Case Report

Takotsubo Syndrome Presenting as MINOCA in a Patient with Hypertrophic Cardiomyopathy and Pre-Existing Advanced Conduction Disease: Diagnostic and Therapeutic Challenges

Mohammad Jabalameli, Ali Hosseinsabet, Yaser Jenab, Ali Vasheghani Farahani ,Haleh Ashraf *

Tehran Heart Center,Tehran University of Medical Sciences,Tehran, Iran.

Citation: Jabalameli M, Hosseinsabet A, Jenab Y, Vasheghani Farahani A, Ashraf H. Takotsubo Syndrome Presenting as MINOCA in a Patient with Hypertrophic Cardiomyopathy and Pre-Existing Advanced Conduction Disease: Diagnostic and Therapeutic Challenges. Res Heart Yield Transl Med 2026; 21(4):333-338.

https://doi.org/10.18502/rhythm.v21i4.23097

Highlights

- CMR is indispensable for establishing Takotsubo syndrome in MINOCA presentations-identifying extensive apical edema exceeding LGE-even when clinical scores like the InterTAK score are low.
- In cardiomyopathies characterized by dynamic LVOT obstruction and small/hypercontractile chambers (e.g., HCM, Takotsubo syndrome), cautious volume resuscitation and beta-blockade are vital to alleviate gradients and stabilize hemodynamics.
- Temporary pacing safely bridges patients with high-grade conduction disease, permitting the necessary administration of beta-blockers without precipitating critical bradyarrhythmias.

Article info:

Received: 26 Jun. 2026
Revised: 04 Jul. 2026
Accepted: 07 Jul. 2026

*** Corresponding Author:**
Haleh Ashraf
Cardiovascular Diseases Research Institute, Tehran Heart Center, Tehran University of Medical Sciences, Tehran, Iran.
Tel: +98 911 183 9831
E-mail: halehashraf@gmail.com

ABSTRACT

Background: Takotsubo syndrome (TTS) may mimic acute myocardial infarction and occasionally occurs in patients with hypertrophic cardiomyopathy (HCM), creating important diagnostic and therapeutic challenges.

Case Summary: A 76-year-old man with diabetes mellitus, hypertension, smoking history, HCM, and prior coronary angioplasty presented with chest pain and anterior ST-segment elevation. Transthoracic echocardiography demonstrated severe left ventricular systolic dysfunction with apical hypokinesia, whereas coronary angiography showed no obstructive culprit lesion, consistent with myocardial infarction with non-obstructive coronary arteries (MINOCA). During hospitalization, the patient developed worsening dyspnea, tachycardia, and hypotension. Repeat echocardiography demonstrated persistent severe left ventricular dysfunction and hypertrophy of the mid-anteroseptal wall suggestive of dynamic obstructive physiology in the setting of HCM. Because β-blocker therapy was considered necessary for hemodynamic stabilization in a patient with previously documented high-grade atrioventricular block and bifascicular block, a temporary transvenous pacemaker was implanted before initiation of β-blockade. Cardiac magnetic resonance imaging demonstrated extensive apical edema with limited late gadolinium enhancement, supporting TTS rather than ischemic myocardial infarction or myocarditis superimposed on underlying HCM. Owing to preexisting conduction disease, the patient ultimately underwent implantable cardioverter-defibrillator placement. One month later, complete recovery of ventricular systolic function was observed.

Discussion: This case highlights the complex interaction between TTS and HCM and emphasizes the importance of distinguishing preexisting conduction disease from arrhythmias directly attributable to TTS.

Keywords: Takotsubo Syndrome (TTS); Hypertrophic Cardiomyopathy (HCM); Atrioventricular Block; Left Ventricular Outflow Tract Obstruction

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Introduction

Takotsubo syndrome (TTS) is an acute heart failure syndrome characterized by transient left ventricular (LV) systolic dysfunction in the absence of obstructive coronary artery disease.[1,2]

Its presentation frequently mimics acute myocardial infarction, including chest pain, ST-segment elevation, elevated cardiac biomarkers, and regional wall motion abnormalities. Although emotional or physical stressors are commonly identified, they may be absent in a considerable proportion of patients.[1,2]

Hypertrophic cardiomyopathy (HCM) may complicate both the presentation and management of TTS because catecholamine-mediated hypercontractility can aggravate dynamic left ventricular outflow tract (LVOT) obstruction and worsen hemodynamic instability.[3,4] Conduction abnormalities have occasionally been reported in association with TTS; nonetheless, differentiating preexisting conduction disease from acute TTS-related arrhythmia may be clinically important because management and prognostic implications differ substantially.[5]

We report a case of TTS presenting as myocardial infarction with non-obstructive coronary arteries (MINOCA) in a patient with underlying HCM and previously documented advanced conduction disease, in whom temporary pacing support was required to permit safe β-blocker therapy during acute hemodynamic deterioration.

