive-type HCM in some patients. We present a case of a female patient diagnosed with TCM based on clinical, laboratory, and imaging tests. Differentiating TCM from HCM was challenging due to ventriculography and echocardiography findings, as hyperdynamic contraction of the basal segments of the left ventricle caused an increased left ventricular outflow tract (LVOT) gradient and severe mitral valve regurgitation. Detailed evaluation and close echocardiographic follow-up are essential in such rare cases.

**Keywords:** Left ventricular outflow obstruction, mitral regurgitation, Takotsubo cardiomyopathy

**ÖZET**

Takotsubo kardiyomiyopatisi (TKM), ekokardiyografi veya sol ventrikülografi ile teşhis edilebilen geçici sol ventriküler fonksiyon bozukluğu ile karakterizedir. Çoğu durumda duygusal, fiziksel veya birleşik bir tetikleyici vardır. Akut koroner sendrom, TKM hastalarında EKG anormallikleri ve kardiyak biyobelirteçlerin yükselmesiyle ilişkili olarak en sık konulan yanlış tanılardan biridir. Koroner anjiyografide genellikle koroner arterde darlık veya tıkanıklık görülmez. Hipertrofik kardiyomiyopati (HKM), çeşitli fenotiplere sahip hipertrofik sol ventrikül ile karakterize tamamen farklı bir patolojidir. Ancak birkaç yazıda TKM'nin bazı hastalarda obstrüktif tip HKM'yi taklit ettiği de bildirildi. Burada klinik, laboratuvar ve görüntüleme testlerine göre TKM tanısı konulan bir kadın hastayı sunduk. Bununla birlikte, sol ventrikülografi ve ekokardiyografi bulguları, sol ventrikülün bazal segmentlerinin hiperdinamik kasılmasının, sol ventriküler çıkış yolu (SVÇY) gradyanında artışa ve ciddi mitral kapak yetersizliğine yol açması nedeniyle, TKM'yi HKM'den ayırt etmekte zorlayıcıydı. Bu kadar nadir görülen bir olguda TKM'nin HKM'den ayrımının yapılabilmesi için detaylı değerlendirme ve ekokardiyografi ile yakın takip gerekmektedir.

**Anahtar Kelimeler:** Sol ventriküler çıkış yolu tıkanıklığı, mitral yetersizlik, Takotsubo kardiyomiyopatisi

**CASE REPORT**
OLGU SUNUMU

Takotsubo cardiomyopathy (TCM), or stress-induced cardiomyopathy, is an acute, temporary condition predominantly presenting with chest pain, dyspnea, and syncope. The majority of cases occur in postmenopausal women following a physical, emotional, or combined trigger. Elevated cardiac biomarkers and various electrocardiogram (ECG) abnormalities are common, often leading to confusion with acute coronary syndrome in clinical practice. However, coronary angiography generally shows no significant coronary artery stenosis or occlusion. Left ventricular (LV) dysfunction is readily identifiable on transthoracic echocardiography or left ventriculogram. The apical ballooning subtype is the most common form of TCM. Despite its dramatic clinical presentation and significant risk of heart failure, cardiogenic shock, and arrhythmias, the long-term prognosis is generally favorable. In most patients, LV function improves over time.[1] The literature includes a few reported cases that highlight the difficulty in differentiating TCM from obstructive type hypertrophic cardiomyopathy (HCM).[2-6] We report the case of a postmenopausal woman who developed TCM mimicking obstructive type HCM following an emotional trigger.

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**Received:** August 31, 2023
**Accepted:** October 26, 2023

**Cite this article as:** Zekeriyeyev S, Canpolat U. Takotsubo cardiomyopathy mimicking obstructive hypertrophic cardiomyopathy. *Turk Kardiyol Dern Ars.* 2024;52(6):455-459.

