



The “Broken Heart Syndrome”: Understanding Takotsubo Cardiomyopathy

Dawn Derrick, MSN

PRIME POINTS

- “Broken heart syndrome,” or takotsubo cardiomyopathy, is a spontaneously reversible form of cardiomyopathy that is often induced by emotional or physical stress.
- Signs and symptoms include chest pain, dyspnea, electrocardiographic changes, and elevated levels of cardiac biomarkers.
- Patients often have signs and symptoms of fluid overload and can have acute pulmonary edema.
- Most patients are postmenopausal women, many with no risk factors for coronary artery disease.
- The left ventricular abnormality reverses spontaneously in days or weeks.

The damaging effects of stress on the cardiovascular system are widely documented and accepted by the general public and the medical community. A specific syndrome of stress-related reversible cardiomyopathy, however, has recently been observed with greater frequency. Increasingly referred to as the “broken heart syndrome,” this condition mimics myocardial infarction in patients without obstructive coronary artery disease. Initial signs and symptoms resemble those of acute coronary syndrome; chest pain, dyspnea, electrocardiographic (ECG) changes, and elevated levels of cardiac biomarkers are common.¹

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CE Continuing Education

This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. Describe the clinical manifestations of takotsubo cardiomyopathy.
2. Recognize the importance of prompt diagnosis and management of takotsubo cardiomyopathy
3. Understand the proposed pathological mechanisms associated with takotsubo cardiomyopathy

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It is important for critical care nurses to be aware of this syndrome and what differentiates it from classic acute myocardial infarction. Proper understanding will guide appropriate assessment, monitoring, management, and education of patients.

The hallmark of the syndrome is a characteristic transient contractile abnormality of the left ventricle causing a balloonlike morphology that can be detected with left ventricular angiography or contrast echocardiography. Damage of the left ventricle causes a contractile defect of the apex of the heart. During systole, or ventricular contraction, ventricular imaging shows a rounded, hypokinetic apex with a narrow, hypercontracted base (Figure 1). This phenomenon was first described in 1991 by Dote et al,² who named the syndrome “takotsubo-like cardiomyopathy” because the appearance resembles a pot historically used in Japan to catch octopus (*tako* in Japanese means octopus; *tsubo* means pot). The cardiac dysfunction is transient, and the left ventricle returns to normal and contractile function is restored within days to weeks.²

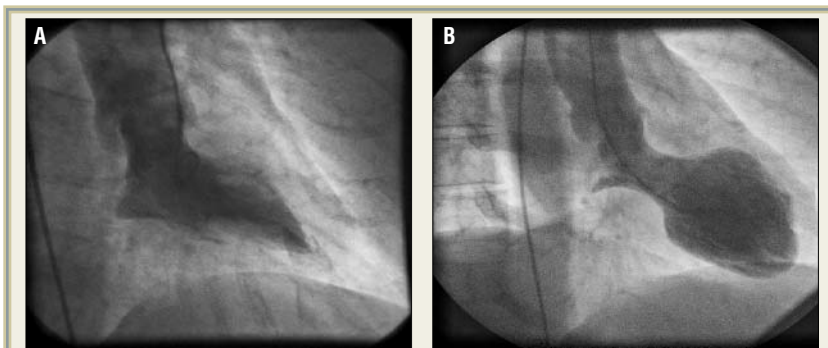


Figure 1 A, Angiogram of normal left ventricle in systole shows contraction of all myocardial segments. B, Angiogram of left ventricle with takotsubo defect shows contraction of the base with akinesis of the apex.

