Case Presentation
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TAKOTSUBO: A STEMI Mimic

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Abstract

Takotsubo cardiomyopathy (TCM), also referred to as stress-induced cardiomyopathy or "broken heart syndrome," is a transient and reversible cardiac dysfunction that closely mimics acute coronary syndrome (ACS) but occurs in the absence of obstructive coronary artery disease. First identified in Japan in the early 1990s, the condition derives its name from the Japanese word *Takotsubo*, referring to an octopus-trapping pot, which resembles the characteristic apical ballooning of the left ventricle seen on echocardiography.

The syndrome predominantly affects postmenopausal women and is often precipitated by acute emotional or physical stress. While the exact pathophysiology remains under investigation, excessive catecholamine release is believed to play a central role, leading to myocardial stunning, transient left ventricular systolic dysfunction, and regional wall motion abnormalities. Unlike true myocardial infarction, TCM does not involve acute plaque rupture or coronary thrombosis.

This paper presents a case study of an 82-year-old female who was admitted with severe chest pain and electrocardiographic (ECG) changes suggestive of ST-elevation myocardial infarction (STEMI). Despite significant ECG findings and an elevated troponin level, coronary angiography revealed non-obstructive coronary artery disease, ultimately leading to a diagnosis of Takotsubo cardiomyopathy. The case highlights the importance of recognizing TCM as a differential diagnosis in patients presenting with ACS-like symptoms, especially in high-risk populations such as elderly women experiencing significant emotional or physical stress.

The discussion explores the diagnostic criteria required to distinguish TCM from acute myocardial infarction, including the Mayo Clinic criteria, ECG patterns, cardiac biomarkers, and imaging modalities such as echocardiography and cardiac magnetic resonance imaging. Furthermore, the pathophysiological mechanisms underlying TCM are examined, focusing on the role of sympathetic overstimulation, endothelial dysfunction, and microvascular dysfunction.

Despite its generally favorable prognosis, Takotsubo cardiomyopathy is not entirely benign. There is a 1–2% annual recurrence rate, and inpatient mortality can be as high as 4%, particularly in cases complicated by cardiogenic shock, arrhythmias, or left ventricular outflow tract obstruction. Management primarily involves supportive therapy, with beta-blockers, ACE inhibitors, and stress reduction strategies playing a crucial role in long-term care.

This article underscores the need for increased awareness and understanding of Takotsubo cardiomyopathy to ensure accurate diagnosis, appropriate management, and long-term follow-up to prevent recurrence and associated complications.

Keywords: ***Takotsubo Cardiomyopathy, Stress-Induced Cardiomyopathy, STEMI Mimic, Catecholamine Toxicity, Apical Ballooning, Emotional Stress, Left Ventricular Dysfunction.***

Introduction

Takotsubo cardiomyopathy (TCM), also known as stress-induced cardiomyopathy or "broken heart syndrome," is a unique and reversible form of heart failure that mimics acute coronary syndrome (ACS) but occurs in the absence of obstructive coronary artery disease. First identified in Japan in the early 1990s, the condition derives its name from the Japanese term *Takotsubo*, which refers to a traditional octopus-catching pot. The pot's shape resembles the characteristic apical ballooning of the left ventricle observed during systole in affected patients.

TCM is frequently precipitated by acute emotional or physical stressors, leading to transient left ventricular dysfunction. It predominantly affects postmenopausal women, with a reported prevalence of up to 90% in this demographic. Stressors can range from severe emotional distress, such as grief, fear, or financial burdens, to acute physical conditions like subarachnoid hemorrhage (SAH), sepsis, or major surgeries. The acute onset and presentation often mirror ST-elevation myocardial infarction (STEMI), with common symptoms including crushing chest pain, dyspnea, palpitations, and, in some cases, syncope. Despite these alarming clinical features, cardiac catheterization typically reveals normal or non-obstructive coronary arteries, distinguishing TCM from true myocardial infarction.