DOI:10.5543/tkda.2023.93429

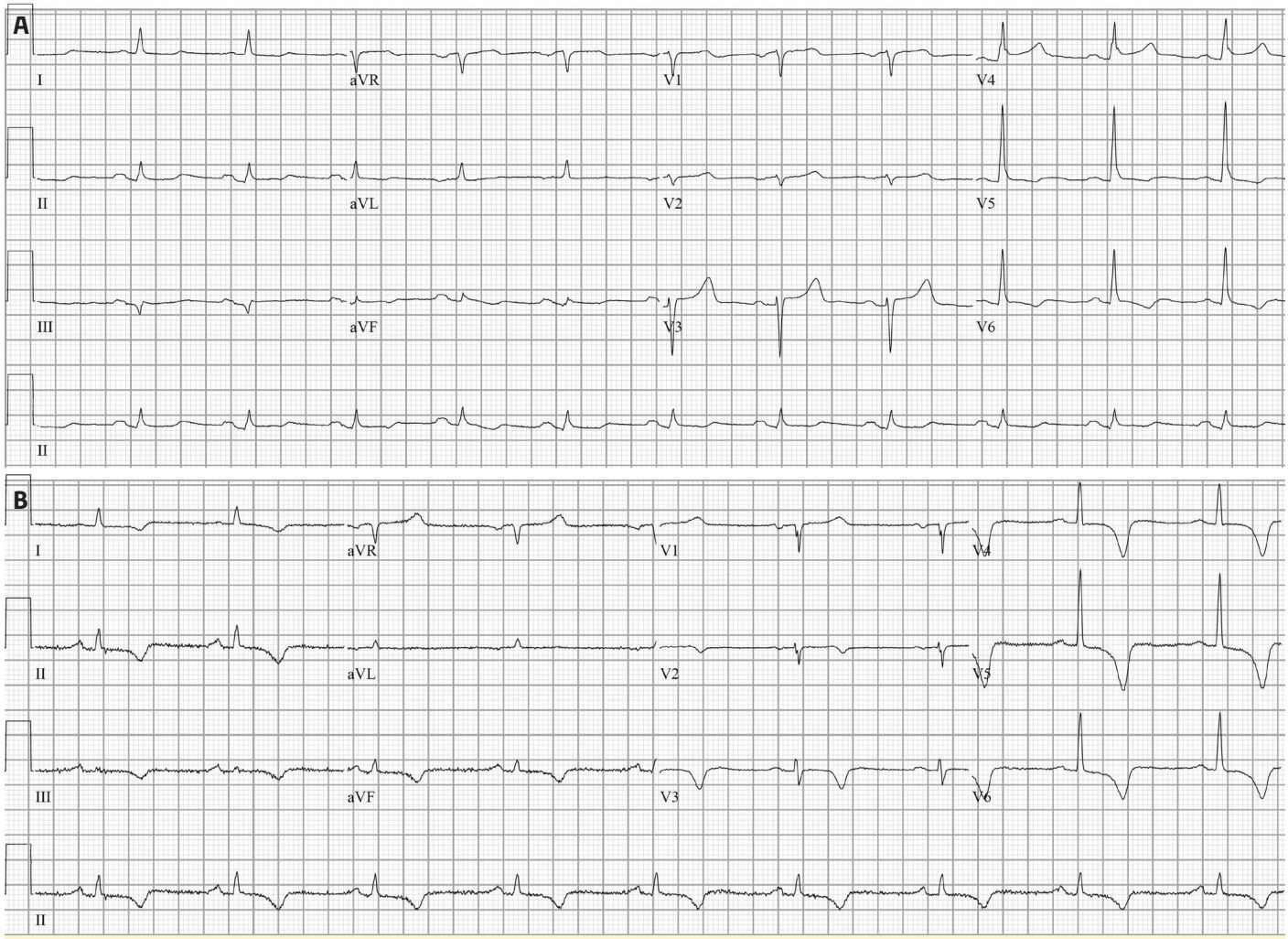


**Figure 1. (A) 12-lead electrocardiogram (ECG) recording at presentation showing ST-segment depression and T-wave inversion in precordial leads V5 & V6. (B) Serial 12-lead ECG recording depicting the progression from ST-segment depression to symmetrical T-wave inversion across all extremity and precordial leads.**