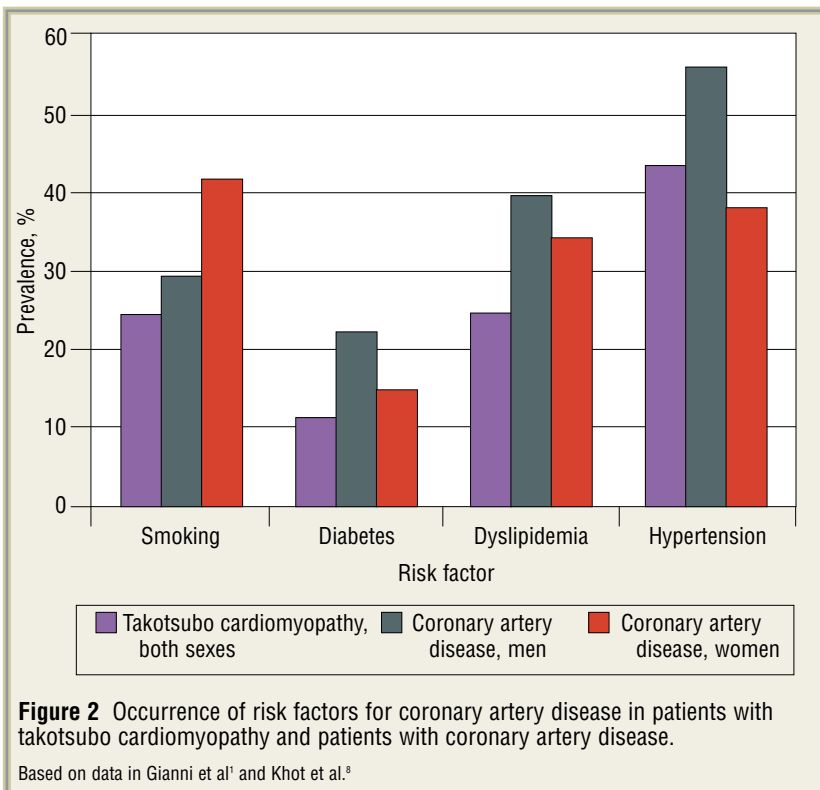


Figure 2 Occurrence of risk factors for coronary artery disease in patients with takotsubo cardiomyopathy and patients with coronary artery disease.

Based on data in Gianni et al¹ and Khot et al.⁹

Terms used to describe this syndrome in published reports include transient left ventricular

apical ballooning, stress cardiomyopathy, and ampulla cardiomyopathy.³ In this article, I use the term

Author

Dawn Derrick is an advanced practice nurse certified nurse practitioner at NorthShore University Health System in Evanston, Illinois.

Corresponding author: Dawn Derrick, MSN, Department of Cardiology, Evanston Hospital, Walgreens Building, Third Floor, 2650 Ridge Avenue, Evanston, IL 60201 (e-mail: dderrick@northshore.org).

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takotsubo [tah-ku-su-bu] cardiomyopathy. I describe the pathophysiology of takotsubo cardiomyopathy, signs and symptoms, diagnosis, treatment, and prognosis. A case report is included.

Incidence

The National Heart, Lung, and Blood Institute estimated that in 2007 in the United States, about 1.2 million people would have a myocardial infarction.⁴ Akashi et al⁵ reported that takotsubo cardiomyopathy may account for about 1% of all acute myocardial infarctions, suggesting that about 12 000 Americans might have had the syndrome in 2007. Bybee et al,⁶ in a review of the literature, reported that the syndrome accounted for 1.5% and 2.2% of Q-wave and ST-segment acute coronary syndrome, respectively, in the patients studied.

Most patients who have takotsubo cardiomyopathy are postmenopausal women.^{1,7} Gianni et al¹ reviewed 14 studies and found that of 286 patients with the syndrome, 254 (89%) were female. The mean age of the patients was 68.5 years. Of the 14 studies, 11 included data on risk factors for coronary disease. Figure 2 compares the presence of risk factors in patients with takotsubo cardiomyopathy and patients with coronary artery disease. Cardiovascular risk factors are generally present to a lesser degree in patients with takotsubo cardiomyopathy than in patients with coronary artery disease.^{1,8}

Etiology

Severe reversible left ventricular dysfunction (myocardial stunning) in patients without marked coronary disease is thought to be caused by

excessive exposure to catecholamines mediated by exaggerated sympathetic stimulation.⁷ Although this situation is known to occur in critically ill patients in the absence of coronary artery disease⁹ (Table 1), the takotsubo variant involves patients in their usual state of health, often after an episode of emotional or physical distress.

Pathophysiology

The pathophysiological mechanisms associated with takotsubo cardiomyopathy remain unclear, although broadly speculated on. The most accepted theory is catecholamine excess due to activation of sympathetic tone. Other theories considered include ischemia from multivessel coronary spasm or a transient atherosclerotic plaque.