Case Presentation

A 76-year-old man with a history of diabetes mellitus, hypertension, a smoking history, hypertrophic cardiomyopathy diagnosed 13 years earlier by cardiac magnetic resonance imaging (CMR), and previous coronary angioplasty of the posterolateral branch performed 13 years prior presented to the emergency department with intermittent chest pain of 2 weeks' duration that had markedly worsened in the preceding 5 hours. He did not report any emotional stress, recent infection, trauma, surgery, or other identifiable physical triggers.

At presentation, the patient was hemodynamically stable. Electrocardiography (ECG) revealed sinus tachycardia with ST-segment elevation in anterior leads, bifascicular block, and first-degree atrioventricular (AV) block (Figure 1), raising concern for acute anterior myocardial infarction and prompting immediate catheterization. Preprocedural transthoracic echocardiography demonstrated severe LV systolic dysfunction with an estimated ejection fraction (EF) of 25% and apical hypokinesia.

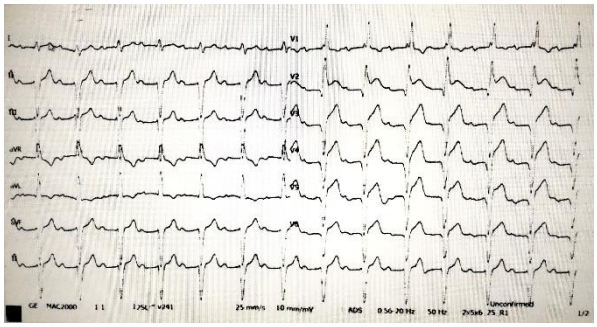


Figure 1. Presenting electrocardiogram demonstrating anterior ST-segment elevation with concomitant first-degree atrioventricular block and bifascicular block

Coronary angiography revealed no obstructive culprit lesion to account for the ECG findings or regional wall motion abnormalities. Only a moderate lesion in the mid segment of the right coronary artery was identified, and the stent in the posterolateral branch was patent. Given the discordance between the angiographic findings, the ECG, and echocardiographic abnormalities, no culprit lesion was identified. Additional intracoronary imaging-intravascular ultrasound (IVUS) and optical coherence tomography (OCT)- and coronary vasoreactivity testing were not performed. Laboratory studies showed a troponin level of 2128 ng/L, a B-type natriuretic peptide (BNP) level of 193 pg/mL, a creatinine level of 1.8 mg/dL, and negative C-reactive protein. In the absence of obstructive coronary artery disease, the presentation was initially considered consistent with MINOCA. Nevertheless, alternative mechanisms such as plaque disruption, coronary vasospasm, and microvascular dysfunction could not be definitively excluded. The patient was subsequently admitted to the coronary care unit for further evaluation and monitoring.

Notably, further review of the patient's history revealed preexisting conduction disease prior to this presentation. Two weeks earlier, the patient

had presented to the emergency department with atypical chest pain. At that time, serial troponin measurements were negative, and echocardiography showed normal ventricular systolic function. However, the ECG demonstrated Wenckebach AV block, right bundle branch block, and left anterior fascicular block. Hospital admission was advised but declined by the patient for personal reasons. The patient had been receiving metoprolol (50 mg twice daily), which was discontinued because of the observed conduction abnormalities. A 24-hour Holter monitor was arranged for further evaluation. Review of the Holter data during the current admission revealed episodes of high-grade AV block, confirming that advanced conduction disease predated the development of TTS.

Initial treatment included aspirin, clopidogrel, enalapril, and therapeutic-dose heparin, as transient coronary thrombosis with spontaneous reperfusion could not be excluded. CMR was requested early during hospitalization but was delayed because of scheduling limitations.

On the fourth day during hospitalization, the patient developed progressive dyspnea and orthopnea associated with sinus tachycardia of up to 110 beats/min and hypotension, with blood pressure decreasing to 90/65 mm Hg. Repeat echocardiography showed severe LV systolic dysfunction (EF=20%–25%), pronounced anterior regional wall motion abnormality, mid-anteroseptal hypertrophy (maximal thickness, 19 mm), moderate mitral regurgitation, mild–moderate tricuspid regurgitation, pulmonary artery pressure of 68 mm Hg, severely dilated inferior vena cava without inspiratory collapse, and LVOT obstruction (peak gradient=55 mm Hg) (Figure 2).