**Case Report**

A 67-year-old postmenopausal woman with a history of hypertension presented to the emergency department with complaints of epigastric pain and sweating that had lasted for 30 minutes. She was hemodynamically stable. Physical examination was unremarkable except for a severe holo-systolic murmur at the apex. The 12-lead electrocardiography on admission showed sinus rhythm with non-specific ST-segment and T-wave changes in the extremity and precordial derivations (Figure 1A). Serial ECGs revealed prominent and generalized T-wave inversions (Figure 1B). The initial troponin I level was significantly elevated at 1279.2 ng/L, which was above the 99th percentile upper reference limit. Bedside echocardiography of low image quality indicated a left ventricular ejection fraction of 40% and severe hypokinesia of the anterior mid/apical septum and apex. Non-ST-segment elevation myocardial infarction (NSTEMI) was initially considered in the differential diagnosis, and an early invasive approach was implemented due to persistent symptoms. Coronary angiography showed no evidence of obstructive or occlusive lesions in the coronary arteries. Given the discrepancy between the clinical presentation, laboratory data, and coronary angiography findings, we decided to perform left ventriculography to rule out TCM. The ventriculography revealed akinesia and ballooning of the apical segments, while the wall motion of the basal segments was preserved (Figure 2A-B, Video 1). Furthermore, there was an enlarged left atrium and severe mitral valve regurgitation. Detailed transthoracic echocardiography confirmed a left ventricular ejection fraction of 40%, an interventricular septum thickness of 12 mm, akinesia, ballooning of the apical segments

## ABBREVIATIONS

| | |
|---|---|
| ACS | Acute coronary syndrome |
| ECG | Electrocardiogram |
| HCM | Hypertrophic cardiomyopathy |
| LGE | Late gadolinium enhancement |
| LV | Left ventricular |
| LVOT | Left ventricular outflow tract |
| MRI | Magnetic resonance imaging |
| SAM | Systolic anterior motion |
| TCM | Takotsubo cardiomyopathy |

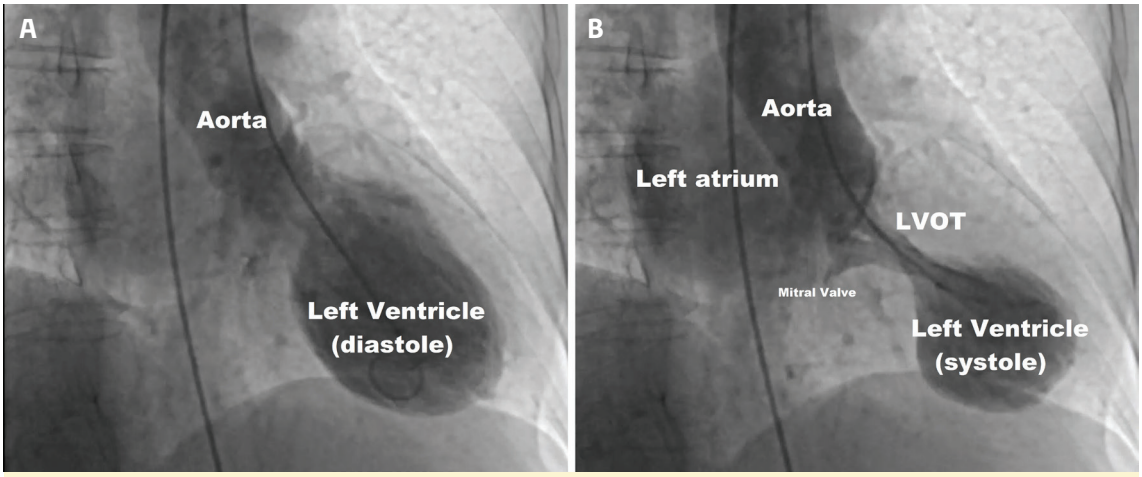


**Figure 2. Ventriculography in the right anterior oblique view displaying left ventricular wall motion abnormalities. (A) End-diastole, (B) End-systole, showing apical ballooning, severe mitral regurgitation, and left atrial enlargement.**