Role of Catecholamines

Patients with takotsubo cardiomyopathy can have abnormally high serum levels of circulating catecholamines.⁷ Wittstein et al⁷ monitored plasma levels of epinephrine, norepinephrine, and dopamine in patients with takotsubo cardiomyopathy on hospital days 1 and 2. Levels were also measured in patients hospitalized with classic myocardial infarction. Patients with takotsubo cardiomyopathy had levels 7 to 34 times as high as published normal values and 2 to 3 times as high as the levels in patients with classic myocardial infarction.⁷

Histological examination of biopsy samples from the affected left ventricle of patients with takotsubo cardiomyopathy have shown contraction band necrosis, which is associated with clinical states of catecholamine excess.⁴ Catecholamine

Table 1 Causes of reversible myocardial dysfunction described in critically ill patients with no cardiac pathology^a

Neurogenic stunned myocardium
Subarachnoid hemorrhage
Stroke
Subdural hematoma
Cranial trauma
Electroconvulsive therapy
Acute respiratory failure
Upper airway obstruction
Asthma
Pulmonary embolism
Acute lung injury
Acute respiratory distress syndrome
Anaphylaxis
Trauma injuries
Pulmonary contusion
Multiple trauma
Hemorrhagic shock
Blast injury
Burn injuries
Postsurgical pathology transplant
Sepsis
Systemic inflammatory response syndrome
Pancreatitis
Cardiac arrest
Poisoning
Rhabdomyolysis
Episodes of arterial hypertension/pheochromocytoma
Thyroid disease
Arrhythmias
Hyperthermia/hypothermia
Obstructive jaundice
Emotional stress
Nutrition

^a Reprinted from Ruiz Balién,⁹ with permission.

excess has reversible toxic effects on myocardium that have been documented in cases of pheochromocytoma (a catecholamine-secreting tumor).¹⁰ In takotsubo cardiomyopathy, the apex of the left ventricle appears to be selectively vulnerable to these effects. The basal segments are spared, suggesting a difference

in sympathetic innervation or adrenergic receptor sensitivity in the apex.⁶ The exact mechanism of catecholamine-induced damage and selective apical involvement is not yet understood.

Coronary Artery Spasm

One of the first theories for the cause of takotsubo cardiomyopathy was multivessel coronary spasm.³ Coronary spasm can cause ischemia in the absence of obstructive coronary artery disease, and occlusive spasm causes transmural ischemia with ST-segment elevation.^{11(p1160)} Feasibly, multivessel coronary artery spasm could account for the diffuse takotsubo defect. Many researchers^{5,6,7,12,13} have tested the spasm theory by performing angiography during persistent ST-segment elevation and did not observe or provoke any spasm. Bybee et al,⁶ in a systematic review of 7 studies, identified 3 studies in which provokable multivessel spasm occurred in 13 of 73 patients (18%) with the syndrome who were tested. This result suggests that although spasm may play a role in takotsubo cardiomyopathy, it does not explain most cases.

Transient Occlusion by Atherosclerotic Plaque

Although obstructive coronary artery disease (>50% narrowing) is ruled out by angiography, some researchers hypothesize that a ruptured coronary plaque could be the underlying cause of takotsubo cardiomyopathy.¹⁴ Spontaneous intermittent occlusion and recanalization of coronary arteries due to a combination of thrombosis and vasoconstriction are common during early acute coronary syndrome.¹⁴ Can a

ruptured plaque cause severe or total coronary occlusion that then undergoes spontaneous recanalization before angiography?

In classic myocardial infarction, the size of the infarct corresponds to the amount of myocardium supplied by the obstructed artery. In takotsubo cardiomyopathy, the area of affected myocardium is much larger than the normal distribution of a single coronary artery. Involvement of more myocardium than could be supplied by a single artery seems to negate the possibility of transient coronary occlusion. Ibanez et al¹⁴ used intravascular ultrasound to image the LAD in 5 patients with takotsubo cardiomyopathy. Although all patients had less than 50% stenosis on angiograms, intravascular ultrasound showed ulcerated ather-

osclerotic lesions in each patient's LAD. Further evaluation of the LAD in these patients showed that the artery bent around the apex and extended along the diaphragmatic left ventricle. Ibanez et al¹⁴ proposed that in patients with a "well-developed" LAD such as those studied, widespread apical akinesis can be caused by transient occlusion; implying that takotsubo cardiomyopathy in these patients is essentially an aborted myocardial infarction.