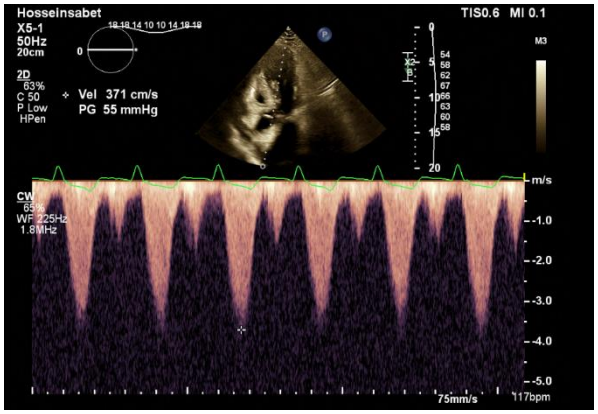


Figure 2. Significant left ventricular outflow tract gradient demonstrated by continuous-wave Doppler

The combination of worsening hemodynamic status, persistent tachycardia, and underlying HCM raised concern for dynamic obstructive physiology despite severe systolic dysfunction. Careful isotonic saline hydration was initiated, and β-blocker therapy was considered necessary. The dilated inferior vena cava without inspiratory collapse indicated elevated right atrial pressure, which-in conjunction with severe LVOT gradient-was more consistent with obstructive physiology rather than primary myocardial pump failure, further supporting the decision of cautious administration of intravenous fluids and β-blocker. Still, given the patient's documented high-grade AV block and bifascicular conduction disease preceding this episode, initiation of β-blockade without pacing support was deemed unsafe. A temporary transvenous pacemaker was, therefore, implanted prior to β-blocker administration. A single-dose bolus of intravenous esmolol (500 μg/kg) reduced the heart rate to approximately 85 beats/min without worsening hypotension or pulmonary congestion. Mild improvement in dyspnea was observed shortly afterward. The patient was subsequently transitioned to oral metoprolol (12.5 mg every 8 hours) while cautious fluid administration was continued. Over the following day, the patient's respiratory distress improved significantly, pulmonary rales resolved, and vital signs stabilized (heart rate, 77 beats/min; blood pressure, 105/70 mm Hg). Repeat echocardiography demonstrated improvement in EF to approximately 30% without significant LVOT gradient.

On hospital day 6, following continued clinical stabilization, CMR was performed. Because the temporary pacing system was not CMR-compatible, it was removed before image acquisition. CMR revealed moderate LV dysfunction with an LVEF of 36%, marked apical ballooning without thrombus, extensive apical edema and inflammation, and limited late gadolinium enhancement (LGE) that was disproportionately small relative to the extent of myocardial edema. These findings strongly supported TTS superimposed on underlying HCM rather than ischemic myocardial infarction[7] (Figures 3 and 4). Subsequently, heparin and clopidogrel were discontinued because of the low probability of acute myocardial infarction after CMR.

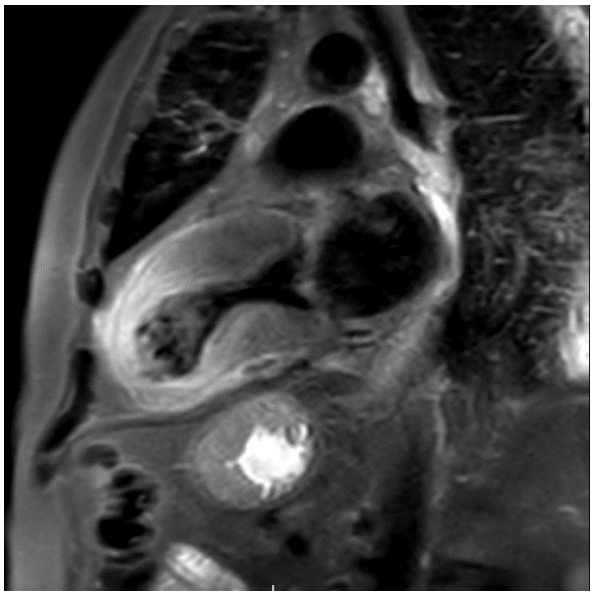


Figure 3. Short tau inversion recovery (STIR) sequence of cardiac magnetic resonance imaging demonstrating significant apical edema (vertical long-axis [2-chamber] view)