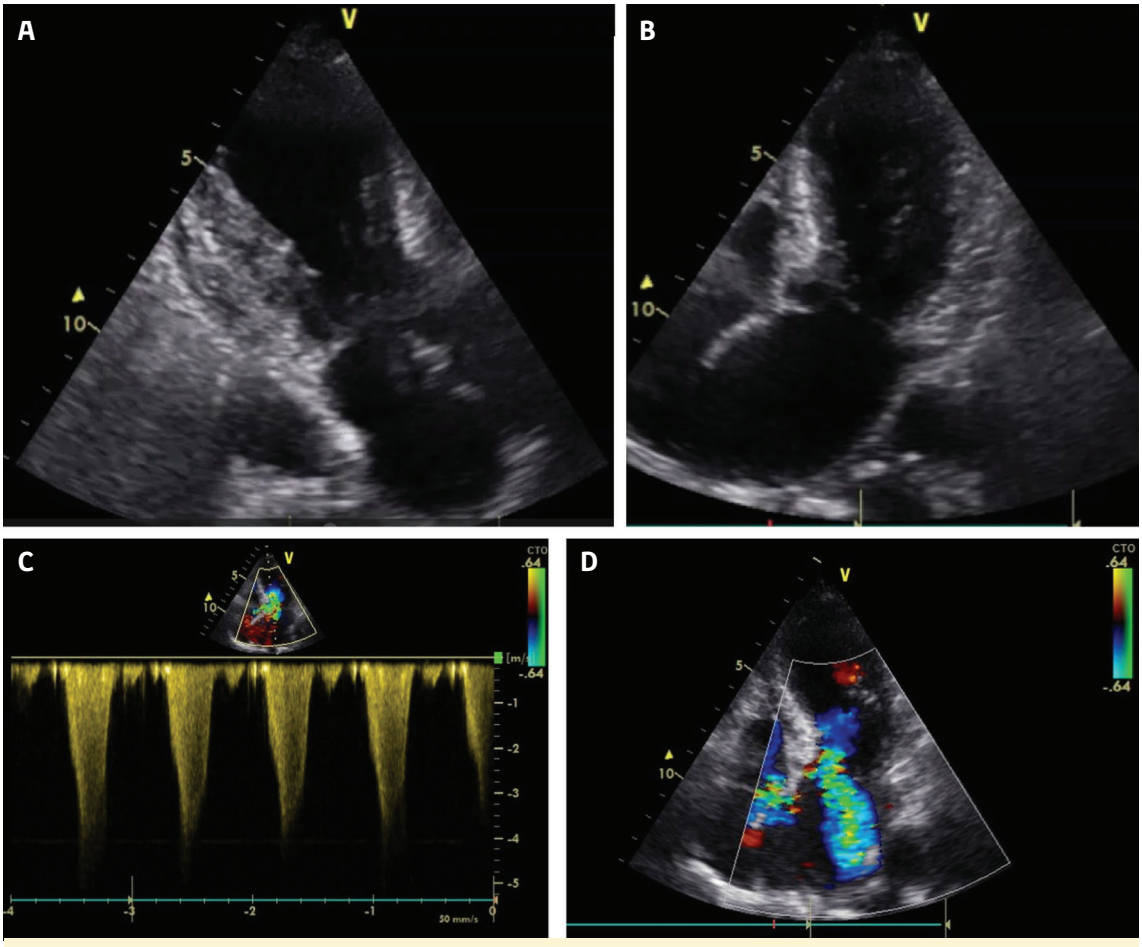


**Figure 3. Two-dimensional echocardiography at presentation. Observe the clear left ventricular outflow tract obstruction with an approximately 75 mmHg gradient (A-C) and severe mitral insufficiency (D).**