Higher Prevalence in Women

Another confounding variable is that takotsubo cardiomyopathy occurs predominantly in postmenopausal women. Does a difference exist between the sexes in the psychological response to stress that triggers an abnormal physiological

change? Is the cardiovascular system of women physiologically or anatomically more sensitive than that of men to catecholamines? Sex hormones may influence the sympathetic nervous system and may affect coronary vasoreactivity or the tendency to spasm. Perhaps endothelial dysfunction, known to worsen after menopause (because of lowered estrogen levels) further increases vulnerability to sympathetically mediated myocardial stunning.¹

Clinical Features

Patients with takotsubo cardiomyopathy have signs and symptoms suggestive of acute coronary syndrome, usually chest pain, dyspnea, ST-segment changes on ECGs, and elevated levels of cardiac biomarkers.^{3,6,15,16} Degree of symptom severity

CASE STUDY

A 65-year-old woman with no significant medical history was brought to the emergency department because of a sudden onset of substernal chest pressure. She had been riding in a car after the funeral of a child who was a close friend of her family. The chest pressure progressed to pain reported as severe (8 on a 10-point pain scale) and constant and was associated with shortness of breath. The pain did not radiate, and she could identify no aggravating or alleviating factors.

Blood pressure was 106/50 mm Hg and oxygen saturation was 89% on room air. The patient was visibly tachypneic (respiratory rate, 32/min) and diaphoretic. Results of a physical examination were consistent with mild pulmonary edema, including jugular venous distension and crackles in the basilar lung fields. An ECG showed sinus tachycardia with a heart rate of 112/min and ST-segment elevation in leads V₂ through V₆. She was given oxygen by nasal cannula, aspirin, and sublingual nitroglycerin along with metoprolol, furosemide, and an infusion of heparin. Her pain improved to 4 (on a 10-point scale). She was taken urgently to the cardiac

catheterization laboratory approximately 1 hour after arrival in the emergency department. In the catheterization laboratory, she report dizziness, and her blood pressure was 69/30 mm Hg. A dopamine infusion was started.

Cardiac catheterization and coronary angiography were performed. All coronary arteries were patent with no marked obstruction. The left anterior descending artery (LAD) tapered at the apex but did not circumnavigate the apex (Figure 3). The left ventricle had a large, severely hypokinetic to akinetic segment involving the anterolateral, apical, and distal diaphragmatic walls. All other myocardial segments contracted normally. The estimated ejection fraction was 25%.

Hemodynamic measurements revealed an aortic pressure of 68/32 mm Hg, left ventricular pressure of 76/18 mm Hg, pulmonary capillary wedge pressure of 19 mm Hg, pulmonary artery pressure of 34/20 mm Hg with a mean of 25 mm Hg, right ventricular pressure of 41/11 mm Hg, and right atrial pressure of 9 mm Hg.

Takotsubo cardiomyopathy was diagnosed. An intra-aortic balloon pump was placed to mechanically support the left ventricle. The dopamine was discontinued to avoid further catecholamine stimulation. The

varies widely. Because of acute left ventricular dysfunction, some patients have pulmonary edema or cardiogenic shock.^{1,3,7} Onset of signs and symptoms is usually sudden and usually occurs after an emotional stressor such as the death of a loved one or a physical stressor such as an asthma attack (Table 2). A systematic review¹ of 254 patients with takotsubo cardiomyopathy indicated that 27% had an emotional stressor, 39% had a physical stressor, and 34% could not identify a stressor.

ECG Findings

The most common ECG finding in takotsubo cardiomyopathy is ST-segment elevation, typically in the precordial leads⁵ (Figure 4), but ECGs can have normal findings or show T-wave abnormalities or Q waves⁵

Table 2 Emotional and physical stressors associated with takotsubo cardiomyopathy

Emotional stressors	
Unexpected death of relative or friend ^{1,7}	
Domestic abuse ¹	
Confrontational argument ^{1,7}	
Catastrophic medical diagnosis ¹	
Devastating business ¹	
Armed robbery ⁷	
Gambling losses ¹	
Surprise party ⁷	
Surprise reunion ⁷	
Car accident ⁷	
Fear of procedure ⁷	
Fear of choking ⁷	
Court appearance ⁷	
Public performance ⁷	
Physical stressors	
Exacerbated systemic disorders ¹	
Noncardiac invasive procedures ^{1,13}	
Exhausting physical effort ^{1,5}	
Asthma attack ¹	
Pneumothorax ⁵	
Ventricular fibrillation ⁵	
Cold exposure ⁵	

(Figure 5). Evolutionary changes sometimes occur in the first few days, including resolution of the ST-segment elevation with development of diffuse and often deep T-wave inversion that involves most leads.¹⁵

Cardiac Biomarkers

Most patients with takotsubo cardiomyopathy have elevated levels of cardiac biomarkers, although usually not to the extent associated with classic myocardial infarction.⁶ In the Global Use of Strategies to Open Occluded Arteries in Acute Coronary Syndromes (GUSTO IIa) troponin substudy,¹⁷ 222 patients with classic myocardial infarction associated with ECG changes were studied. Their levels of CKMB and troponin and the degrees of elevation above the upper limit of normal