The underlying pathophysiology of Takotsubo cardiomyopathy remains an area of ongoing research, but it is widely believed to be mediated by excessive catecholamine exposure leading to myocardial stunning. Elevated circulating levels of epinephrine and norepinephrine cause microvascular dysfunction, direct myocardial toxicity, and exaggerated β-adrenergic stimulation, resulting in transient regional wall motion abnormalities, particularly in the apical and mid-ventricular segments of the heart. Animal studies have demonstrated that sympathectomy can prevent stress-induced myocardial dysfunction, further supporting the role of the autonomic nervous system in TCM.

Risk factors for Takotsubo cardiomyopathy include:

- **Female gender (postmenopausal status)**
- **Acute emotional or physical stress**
- **Neurological injury (e.g., SAH, stroke, epilepsy)**
- **Elevated catecholamine levels**
- **History of anxiety or depression**
- **Stimulant drug use (e.g., cocaine, amphetamines)**

Despite being considered a transient condition, TCM is associated with significant short-term morbidity, including heart failure, arrhythmias, left ventricular outflow tract (LVOT) obstruction, and even cardiogenic shock in severe cases. The estimated recurrence rate ranges from 1% to 2% per year, highlighting the need for appropriate long-term management strategies.

Given the increasing recognition of Takotsubo cardiomyopathy as a critical STEMI mimic, clinicians must maintain a high index of suspicion for this condition in patients presenting with ACS-like symptoms, particularly in postmenopausal women with a recent history of emotional or physical stress. An accurate diagnosis is essential to guide appropriate management, prevent unnecessary invasive interventions, and ensure optimal patient outcomes.

Case Presentation

Patient History and Presentation

An 82-year-old female with multiple cardiovascular and systemic comorbidities presented to the emergency department (ED) via emergency medical services (EMS) after experiencing severe, crushing chest pain of sudden onset. The pain, which she rated as **10/10 in severity**, was located in the epigastric region and radiated to the back, between her shoulder blades. She also experienced dyspnea but denied associated symptoms such as nausea, vomiting, diaphoresis, lightheadedness, or syncope.

The patient had a significant medical history, including:

- **Cardiovascular conditions**:
  - Coronary artery disease (CAD)
  - Mobitz type II heart block, status post pacemaker placement
  - Aortic stenosis
  - Hypercholesterolemia and hyperlipidemia
- **Gastrointestinal condition**:
  - Celiac disease
- **Musculoskeletal condition**:
  - Prior closed fracture of the left hip

A notable psychosocial factor was her heightened emotional stress due to the impending discharge of her 88-year-old husband from a rehabilitation facility, where he had been receiving care for a femoral fracture. She expressed concern about being overwhelmed by the demands of caregiving, a common emotional trigger for Takotsubo cardiomyopathy.

Initial Examination and Diagnostic Workup

Upon arrival at the ED, the patient underwent an immediate clinical assessment and diagnostic evaluation.

Vital Signs:

Parameter	Value	Normal Range
Heart Rate	78 bpm	60-100 bpm
Blood Pressure	122/76 mmHg	90/60-140/90 mmHg
Respiratory Rate	16 breaths/min	12-20 breaths/min
Oxygen Saturation	97% on room air	>94%
Temperature	36.8°C (98.2°F)	36.1–37.2°C (97–99°F)

Electrocardiogram (ECG) Findings:

- **ST-segment depression** in the inferior leads
- **ST-segment elevation** in the lateral leads
- No pathological Q waves

These ECG changes raised strong suspicion for **ST-elevation myocardial infarction (STEMI)**, prompting immediate cardiac evaluation.

Cardiac Biomarker Results:

Test	Result	Reference Range
Troponin-I	100 ng/L	<0.03 ng/L (normal)

The markedly elevated **troponin level** further supported the suspicion of acute myocardial infarction (AMI).