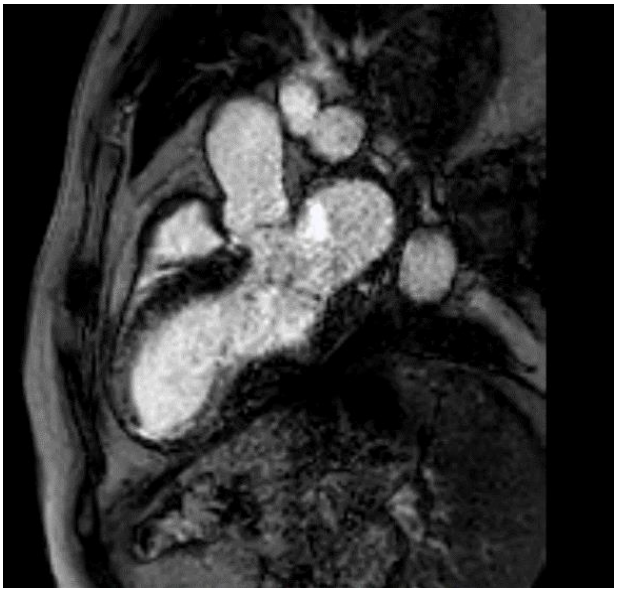


Figure 4. Phase-sensitive inversion recovery (PSIR) late gadolinium enhancement (LGE) sequence of cardiac magnetic resonance imaging demonstrating mild apical LGE (vertical long-axis [2-chamber] view).

Following CMR acquisition, the patient again developed high-grade AV block with the heart rate decreasing to 41 beats/min, although he remained hemodynamically stable. Importantly, this conduction abnormality was interpreted as a manifestation of his previously documented conduction system disease rather than a new arrhythmic complication caused by TTS. Given the preexisting high-grade AV block requiring permanent pacing, severe LV dysfunction with apical ballooning, and underlying HCM, a dual-chamber implantable cardioverter-defibrillator (ICD) was placed.

The patient subsequently demonstrated progressive clinical recovery and became fully ambulatory. Considering the markedly depressed ventricular function and the presence of marked apical ballooning, apixaban (5 mg twice daily) was initiated to reduce thromboembolic risk. He was discharged 4 days later on metoprolol, enalapril, and apixaban.

At 1-month follow-up, the patient was asymptomatic with complete functional recovery. ICD interrogation demonstrated no ventricular arrhythmias or device therapies, with an atrial pacing burden of 42% and a ventricular pacing burden of 99%. Repeat echocardiography showed normalization of LV systolic function with an EF of 55% and complete resolution of the apical ballooning and regional wall motion abnormalities. No residual LVOT gradient was observed. Given the recovery of ventricular function and resolution of the ballooning appearance, apixaban therapy was discontinued, while metoprolol and enalapril were continued.

Discussion

This case illustrates several clinically important aspects of TTS in the setting of HCM. First, the patient presented with a classic acute coronary syndrome phenotype, including chest pain, ST-segment elevation, elevated troponin levels, and severe regional wall motion abnormalities. Nonetheless, coronary angiography demonstrated no obstructive culprit lesion, establishing the diagnosis of MINOCA according to the Fourth Universal Definition of Myocardial Infarction.[6] The markedly elevated troponin levels, only mildly elevated BNP concentration, normal QT interval, and absence of traditional TTS predictors-such as female sex, preceding emotional or physical stressors, psychiatric disorders, or neurologic disorders rendered this presentation atypical. Application of the InterTAK Diagnostic Score yielded a score of 0 point, corresponding to a very low pretest probability of TTS.[7] Consequently, the diagnosis remained uncertain on initial presentation and required further evaluation with CMR. Moreover, the biomarker profile initially raised concern for ischemic myocardial injury, as TTS is typically characterized by a modest troponin elevation relative to the degree of LV

dysfunction and disproportionately elevated natriuretic peptide levels.[1] However, CMR demonstrated diffuse myocardial edema with minimal focal LGE, a pattern inconsistent with myocardial infarction, which generally exhibits LGE in a coronary artery distribution.[8]

Taken together, the overall clinical presentation and characteristic CMR findings strongly favored TTS as the leading diagnosis, which was subsequently supported by the complete recovery of LV systolic function on follow-up imaging within a short period.