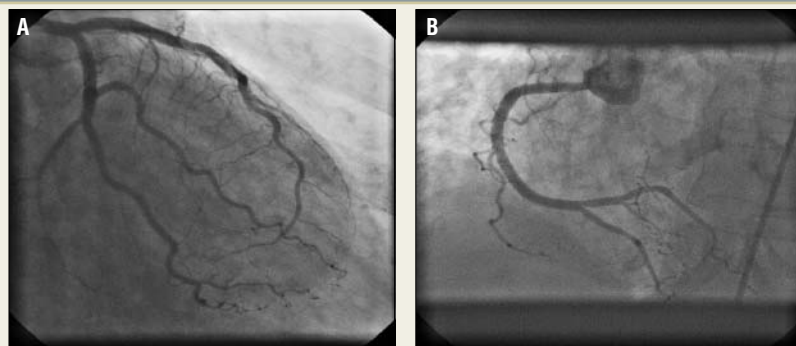


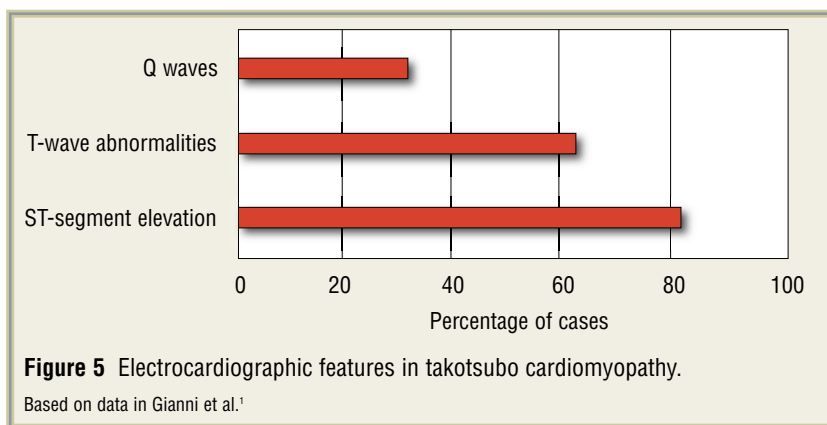
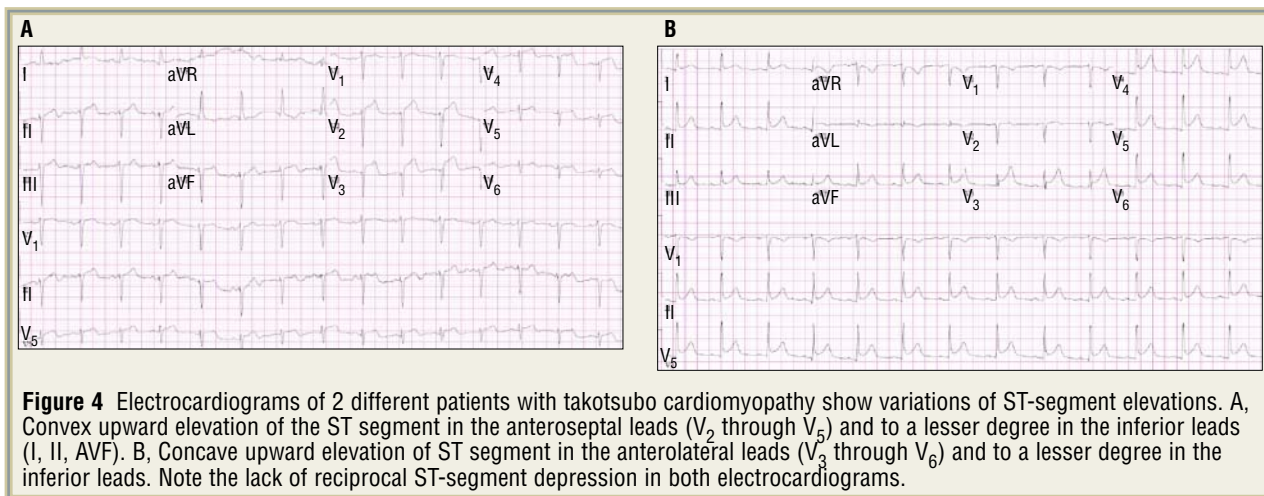
Figure 3 Angiograms of left (A) and right (B) coronary arteries in a patient with takotsubo cardiomyopathy show no obstructive coronary disease.