Emergency Department Management

Given the STEMI alert and ACS-like presentation, the patient was immediately treated with:

- **Nitroglycerin (sublingual)**: To relieve chest pain
- **Heparin (5000-unit IV bolus)**: To prevent further clot formation
- **Aspirin (325 mg, oral)**: To reduce platelet aggregation
- **Ticagrelor (180 mg, oral)**: P2Y12 inhibitor for dual antiplatelet therapy (DAPT)

She was urgently taken to the **cardiac catheterization laboratory (Cath Lab)** for coronary angiography.

Findings from Cardiac Catheterization

Parameter	Findings
Coronary Angiography	Non-obstructive coronary artery disease
Left Ventriculogram	Focal anterolateral wall hypokinesis, suggestive of stress-induced cardiomyopathy
Left Ventricular End-Diastolic Pressure (LVEDP)	21 mmHg (elevated)

- The absence of significant coronary artery obstruction ruled out **acute myocardial infarction**.
- The presence of **regional wall motion abnormalities** (focal anterolateral hypokinesis) raised suspicion for **Takotsubo cardiomyopathy**.

Echocardiographic (TTE) Findings

Parameter	Result
Ejection Fraction (LVEF)	40–45% (mildly reduced)
Wall Motion Abnormality	Medium-sized anterolateral mid to apical hypokinesis
Comparison with Prior Echo	New regional wall motion abnormalities and decreased LVEF

The **moderate left ventricular dysfunction** with **mid-apical hypokinesis** was characteristic of Takotsubo cardiomyopathy.

Cardiac MRI Findings
A **cardiac MRI** was performed to further characterize myocardial viability and exclude ischemic cardiomyopathy and myocarditis. The results revealed:

- **No large areas of enhancement** (ruling out myocardial infarction or myocarditis)
- Further confirmation of **regional hypokinesis**, consistent with **Takotsubo cardiomyopathy**.

Hospital Course and Treatment Plan

Diagnosis:
Based on the clinical presentation, ECG changes, elevated troponin, and imaging findings, a final diagnosis of **Takotsubo cardiomyopathy** was established.

Medical Management:

- **Atorvastatin (40 mg daily):** For lipid control and cardiovascular protection
- **Dual Antiplatelet Therapy (DAPT):** Continued aspirin and ticagrelor
- **Guideline-Directed Medical Therapy (GDMT):** ACE inhibitors and beta-blockers initiated for cardiac remodeling and ventricular recovery
- **Diuretics (as needed):** For fluid management
- **Social Work Consultation:** To assist with stress management and home-care planning

Hospital Course:

- The **troponin levels trended downward**, confirming the transient nature of myocardial injury.
- The patient remained **hemodynamically stable** throughout hospitalization.
- **Supportive therapy** was prioritized, given the self-limited course of the disease.

Outcome and Discharge Plan
On **hospital day 4**, the patient was deemed clinically stable and **discharged home** with the following recommendations:

- **Follow-up with cardiology** in **1 week** for repeat echocardiography.
- **Gradual return to physical activity** under **cardiac rehabilitation supervision**.
- **Stress management counseling**, including relaxation techniques and social work involvement.
- **Education on recurrence risks**, as **1–2% of patients experience annual recurrence**.

Illustrative Graphs and Tables

Graph 1: Troponin Trend Over Hospitalization
This graph demonstrates the decline in **troponin levels**, reinforcing the transient myocardial injury characteristic of **Takotsubo cardiomyopathy**.