of the left ventricle, and hyperkinesia of the basal segments of the left ventricle. This hyperkinesia caused obvious systolic anterior motion (SAM) of the mitral valve and left ventricular outflow tract (LVOT) obstruction (Video 2). The resting outflow gradient measured by echocardiography was approximately 75 mmHg (Figure 3A-C). Other findings included minimal prolapse of the anterior mitral valve leaflet, thickened mitral valve leaflets/subvalvular apparatus, severe mitral regurgitation,

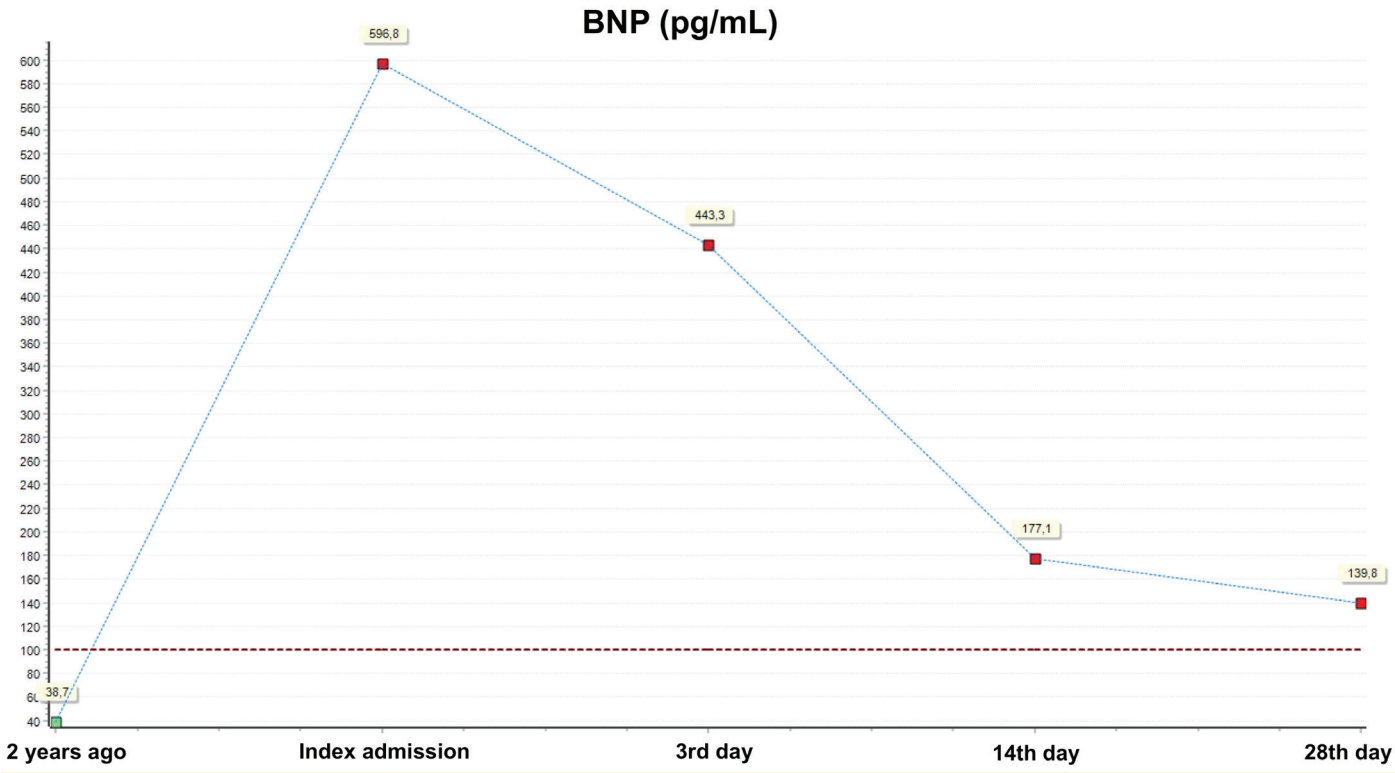


**Figure 4. The brain natriuretic peptide (BNP) values of the patient over time.**

and left atrial enlargement (Figure 3D). Thus, stress-induced cardiomyopathy, or TCM mimicking obstructive type HCM, was considered the most probable diagnosis. A detailed history taken from the patient in the coronary care unit confirmed an emotional trigger as a risk factor for TCM (the recent death of her brother). Medications of Carvedilol 6.25 mg twice daily, Ramipril 10 mg once daily, and Spironolactone 25 mg once daily were initiated during the index hospitalization. The in-hospital course was uneventful, and she was discharged with the same treatment regimen. Control transthoracic echocardiography at the 4th-week visit showed complete improvement of LV systolic function, absence of SAM of the mitral valve, no LVOT gradient, and moderate-to-severe mitral regurgitation, with estimated systolic pulmonary artery pressure of 40 mmHg (Video 3). Control cardiac magnetic resonance imaging (MRI) at the 4th-week visit confirmed normal LV systolic function, no late gadolinium enhancement, interventricular thickness of 12 mm, mitral valve prolapse, moderate-to-severe mitral regurgitation, and no LVOT obstruction (Video 4). The brain natriuretic peptide (BNP) level at index hospitalization was 596.8 pg/mL, which significantly decreased to 139.8 pg/mL by the fourth week after optimal medical treatment (Figure 4). Given the patient was clinically asymptomatic under guideline-directed medication, a close clinical follow-up was planned for the mitral regurgitation.