patient required 48 hours of support with the pump. She was monitored in the intensive care unit, where she continued to receive intravenous furosemide and had good diuresis. She had no further chest pain. Her levels of cardiac biomarkers were monitored and showed a pattern of elevation consistent with myocardial infarction. Peak levels of cardiac biomarkers occurred approximately 24 hours after her arrival in the emergency department: troponin I, 3.31 ng/mL; creatine kinase-MB fraction (CKMB), 28.3 ng/mL; myoglobin, 902 ng/mL. On the third day, she was weaned

off of the intra-aortic balloon pump. On the fourth day, she was transferred out of the intensive care unit. Her medications were changed to oral furosemide, and a β -blocker was added to her medication regimen, and when she tolerated that without hypotension, an angiotensin-converting enzyme inhibitor was added.

On the fifth day after admission, an echocardiogram showed a left ventricular ejection fraction greater than 55% with normal systolic function.

The previous contractile abnormalities were not detected, suggesting that the left ventricle had made a complete recovery. On physical examination, the patient appeared euvoletic with no clinical indications of fluid overload. Use of the diuretic and the angiotensin-converting enzyme inhibitor were stopped. The β -blocker was continued to attenuate her intrinsic catecholamine activity. After discharge from the hospital, she continued to recover and returned to her former lifestyle with no permanent limitations.



were examined. Among these patients, 95% had more than a 2-fold increase in CKMB level and more than a 3- to 11-fold increase in troponin level.¹⁷ Gianni et al,¹ in a review of 14 studies on patients with takotsubo cardiomyopathy, found 6 studies in which troponin levels were measured and 3 studies in which CKMB levels were measured. The troponin level was elevated in 106 of 123 patients (86.2%), and the CKMB level was elevated in 17 of 23 (73.9%),¹ well below the expected 95% elevation that occurs in patients with classic myocardial infarction. Kazuki et al¹³ compared 10 patients who had takotsubo cardiomyopathy with 16 patients who had classic acute coronary syndrome. The mean elevation of levels of cardiac

biomarkers in acute coronary syndrome was 10 times that in the patients with takotsubo cardiomyopathy. The mean level of CKMB (normal, 25 IU/L) in patients with takotsubo cardiomyopathy was 34 IU/L (SD, 23) compared with a mean of 326 IU/L (SD, 98) in patients with acute coronary syndrome.¹³

Diagnosis

No way currently exists to immediately distinguish the signs and symptoms of takotsubo cardiomyopathy from those of myocardial infarction caused by acute coronary thrombosis. Urgent cardiac catheterization is often performed. A diagnosis of takotsubo cardiomyopathy is suspected when obstructive coro-

nary disease is not present to explain the patient's degree of left ventricular dysfunction. Diagnosis is confirmed by observation of the typical octopus pot morphology of the left ventricle. Identification of a triggering emotional or physical stressor is considered supportive of but not necessary to the diagnosis.¹⁵ An echocardiogram must be obtained within days or weeks after the acute phase to confirm that abnormalities have reversed. Table 3 summarizes the clinical findings that lead to a diagnosis of takotsubo cardiomyopathy.

Treatment

Because takotsubo cardiomyopathy is initially indistinguishable from classic acute coronary syndrome, immediate treatment should include management of coronary ischemia and pulmonary edema. This management includes continuous telemetry monitoring and administration of aspirin, anticoagulants with direct thrombin inhibition and or glycoprotein IIb/IIIa receptor inhibition, nitrates, β -blockers, and diuretics. Once takotsubo cardiomyopathy is diagnosed, treatment is primarily supportive. Akashi et al⁵ noted complete reversal

Table 3 Clinical findings in takotsubo cardiomyopathy

Sudden onset of chest pain or shortness of breath
Precipitant physical or emotional stressor
Electrocardiographic changes suggestive of acute myocardial infarction
Mild to moderate elevation of cardiac biomarkers
Typical octopus pot morphology on echocardiogram or left ventriculogram
No evidence of obstructive coronary artery disease on cardiac catheterization
Complete resolution of left ventricular dysfunction weeks after event

of contractile abnormalities and recovery with no treatment. Aspirin can be discontinued unless coronary disease or peripheral vascular disease is concomitant. β -Blockers may be continued long-term to protect against catecholamine sensitivity, which may predispose to this syndrome. Heparin and coumadin should be used if apical thrombus is present, or a severe apical defect makes thrombus formation likely.⁶

Complications

Most complications of takotsubo cardiomyopathy occur during the acute phase of illness. Late complications are rare because the syndrome is reversible and the damage is not permanent. The reported complication rate is about 19%.¹⁶ Heart failure and pulmonary edema occur in 3% to 46% of patients, and mortality rates are 1%^{6,17} to approximately 3%.¹⁶ Reported complications are summarized in Table 4.