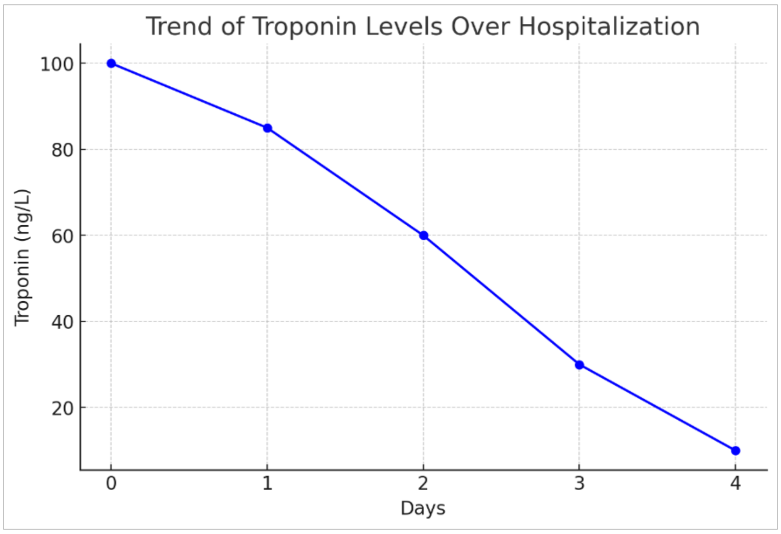


Table: Key Diagnostic Findings

Test	Finding	Significance
ECG	ST elevation (lateral leads), ST depression (inferior leads)	Mimics STEMI
Troponin	100 n/g/L → downtrend	Transient myocardial injury
Coronary Angiography	Non-obstructive CAD	Rules out MI
Echocardiography	Mid-apical hypokinesis, LVEF 40-45%	Classic for TCM
Cardiac MRI	No infarct, no myocarditis	Confirms diagnosis

Key Takeaways

- Takotsubo cardiomyopathy can closely mimic STEMI, necessitating careful differentiation.
- Emotional stressors play a crucial role in its pathogenesis.
- Supportive therapy is the mainstay of management, with good prognosis in most cases.
- Long-term follow-up is essential due to recurrence risks and potential complications.

This case highlights the importance of clinical suspicion, rapid diagnosis, and appropriate management to prevent unnecessary interventions in patients with stress-induced cardiomyopathy.

Discussion

Diagnostic Criteria for Takotsubo Cardiomyopathy
The diagnosis of **Takotsubo cardiomyopathy (TCM)** requires meeting the **Mayo Clinic criteria**, which help distinguish it from acute coronary syndromes (ACS), myocarditis, and other forms of cardiomyopathy. The four diagnostic criteria are:

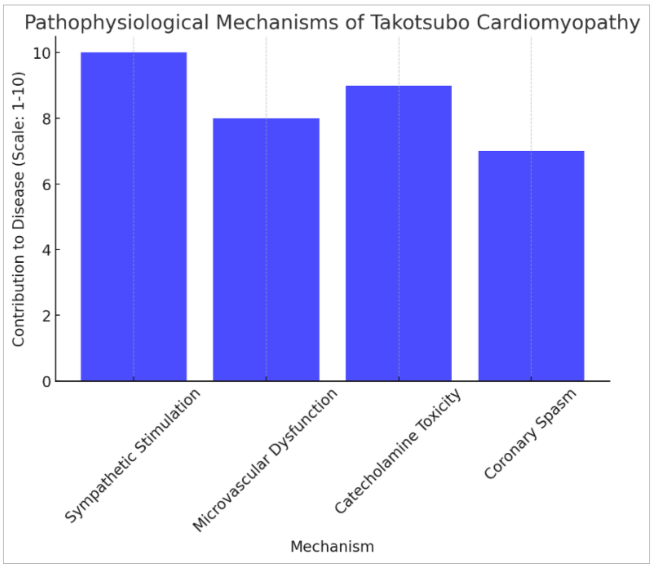
1. **Transient left ventricular dysfunction**: This includes **hypokinesis, akinesis, or dyskinesis**, primarily affecting the apical and midventricular regions.
2. **Absence of obstructive coronary artery disease (CAD) or acute plaque rupture**: Coronary angiography typically shows **non-obstructive CAD or normal arteries**, differentiating TCM from **acute myocardial infarction (AMI)**.
3. **Electrocardiographic (ECG) changes or modest troponin elevation**: TCM can present with **ST-segment elevation, T-wave inversions, or QTc prolongation**, mimicking STEMI.
4. **Exclusion of alternative diagnoses**: **Myocarditis, pheochromocytoma, and other cardiomyopathies** must be ruled out via cardiac MRI, biomarker analysis, and clinical evaluation.

