

Takotsubo Cardiomyopathy: A Clinical Case of Stress-Induced Cardiomyopathy Without Precipitating Factors

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Abstract

Background: Takotsubo cardiomyopathy is a transient left ventricular dysfunction typically triggered by emotional or physical stress. It is characterized by regional wall motion abnormalities that extend beyond a single coronary artery distribution and often resolves within weeks.

Case Summary: A 45-year-old woman with hypertension and anxiety presented with acute retrosternal chest pain radiating to the left arm. She denied recent psychological or physical stressors. Serial troponins were elevated, and echocardiography showed apical hypokinesia. Coronary angiography revealed no obstructive coronary disease, confirming stress-induced cardiomyopathy. She was managed conservatively and discharged in stable condition.

Discussion: Although classically seen in postmenopausal women under stress, Takotsubo cardiomyopathy can occur without identifiable triggers. This case highlights the importance of recognizing atypical presentations to avoid unnecessary interventions and guide appropriate care.

Keywords: Takotsubo cardiomyopathy; Acute coronary syndrome; Cardiac biomarkers; Electrocardiographic abnormalities; Brain natriuretic peptide

Take-Home Messages

Takotsubo cardiomyopathy may occur in the absence of emotional or physical stressors. It should be considered in patients with chest pain and non-obstructive coronary findings.

History of Presentation

The patient was a forty-five-year-old female who awoke in the early morning with abrupt-onset substernal chest pressure accompanied by lightheadedness. She denied orthopnea, paroxysmal nocturnal dyspnea, nausea, vomiting, peripheral edema, or syncope.

Notably, she did not report any recent psychological trauma or intense physical exertion preceding the onset of symptoms. The nature of her symptoms was inconsistent with previous anxiety-related episodes. Upon arrival to the emergency department, the patient's blood pressure was elevated at 187/83 mm Hg. Her heart rate, respiratory rate, and oxygen saturation on room air were within normal limits. Physical examination revealed no acute abnormalities.

Past Medical History

She reported a past medical history of essential hypertension and generalized anxiety disorder. Her medication regimen included venlafaxine and combined oral contraceptives. She denied tobacco, alcohol, or recreational drug use. Her family history was significant for maternal coronary artery disease and congestive heart failure.

Differential Diagnosis

The patient's initial presentation with abrupt-onset substernal chest pressure and associated lightheadedness raised concern for a cardiopulmonary etiology. Her elevated blood pressure on arrival (187/83 mm Hg), in the setting of chest discomfort, suggested the possibility of hypertensive emergency or acute coronary syndrome. The absence of orthopnea, paroxysmal nocturnal dyspnea, peripheral edema, or signs of heart failure on physical examination made decompensated heart failure less likely. Similarly, the lack of fever, tachypnea, or abnormal lung findings decreased suspicion for infectious or pulmonary causes such as pneumonia. Although she denied recent emotional or physical stressors, the nature of her symptoms being inconsistent with prior anxiety episodes reduced the likelihood of a primary anxiety or panic attack. Her stable vital signs aside from elevated blood pressure, combined with an unremarkable physical exam, supported further evaluation for acute coronary syndrome, aortic pathology, or other causes of myocardial strain.

Investigations

A chest radiograph was unremarkable. Electrocardiographic evaluation demonstrated sinus rhythm at a rate of 77 beats per minute with moderate T-wave abnormalities. Laboratory testing revealed an initial troponin-I concentration of 192 ng/L, followed by elevated values of 1456 ng/L and 1474 ng/L. A D-dimer level was 198 ng/mL. Complete blood count and basic metabolic panel were unremarkable, except for a blood urea nitrogen concentration of 8 mg/dL. Transthoracic echocardiography demonstrated moderate apical hypokinesia of the left ventricle, moderate aortic regurgitation, mild tricuspid regurgitation, and poor visualization of the pulmonic valve. On hospital day one, a cardiac computed tomography calcium scoring scan showed a calcium score of zero and incidentally noted calcified granulomas in the left lower lobe of the lung. Left heart catheterization revealed a dyskinetic left ventricular apex, mild to moderate coronary artery disease involving the left anterior descending artery, and codominance of the right and left circumflex arteries. Left ventricular ejection fraction was preserved at greater than 50%, consistent with stress cardiomyopathy.