Heart Failure

Patients with left-ventricular failure can require mechanical support with intra-aortic counterpulsation by balloon pump during the acute phase. Adrenergic agents are usually avoided to prevent additional catecholamine stimulation. Diuretics

are often helpful for pulmonary edema and fluid overload. Standard therapy for left ventricular dysfunction should be initiated, including use of angiotensin-converting enzyme inhibitors and β -blockers.

Thrombus Formation

Heparin and warfarin may be used to treat or prevent left ventricular apical thrombus, which is a risk of significant left ventricular dysfunction. Apical thrombi form because of stasis of blood in the akinetic segments. This same protocol is used to prevent thrombus in patients with ventricular aneurysms as a complication of classic myocardial infarction.¹⁹ Warfarin can be discontinued once ventricular function has returned to normal.⁶

Table 4 Reported complications in takotsubo cardiomyopathy

Left ventricular failure with or without pulmonary edema ^{1,6,16}
Cardiogenic shock ^{6,16}
Left ventricular outflow tract obstruction ^{1,6}
Mitral regurgitation from "systolic anterior motion" ⁶
Ventricular arrhythmias ⁵
Transient complete atrioventricular block ¹⁸
Left ventricular mural thrombus ^{6,16}
Left ventricular free wall rupture ⁶
Death ⁶

Left Ventricular Outflow Tract Obstruction

Patients with takotsubo cardiomyopathy are at risk for obstruction of the left ventricular outflow tract (LVOT).¹ The LVOT is the path that blood takes along the septal wall as the blood is being ejected from the left ventricle through the aortic valve. Obstruction of this pathway is caused by exaggerated contraction of the base of the ventricle when hypokinesis of the apex occurs. The unbalanced contraction of the ventricle reduces the size of the outflow tract, resulting in acceleration of blood flow through this pathway during systole. Accelerated flow decreases the pressure above the neighboring mitral valve, causing a suctioning effect of the anterior mitral valve leaflet toward the septum. This "systolic anterior motion" of the mitral valve toward the septum causes further LVOT obstruction,²⁰ which can cause hypotension. Echocardiography or catheterization of the left side of the heart is used to diagnose this complication. The hypotension of LVOT obstruction can be managed in several ways. β -Blockers can reduce the hypercontractility of the base

and lessen the degree of obstruction. β -Blockers also increase ventricular filling during diastole. Reduction in pressure gradient between the left ventricular apex and outflow tract has been observed

after intravenous injection of propranolol.³ Increased blood volume increases the functional size of the outflow tract and reduces the obstruction.²⁰ Intravenous fluids can be used to increase volume if the patient does not have pulmonary edema.

Prognosis

Overall, without significant comorbid diseases, prognosis for patients with takotsubo cardiomyopathy is good once the acute phase has passed. Recommendations for follow-up include echocardiography at approximately 4 to 6 weeks after discharge to document normalization of left ventricular function. Complete resolution of contractile abnormalities within weeks is characteristic of this syndrome, so another diagnosis should be considered for patients with persistent contractile abnormalities.

Reoccurrence of takotsubo cardiomyopathy is considered rare and has been reported in no more than 10% of cases.¹⁵ Gianni et al¹ identified 4 studies documenting a mean recurrence rate of 3.5%. However, the syndrome has been documented for a relatively short time, and so the natural history remains largely unknown. Patients should be followed up regularly as outpatients.

Nursing Implications

Takotsubo cardiomyopathy is an important health issue involving postmenopausal women without

severe coronary artery disease and often without classic risk factors for coronary artery disease. Patients are affected suddenly, with no warning, often at a time of crisis in their life. Health care providers should be prepared to provide education and counseling to patients with this unique diagnosis. Takotsubo cardiomyopathy should be considered when patients have signs and symptoms of acute coronary syndrome and appear to be at low risk for coronary artery disease, especially if a stressful event occurred before the onset of the signs and symptoms.