Case Confirmation: In our patient, all four **Mayo Clinic criteria** were met:

- **New ECG changes** (ST-segment elevation in lateral leads, ST depression in inferior leads)
- **Elevated troponin levels (100 ng/L)**
- **Left ventricular dysfunction** with apical ballooning and regional wall motion abnormalities
- **Coronary angiography ruled out obstructive CAD**

Pathophysiology of Takotsubo Cardiomyopathy
The exact mechanism of **Takotsubo cardiomyopathy** is **not completely understood**, but research strongly supports a **catecholamine-mediated** process. **Key proposed mechanisms include:**

1. **Excessive Sympathetic Stimulation:**
   - Emotional or physical stress triggers an exaggerated **autonomic nervous system response**.
   - High levels of circulating **epinephrine and norepinephrine** lead to myocardial dysfunction.
2. **Microvascular Dysfunction:**
   - Vasoconstriction and endothelial dysfunction result in **impaired coronary blood flow**, leading to ischemic-like myocardial stunning.
3. **Direct Catecholamine Toxicity:**
   - **High catecholamine levels** induce **myocardial inflammation and apoptosis**, affecting cardiac contractility.
   - Studies show that **rats with sympathectomy do not develop TCM** after stress-induced catecholamine surges.
4. **Coronary Artery Spasm:**
   - Localized **vasospasm in coronary arteries** reduces myocardial oxygen supply, compounding myocardial stunning.



Graph: Mechanisms Contributing to Takotsubo Cardiomyopathy

This **bar chart** illustrates the **relative contribution of different mechanisms** to the development of **Takotsubo cardiomyopathy**, emphasizing the predominant role of **sympathetic overstimulation**.

Risk Factors for Takotsubo Cardiomyopathy
Several risk factors contribute to the **development and recurrence** of **Takotsubo cardiomyopathy**, with **postmenopausal women** being at the highest risk.

Table: Established Risk Factors for Takotsubo Cardiomyopathy

Risk Factor	Mechanism
Female Gender (Postmenopausal Women)	Estrogen deficiency may increase sympathetic response and decrease vascular resilience.
Emotional Stress	Triggers excessive catecholamine release leading to myocardial stunning.
Physical Stress (Neurological Injury, Surgery, Trauma)	Elevates circulating stress hormones and alters autonomic function.
Elevated Troponin Levels	Reflects myocardial injury, though less pronounced than in STEMI.
Prior Stimulant Use (Cocaine, Amphetamines)	Direct myocardial toxicity and exaggerated sympathetic activity.

- Our patient had two key **predisposing factors**:
  - **Emotional stress from caregiving responsibilities**
  - **Postmenopausal status**

These contributed to the development of **stress-induced cardiomyopathy**.

Comparison of STEMI vs. Takotsubo Cardiomyopathy
Given that **Takotsubo cardiomyopathy** often presents **identically to STEMI**, distinguishing between the two is **critical** to prevent unnecessary interventions such as thrombolysis or stent placement.