Management

In the emergency department, the patient received 325 mg of oral aspirin, 80 mg of subcutaneous enoxaparin, and 0.4 mg of sublingual nitroglycerin, which resulted in prompt relief of chest discomfort. A continuous intravenous infusion of nitroglycerin in 5% dextrose in water was initiated at 5 micrograms per minute. Cardiology was consulted, and the patient was admitted with a working diagnosis of non-ST-elevation myocardial infarction. Following cardiac catheterization, she was started on 60 mg of oral diltiazem and 75 mg of clopidogrel daily. Additional medications initiated during the hospital stay included losartan 25 mg

daily, atorvastatin 40 mg daily, metoprolol 50 mg daily, and aspirin 81 mg daily. She was advised to continue her home medications, including venlafaxine and combined oral contraceptives.

Outcome and Follow-Up

By hospital day two, the patient remained hemodynamically stable with no recurrence of chest pain. She was discharged in stable condition with resolution of her presenting symptoms. Follow-up with cardiology was arranged within one week to monitor for recurrence of chest pain or dyspnea and to reassess cardiac function with repeat echocardiography. At discharge, the patient expressed satisfaction with her care and confidence in the treatment plan. Written informed consent was obtained for publication of this case report.

Discussion

First described in 1990 in Japan, Takotsubo cardiomyopathy—also referred to as stress-induced cardiomyopathy or transient apical ballooning syndrome—is characterized by reversible systolic and diastolic dysfunction of the left ventricle, typically precipitated by intense emotional or physical stress [1]. The clinical presentation frequently mimics acute myocardial infarction, including chest pain, electrocardiographic changes, and elevated serum troponin levels [2]. However, unlike myocardial infarction, coronary angiography reveals no significant obstructive coronary artery disease [3]. Excessive catecholaminergic stimulation is believed to mediate the pathophysiological response, leading to myocardial stunning [4]. In this context, brain natriuretic peptide is often markedly elevated, and echocardiography frequently demonstrates wall motion abnormalities that do not conform to a single vascular territory [5]. Although classically associated with severe stress, a subset of patients presents without any overt inciting event [6].

This case highlights a presentation of Takotsubo cardiomyopathy in the absence of an identifiable emotional or physical stressor. The patient, a 45-year-old woman, reported recent job and family-related stress but denied any acute psychological trauma or physical exertion prior to symptom onset. Her clinical course included a peak troponin-I concentration of 1474 ng/L, moderate apical hypokinesia on echocardiography, and a calcium score of zero on cardiac computed tomography. Left heart catheterization demonstrated a dyskinetic apex with preserved ejection fraction and no obstructive coronary artery disease, consistent with stress-induced cardiomyopathy [3]. Although traditionally associated with postmenopausal women following intense emotional experiences, stress-induced cardiomyopathy is increasingly recognized in patients with less obvious triggers or even without any discernible inciting event [1]. This supports the hypothesis that intrinsic autonomic dysregulation or heightened myocardial sensitivity to catecholamines may underlie the syndrome in a subset of patients [6]. Notably, catecholamine levels in Takotsubo cardiomyopathy have been observed to be two to three times higher than those in acute myocardial infarction, suggesting a key role in pathogenesis [4]. The patient's elevated cardiac biomarkers and electrocardiographic abnormalities initially met criteria for non-ST-elevation myocardial infarction, but the absence of occlusive disease on angiography was pivotal for diagnosis. The echocardiographic findings of regional wall motion abnormalities—specifically, apical hypokinesia—were characteristic of the syndrome. Although left ventricular systolic function often normalizes within days to weeks, residual diastolic dysfunction may persist in some individuals [6]. Brain natriuretic peptide levels, which were not measured in this patient, have been shown to be significantly higher in Takotsubo cardiomyopathy compared to acute coronary syndrome and may correlate with disease severity [5].

Emotional or physical stress may lead to increased catecholamine release, resulting in microvascular dysfunction and intracellular calcium overload. Compared to STEMI, TCM is typically associated with lower troponin T levels but markedly elevated brain natriuretic peptide (BNP), reflecting myocardial stunning rather than infarction [(Figure 1) Central Illustration]. Supportive management, including beta-adrenergic blockers and angiotensin receptor antagonists, is generally recommended to reduce sympathetic tone and myocardial stress [1]. After functional recovery, some guidelines suggest tapering angiotensin-modulating therapy while continuing beta-blockers to reduce recurrence risk [3]. Inotropic agents should be avoided as they may exacerbate catecholamine-mediated injury [1].