Second, the coexistence of HCM significantly complicated hemodynamic management. Catecholamine excess in TTS may increase basal contractility and aggravate dynamic LVOT obstruction in predisposed patients. In this patient, apical stunning from TTS caused low global EF, while catecholamine-mediated basal hypercontractility (driven by both HCM and TTS pathophysiology) generated the LVOT gradient. This apparent paradox is well described in TTS with HCM. In the present case, progressive hypotension and pulmonary congestion despite severe systolic dysfunction raised suspicion for dynamic obstructive physiology. Recognition of this mechanism was clinically important because β-blockade and cautious fluid administration resulted in rapid clinical improvement.

Third, this case emphasizes the importance of correctly interpreting conduction abnormalities in patients with TTS. Although arrhythmias and AV block have occasionally been described in TTS,[5] the conduction disease in this patient clearly predated the acute TTS presentation. Advanced conduction abnormalities had already been documented on ECG and Holter monitoring before the current hospitalization. Therefore, the recurrent high-grade AV block observed during admission was considered related to underlying conduction system disease rather than a direct manifestation of TTS. Similar cases of TTS associated with high-grade AV block have been previously reported.[5] Still, we were unable to identify a closely comparable case combining TTS, HCM, and clearly documented preexisting high-grade AV block. This distinction had important management implications. Temporary pacing was instituted not because TTS caused conduction block, but rather to permit safe initiation of β-blocker therapy in a patient with known advanced conduction disease and suspected dynamic obstructive physiology.

The decision to implant an ICD rather than a conventional permanent pacemaker was primarily driven by uncertainty regarding the reversibility of the severe LV dysfunction and apical ballooning at the time of device implantation. Although TTS is generally considered reversible, there was minimal improvement in ventricular function during the first week of hospitalization, making the long-term substrate unclear. If LV dysfunction or apical aneurysm formation had persisted, the coexistence of HCM could have supported consideration of ICD therapy for primary prevention of sudden cardiac death. Because the patient had persistent high-grade AV block requiring permanent pacing, prolonged use of a temporary pacing system was considered undesirable because of the risk of device-related complications. Furthermore, implantation of a conventional permanent pacemaker with subsequent reassessment could have necessitated a second invasive procedure for device upgrade if recovery did not occur. The option of a wearable cardioverter-defibrillator was also considered less suitable in this context, as it would not address the pacing requirement and could still result in the need for repeat device intervention if LV dysfunction persisted. Thus, ICD implantation was chosen as an individualized management strategy under conditions of diagnostic uncertainty rather than as a general recommendation for patients with acute TTS. In retrospect, recovery of ventricular function confirmed the reversible nature of the cardiomyopathy; however, this outcome could not be predicted with certainty at the time the permanent device was implanted.

Fourth, metoprolol therapy was discontinued by the treating clinician because of the underlying conduction abnormalities. As the patient preferred outpatient follow-up and declined hospitalization at the first visit 2 weeks before the TTS episode, a monitored tapering strategy was not feasible, and the medication was therefore discontinued abruptly. Whether this abrupt withdrawal contributed to the subsequent development of TTS remains speculative. Be that as it may, β-

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blocker discontinuation has been proposed as a potential precipitating factor through an adrenergic rebound mechanism in previous reports.[9]

Finally, CMR played a pivotal diagnostic role by demonstrating myocardial edema with limited LGE and subsequent recovery of ventricular function, findings strongly supporting TTS rather than ischemic infarction or inflammatory cardiomyopathy.[8]

Conclusion

TTS should be considered in patients presenting with MINOCA and acute, severe LV dysfunction, particularly when coronary angiography is non-obstructive. Coexisting HCM may create a complex hemodynamic scenario requiring individualized therapy. This case also highlights the importance of distinguishing preexisting conduction disease from arrhythmic manifestations directly attributable to TTS, as this distinction may significantly influence management decisions and device implantation strategies.

Limitations

We did not perform invasive hemodynamic monitoring to confirm LVOT obstruction and to guide the supportive hemodynamic management. Intracoronary imaging and coronary vasoreactivity testing were not performed. Therefore, although the clinical presentation and subsequent imaging findings strongly supported TTS, alternative MINOCA mechanisms-including plaque disruption, coronary vasospasm, and microvascular dysfunction-cannot be completely excluded, although they appear unlikely.

Declarations:

Funding

According to the authors, this article has no financial support.

Conflict of Interest

The authors report no conflict of interest.

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