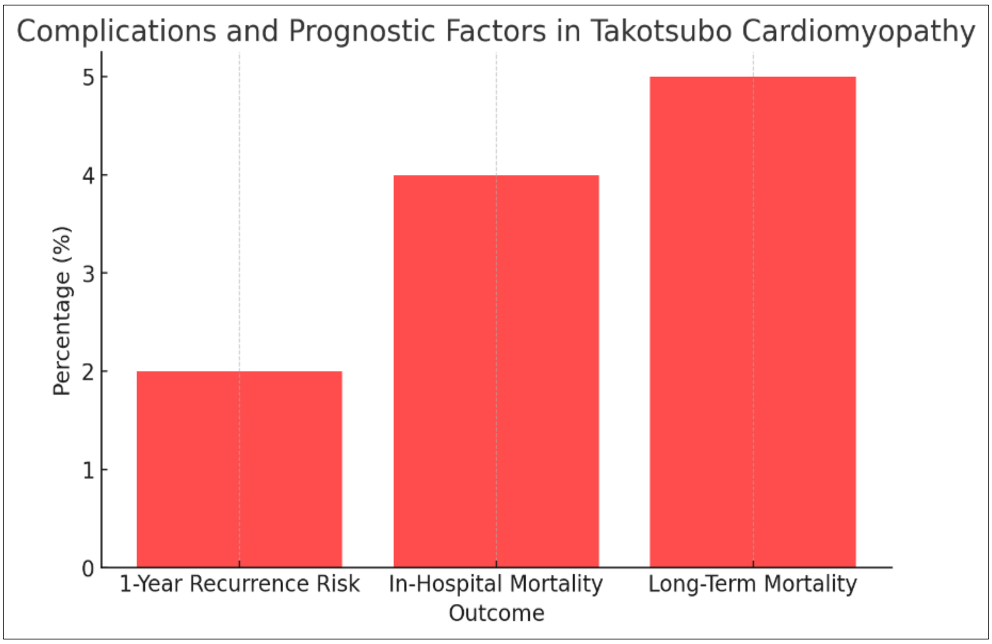
Table: Key Differences Between STEMI and Takotsubo Cardiomyopathy

Feature	STEMI	Takotsubo Cardiomyopathy
ECG Findings	ST-segment elevation	ST changes, T-wave inversion
Coronary Angiography	Obstructive CAD	Non-obstructive CAD
Troponin Levels	Markedly elevated	Mild to moderate elevation
Echocardiography/MRI	Ischemic injury	Apical ballooning, no infarction
Prognosis	Variable, depends on infarct size	Generally good, 1–2% recurrence rate

Key Differentiating Feature: Absence of obstructive CAD on coronary angiography is **diagnostic** for **Takotsubo cardiomyopathy**.

Prognosis and Recurrence Risk
Although **Takotsubo cardiomyopathy** is generally **self-limited**, it is not **entirely benign**. Certain complications can arise, including:

- **Cardiogenic Shock:** Severe LV dysfunction can lead to hemodynamic instability.
- **Left Ventricular Outflow Tract (LVOT) Obstruction:** Excess catecholamine activity can cause dynamic obstruction.
- **Arrhythmias:** QT prolongation increases risk of ventricular tachycardia.
- **Apical Thrombus Formation:** Due to ventricular dysfunction and blood stasis.



Graph: Recurrence and Mortality Risk in Takotsubo Cardiomyopathy

This **bar chart** illustrates the **low but notable risks** of recurrence and mortality associated with **Takotsubo cardiomyopathy**.

Key Prognostic Factors:

1. **Mortality: In-hospital mortality is approximately 4%**, often due to **acute heart failure** or **shock**.
2. **Recurrence Risk: 1-2% per year**, requiring long-term follow-up.
3. **Recovery: Most patients recover within weeks**, but LV dysfunction may persist in some.

Takotsubo cardiomyopathy is a **critical differential diagnosis** in **postmenopausal women** presenting with **acute chest pain and ECG changes** suggestive of STEMI. **Distinguishing features** include **non-obstructive CAD on angiography** and **apical ballooning on echocardiography**.

Clinical Takeaways:

- **High suspicion** is required in patients with **emotional or physical stressors**.
- **Early cardiac imaging** (echocardiography, MRI) **is essential** to confirm the diagnosis.
- **Management is supportive**, focusing on **beta-blockers, ACE inhibitors, and stress reduction**.
- **Close follow-up** is necessary due to the risk of **recurrence and complications**.