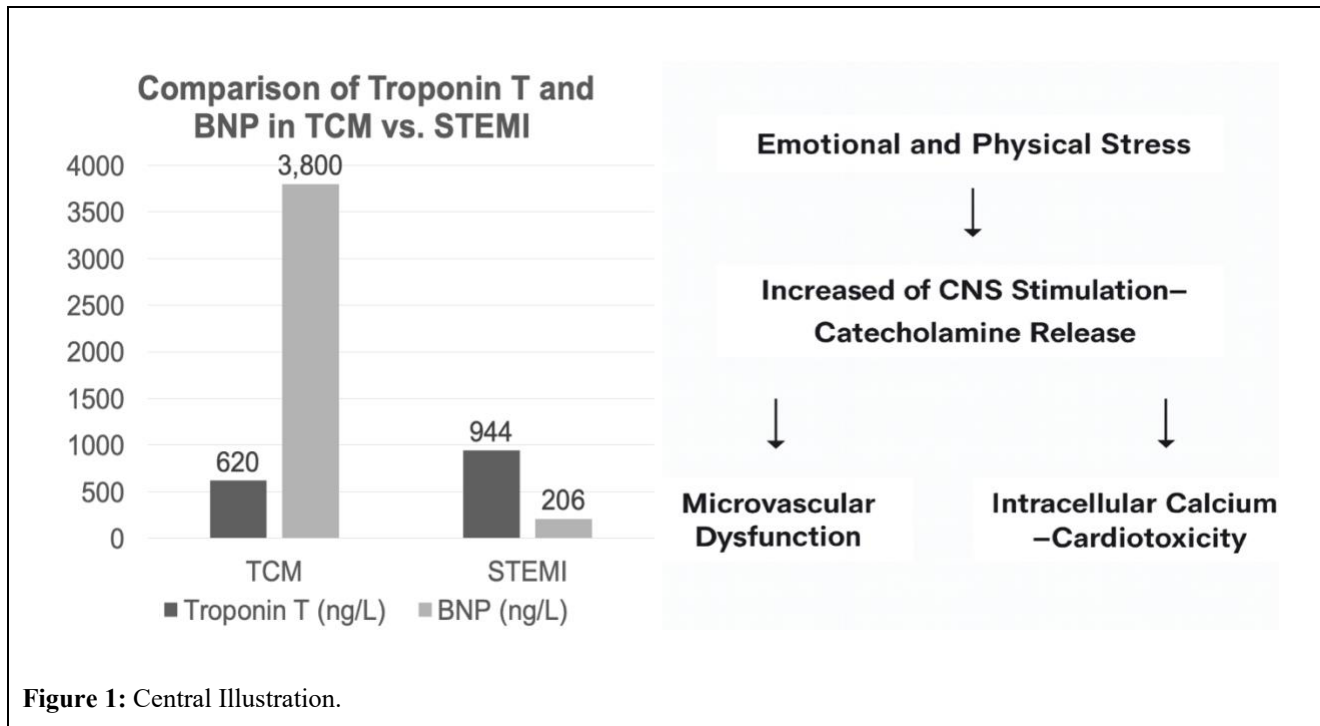


Figure 1: Central Illustration.

Conclusion

This case emphasizes the importance of recognizing Takotsubo cardiomyopathy as a potential cause of acute chest pain, particularly in patients with clinical and biochemical features suggestive of acute coronary syndrome but without angiographic evidence of obstructive coronary disease. While emotional or physical stress is a well-established trigger that may lead to increased catecholamine release, microvascular dysfunction, and intracellular calcium overload, this patient's clinical presentation occurred without any identifiable precipitating event—supporting the growing recognition that Takotsubo cardiomyopathy may also arise in the absence of overt stressors.

Take-Home Messages

1. Takotsubo cardiomyopathy may occur in the absence of clear physical or emotional stressors.
2. It should be considered when there are wall motion abnormalities and no coronary obstruction.
3. Early recognition prevents unnecessary intervention and facilitates proper supportive management.

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Author's Contributions

Anne Eliese Daley, OMS-IV: Drafted the manuscript and performed the literature review.

Gagandeep Grewal, MD: Supervised clinical care and contributed to clinical decision-making and interpretation.

Brandi Smith, FNP-C: Coordinated patient follow-up and data collection from the patient's electronic medical record.

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