## Discussion

Our case involved a patient presenting with epigastric pain, where initial 12-lead ECG and echocardiography abnormalities, along with elevated cardiac biomarker levels, suggested a potential diagnosis of acute coronary syndrome. However, coronary angiography revealed no obstructive or occlusive lesions, aligning with a diagnosis of TCM. Echocardiography showed akinesia and ballooning of the apex, hyperkinesia of the basal segments, subsequent LVOT obstruction, severe mitral regurgitation, and left atrial enlargement. The presence of an emotional trigger in her history supported the diagnosis of stress-induced cardiomyopathy.

Although the exact cause, pathogenesis, and pathophysiology of TCM remain uncertain, the predominant hypothesis suggests that exaggerated sympathetic stimulation leads to a catecholamine surge, causing regional microvascular dysfunction in susceptible patients. This is often accompanied by cellular calcium overload and dysfunction.[7,8] Consequently, patients with TCM often exhibit clinical features similar to those with acute coronary syndrome (ACS), including acute chest pain and/or dyspnea, ECG alterations, and slight elevations in cardiac biomarkers such as troponins, along with increases in BNP and left ventricular dysfunction.[9,10] Differentiating between TCM and obstructive HCM with apical ballooning is crucial due to significant overlap in clinical presentations. The occurrence of LVOT obstruction in TCM has been reported with a prevalence of 25%.[11] In TCM, transient and dynamic intraventricular pressure gradients can arise from dyskinesia of the apical and mid segments and hyperkinesia of the basal segments of the left ventricle, leading to LVOT obstruction.[5] Geometric factors, including a sigmoid interventricular septum, reduced LV volumes, a small LVOT, and abnormal orientation of the mitral apparatus, predispose to systolic anterior motion and dynamic LVOT obstruction.[12] During the acute phase of TCM, myocardial edema can cause myocardial wall swelling and an increase in left ventricular wall thickness.[6] These factors contribute to the