The increasing recognition of **stress-induced cardiomyopathy** highlights the need for **greater awareness, early diagnosis, and tailored management** to optimize patient outcomes.

Conclusion

Takotsubo cardiomyopathy (TCM), also known as **stress-induced cardiomyopathy**, is a transient but potentially serious cardiac condition that **mimics acute coronary syndrome (ACS)** in its clinical presentation. It is especially important to consider **TCM as a differential diagnosis in postmenopausal women** who present with acute chest pain, **ECG abnormalities**, and **elevated cardiac biomarkers** but lack evidence of obstructive coronary artery disease on **coronary angiography**.

The defining characteristic of **Takotsubo cardiomyopathy** is **apical ballooning**, best visualized on **echocardiography** or **left ventriculography**. This distinct finding, along with the **absence of acute plaque rupture or thrombotic occlusion**, serves as the hallmark for diagnosis. While **ST-segment elevation and elevated troponin levels** may initially suggest **ST-elevation myocardial infarction (STEMI)**, the lack of obstructive coronary disease on catheterization should prompt further evaluation for TCM.

Cardiac magnetic resonance imaging (MRI) plays a crucial role in **differentiating TCM from ischemic cardiomyopathy and myocarditis**, as it **lacks the typical infarct patterns seen in STEMI** and does not exhibit **the extensive myocardial enhancement characteristic of myocarditis**. Thus, **multimodal imaging techniques** should be employed to ensure accurate diagnosis and avoid unnecessary **revascularization procedures**.

Management and Prognosis

The management of **Takotsubo cardiomyopathy** is primarily **supportive**, with an emphasis on:

- **Stress reduction** (psychosocial support, relaxation therapy)
- **Medical therapy** (beta-blockers, ACE inhibitors, diuretics as needed)
- **Risk factor modification** (hypertension, dyslipidemia control)
- **Monitoring for complications** (heart failure, arrhythmias, LV thrombus)

Despite its **self-limiting nature**, **Takotsubo cardiomyopathy is not entirely benign**. Complications such as:

- **Cardiogenic shock**
- **Left ventricular outflow tract (LVOT) obstruction**
- **Arrhythmias (ventricular tachycardia, QT prolongation)**
- **Apical thrombus formation with embolization**

can occur, particularly in **elderly patients with comorbidities**.

Need for Long-Term Follow-Up

Although **most patients recover within weeks**, a subset experiences **persistent left ventricular dysfunction**, and **the risk of recurrence ranges from 1–2% per year**. Patients should be followed up with:

- **Repeat echocardiography** to assess ventricular function recovery.
- **Cardiac rehabilitation** to improve physical endurance and reduce psychological stress.
- **Holter monitoring** in cases of **QT prolongation or arrhythmic events**.

Given the **increasing recognition of Takotsubo cardiomyopathy as a STEMI mimic**, clinicians must **maintain a high index of suspicion** and integrate **comprehensive diagnostic modalities** to ensure accurate identification. Timely diagnosis prevents **unnecessary interventions**, optimizes **patient management**, and reduces **long-term morbidity**.

Final Takeaways

- **TCM should always be considered in postmenopausal women presenting with STEMI-like symptoms, particularly after emotional or physical stress.**
- **Coronary angiography is essential to rule out true ischemic events.**
- **Cardiac MRI and echocardiography are key tools for confirming the diagnosis.**
- **Management is mainly supportive but requires vigilant follow-up due to recurrence risks.**
- **Increasing awareness and research are essential to refining long-term treatment strategies for affected patients.**

With **proper diagnosis and supportive management**, most patients with **Takotsubo cardiomyopathy** recover fully, reinforcing the **importance of recognizing this unique and reversible condition**.

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