Nursing care of patients with takotsubo cardiomyopathy involves monitoring hemodynamic status, providing supportive measures, and watching for complications. Nurses need a thorough understanding of the nature of the syndrome and what distinguishes it from classic myocardial infarction. Education of patients and their families about the nature of the syndrome, including its reversibility and low rate of recurrence, is imperative. Expectation for a full recovery will be reassuring to patients and their families. Assessing effective coping is important, especially if a major emotional stressor was the precipitant of the syndrome. The added stress of being ill and hospitalized requires continued psychological support. Patients must be educated on the importance of outpatient follow-up for repeat echocardiography to confirm resolution.

If mild or moderate coronary disease was detected during angiography, the patient should also be counseled on risk-factor modification for coronary artery disease.

Implications for Future Research

Further research is needed to clarify the pathophysiology of takotsubo cardiomyopathy, especially in relationship to women. Also important is distinguishing any potentially modifiable risk factors. Additionally, future research should focus on techniques for differentiating takotsubo cardiomyopathy from classic acute coronary syndrome and on establishing standardized criteria for diagnosis and clinical guidelines for treatment and follow-up. **CCN**

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Financial Disclosures
None reported.

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To learn more about takotsubo cardiomyopathy, read "Transient Left Ventricular Apical Ballooning," by Brenda McCulloch in *Critical Care Nurse* 2007;27(6):20-27. Available at www.ccnonline.org.

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CE Test Test ID C0913: The "Broken Heart Syndrome": Understanding Takotsubo Cardiomyopathy

Learning objectives: 1. Describe the clinical manifestations of takotsubo cardiomyopathy 2. Recognize the importance of prompt diagnosis and management of takotsubo cardiomyopathy 3. Understand the proposed pathological mechanisms associated with takotsubo cardiomyopathy

1. How is takotsubo cardiomyopathy best described?
a. Stress-related reversible cardiomyopathy
b. Ventricular dilatation and grossly impaired systolic function
c. Asymmetrical septal hypertrophic cardiomyopathy
d. Reduced diastolic compliance of the left ventricle
2. What do the initial signs and symptoms of takotsubo cardiomyopathy most closely resemble?
a. Pulmonary embolism
b. Thoracic aortic aneurysm
c. Acute coronary syndrome
d. Pericarditis
3. What does systolic ventricular imaging demonstrate in a patient with takotsubo cardiomyopathy?
a. Rounded, hypokinetic apex with a narrow, hypercontracted base
b. Rounded, hyperkinetic apex with a narrow, hypocontracted base
c. Narrow, hypokinetic apex with a rounded, hypercontracted base
d. Narrow, hyperkinetic apex with a rounded, hypocontracted base
4. What is another name for takotsubo cardiomyopathy?
a. Dilated cardiomyopathy
b. Hypertrophic cardiomyopathy
c. Restrictive cardiomyopathy
d. Ampulla cardiomyopathy
5. Which group of patients has the greatest incidence of takotsubo cardiomyopathy?
a. Postmenopausal women
b. Adolescent girls
c. Perimenopausal women
d. Pregnant women
6. What is the leading source of morbidity, cost, and legal ramifications after cardiac catheterization?
a. Multivessel coronary artery spasm
b. Catecholamine excess
c. Transient atherosclerotic plaque
d. Obstructive coronary artery disease
7. Which of the following is correct about takotsubo cardiomyopathy?
a. The onset of signs and symptoms is usually insidious.
b. Electrocardiographic changes typically involve limb leads.
c. Signs and symptoms usually occur after an emotional stressor.
d. Cardiac biomarkers are usually normal.
8. What is the most common electrocardiography finding in takotsubo cardiomyopathy?
a. ST-segment depression
b. T-wave inversion
c. ST-segment elevation
d. Pathological Q waves
9. What drug may be continued long term to protect against catecholamine sensitivity?
a. Alpha adrenergic blockers
b. Calcium channel blockers
c. Angiotensin-converting enzyme inhibitors
d. Beta adrenergic blockers
10. What is correct about the complications of takotsubo cardiomyopathy?
a. Late complications are frequent.
b. Most complications occur during the acute phase of illness.
c. The reported complication rate is about 3%.
d. Pulmonary edema occurs in 64% of patients.
11. Accelerated blood flow through the left ventricular outflow tract decreases the pressure above which valve?
a. Tricuspid
b. Mitral
c. Pulmonic
d. Aortic
12. What is recommended at approximately 4 to 6 weeks after discharge to document normalization of left ventricular function in a patient diagnosed with takotsubo cardiomyopathy?
a. A multigated acquisition scan
b. Coronary angiography
c. Exercise stress testing
d. Echocardiography

Test answers: Mark only one box for your answer to each question. You may photocopy this form.

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