potential oversight and misdiagnosis of TCM as obstructive HCM with apical aneurysm. Key distinguishing characteristics include the presence of LVOT obstruction, mitral valve pathology, lack of resolution in LV wall thickness, and persistent echocardiographic features of HCM beyond the acute phase.[3,4] This highlights the importance of regular echocardiographic follow-ups for these patients. Additionally, cardiac MRI criteria for the acute phase of TCM have been established, featuring typical regional wall motion abnormalities, edema, and the absence of irreversible tissue injury [late gadolinium enhancement (LGE)]. LGE is usually absent and predicts the normalization of LV systolic functions. The absence of LGE is also valuable in differentiating from acute coronary syndrome and many cases of acute myocarditis.[13,14] Cardiac MRI proved beneficial during the subacute phase of TCM. We performed the cardiac MRI during the fourth-week visit for our patient, which revealed no LGE and improved LV systolic functions, thereby confirming the initial diagnosis of TCM. As an important biomarker in heart failure diagnosis and prognosis,[15] plasma levels of BNP typically rise within 24-48 hours of symptom onset and normalize over a few months. The level of plasma BNP is correlated with the severity of sympathetic overactivation, LV systolic dysfunction, and inflammation.[14] Plasma levels of BNP were elevated in our patient upon admission and significantly decreased following the normalization of LV systolic functions.

In line with our case, subsequent follow-up assessments showed notable improvements in left ventricular function, disappearance of the SAM of the mitral valve leaflet, and LVOT gradient, while mild to moderate mitral regurgitation persisted.

## Conclusion

In conclusion, this case highlights the diagnostic challenges posed by TCM, particularly in distinguishing it from obstructive HCM. The clinical presentation, echocardiographic findings, and patient history all contributed to the accurate diagnosis of stress-induced cardiomyopathy in this case. The favorable outcome, facilitated by appropriate medical management, further underscores the transient and reversible nature of TCM. Clinicians should remain vigilant in considering TCM as a potential diagnosis, particularly when typical cardiac symptoms are accompanied by emotional triggers and atypical angiographic findings. Appropriate management, including medical therapy and vigilant clinical follow-up, is crucial for optimizing patient outcomes.

**Informed Consent:** Written informed consent was obtained from the patient.

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

**Author Contributions:** Concept – U.C., S.Z.; Design – U.C., S.Z.; Supervision – U.C.; Materials – U.C.; Data Collection and/or Processing – U.C., S.Z.; Analysis and/or Interpretation – U.C., S.Z.; Literature Review – U.C., S.Z.; Writing – U.C., S.Z.; Critical Review – U.C., S.Z.

**Use of AI for Writing Assistance:** AI technology for writing assistance was not used during the preparation of the paper.

**Conflict of Interest:** No conflict of interest was declared by the authors.

**Funding:** The authors declared that this study received no financial support.

**Video 1**. Left ventriculography revealing akinesia and ballooning of the apical segments, while the basal segments' wall motion is preserved. Additionally, there is an enlarged left atrium and severe mitral valve regurgitation.

**Video 2**. Transthoracic echocardiography on admission displays a left ventricle ejection fraction of 40%, akinesia of the anterior mid/apical septum and apex, and hyperkinesia of the basal segments of the left ventricle, causing obvious systolic anterior motion (SAM) of the mitral valve and left ventricular outflow tract (LVOT) obstruction. An enlarged left atrium and severe mitral regurgitation are also noted.

**Video 3**. Control transthoracic echocardiography at the 4th-week visit shows complete improvement of the left ventricular (LV) systolic function, absence of SAM of the mitral valve, no LVOT gradient, and moderate-to-severe mitral regurgitation.

**Video 4**. Cardiac magnetic resonance imaging (MRI) at the 4th-week visit confirms normal LV systolic function, mitral valve prolapse, moderate-to-severe mitral regurgitation, and no LVOT obstruction.

## References

1. Ghadri JR, Wittstein IS, Prasad A, et al. International expert consensus document on Takotsubo syndrome (Part I): Clinical characteristics, diagnostic criteria, and pathophysiology. *Eur Heart J.* 2018;39(22):2032–2046. [CrossRef]
2. Benak A, Sramko M, Janek B, Zelizko M, Kautzner J. Successful treatment of cardiogenic shock due to Takotsubo cardiomyopathy with left ventricular outflow tract obstruction and acute mitral regurgitation by impella CP. *Cureus.* 2022;14(3):e23168. [CrossRef]
3. Daralammori Y, El Garhy M, Gayed MR, Farah A, Lauer B, Secknus MA. Hypertrophic obstructive cardiomyopathy masked by tako-tsubo syndrome: A case report. *Case Rep Cardiol.* 2012;2012:486427. [CrossRef]
4. Elhosseiny S, Spagnola J, Royzman R, Lafferty J, Bogin M. Takotsubo cardiomyopathy in a patient with preexisting hypertrophic cardiomyopathy. *Cureus.* 2018;10(11):e3579. [CrossRef]
5. Fefer P, Chelvanathan A, Dick AJ, Teitelbaum EJ, Strauss BH, Cohen EA. Takotsubo cardiomyopathy and left ventricular outflow tract obstruction. *J Interv Cardiol.* 2009;22(5):444–452. [CrossRef]
6. Williams MGL, Liang K, Bucciarelli-Ducci C. Transient recurrent takotsubo cardiomyopathy mimicking apical hypertrophic cardiomyopathy. *Eur Heart J Cardiovasc Imaging.* 2021;22(6):e72. [CrossRef]
7. Amin HZ, Amin LZ, Pradipta A. Takotsubo cardiomyopathy: A brief review. *J Med Life*. 2020;13(1):3–7. [CrossRef]
8. Sealove BA, Tiyyagura S, Fuster V. Takotsubo cardiomyopathy. *J Gen Intern Med.* 2008;23(11):1904–1908. [CrossRef]
9. Lüscher TF, Templin C. Is takotsubo syndrome a microvascular acute coronary syndrome? Towards of a new definition. *Eur Heart J.* 2016;37(37):2816–2820. [CrossRef]
10. Yalta K, Ucar F, Yilmaztepe M, Zorkun C. Takotsubo cardiomyopathy and acute coronary syndromes: Are they always mutually exclusive? *Indian Heart J.* 2018;70(2):326–327. [CrossRef]
11. El Mahmoud R, Mansencal N, Pilliére R, et al. Prevalence and characteristics of left ventricular outflow tract obstruction in Tako-Tsubo syndrome. *Am Heart J.* 2008;156(3):543–548. [CrossRef]
12. Citro R, Bellino M, Merli E, Di Vece D, Sherrid MV. Obstructive hypertrophic cardiomyopathy and Takotsubo syndrome: How to deal with left ventricular ballooning? *J Am Heart Assoc.* 2023;12(21):e032028. [CrossRef]
13. Eitel I, von Knobelsdorff-Brenkenhoff F, Bernhardt P, Carbone I, Muellerleile K, Aldrovandi A, et al. Clinical characteristics and cardiovascular magnetic resonance findings in stress (takotsubo) cardiomyopathy. *JAMA.* 2011;306(3):277–286. [CrossRef]
14. Ghadri JR, Wittstein IS, Prasad A, et al. International expert consensus document on Takotsubo syndrome (Part II): Diagnostic workup, outcome, and management. *Eur Heart J.* 2018;39(22):2047–2062. [CrossRef]
15. Tsutsui H, Albert NM, Coats AJS, et al. Natriuretic peptides: Role in the diagnosis and management of heart failure: a scientific statement from the Heart Failure Association of the European Society of Cardiology, Heart Failure Society of America and Japanese Heart Failure Society. *Eur J Heart Fail.* 2023;25(5):616–631. [CrossRef]