A broken heart classically refers to an emotional state caused by psychological pain. Current research indicates that a broken heart is a true medical condition, not just a play on words. Broken heart syndrome is due to an exaggerated sympathetic response to stress that causes a severe but reversible left ventricular failure. Takotsubo cardiomyopathy is a reversible left ventricular dysfunction triggered by sudden stress in persons with no history of coronary artery disease.

Signs and symptoms of this cardiomyopathy often mimic those of acute coronary syndrome, making differential diagnosis challenging. Patients characteristically have chest pain, increased troponin levels, and ST-segment elevation. To increase understanding of this unique cardiomyopathy, we review a case study of a patient with the condition and the diagnosis, management, and complications during her stay in the intensive care unit (ICU).

The term takotsubo was introduced in 1991 by Dote et al to describe the shape of the left ventricle during an episode of sudden-onset cardiomyopathy. Takotsubo is a Japanese term for a narrow-necked fishing pot used to catch octopus. Tako means octopus, and tsubo means pot. The shape of this fishing pot is the form the left ventricle assumes during takotsubo cardiomyopathy (Figure 1). The anterior of the left ventricle resembles the neck of the pot; the posterior or apical section of the ventricle dilates outward in a balloonlike shape, forming the base of the pot.

Studies from large heart centers indicate a mean of 1 case of takotsubo cardiomyopathy per month in patients admitted because of acute myocardial infarction. Japan, Germany, the United States, Europe, Italy, and Belgium all reported an increase in documented cases of takotsubo cardiomyopathy. Favorable clinical outcomes in patients with takotsubo cardiomyopathy are possible in most cases and require early recognition and prompt treatment.

Interpreting the ECGs of patients with takotsubo cardiomyopathy is a challenge because multiple distinct findings are possible.

For patient management, use the common guidelines for ST-elevation acute myocardial infarction.
cardiomyopathy require improvement in early recognition of the condition and prompt treatment. Favorable clinical outcomes are possible in most cases. Mortality rates vary from 2% to 6%. Like many patients with acute coronary syndromes, patients with takotsubo cardiomyopathy can often be medically managed with minimal interventions. Severe hemodynamic compromise develops in 37% of patients with takotsubo cardiomyopathy and requires acute care and multiple interventions.

In a retrospective study of 16,989 patients in Hamburg, Germany, Hertting et al reviewed diagnostic angiograms obtained during a 3-year period. The angiograms indicated 32 patients in whom a ventriculogram alone revealed takotsubo cardiomyopathy. In these patients, median ejection fraction during the acute phase (defined as the onset of reduced cardiac output with electrocardiographic [ECG] changes, chest pain, and hypotension) was 42%. After treatment, the median ejection fraction increased to 70%. A total of 44% of the 32 patients had chronic obstructive pulmonary disease (COPD). A total of 78% had clinical signs and symptoms of acute myocardial infarction, with positive results for troponin T in 63%. Among the 32 patients, 40% had experienced a markedly stressful event before the onset of signs and symptoms.

Case studies indicate that 94% of patients with takotsubo cardiomyopathy are women of postmenopausal age with no history of severe cardiac problems. Reported cases involving males are rare, about 1 to 2 patients per study, and provide limited data. Multiple studies have been conducted in Europe and the United States, but the majority of information comes from Japan. Of the women involved, 60% were Asian and had a mean age of 70 years; 40% were white and had a mean age of 64 years.

A prospective study of 22 women who had a diagnosis of takotsubo cardiomyopathy was conducted in the Minneapolis-St Paul, Minnesota area. The patients had signs and symptoms consistent with acute myocardial infarction such as chest pain, ST-segment elevation, and cardiogenic shock. In addition to these signs and symptoms, each had experienced a stressful incident, such as the recent death of a spouse, preceding the cardiac event. Hemodynamic compromise requiring interventions such as placement of an intra-aortic balloon pump (IABP) and/or administration of vasopressor agents developed in 37% of the patients. All 22 patients survived.

**Description**

Cardiomyopathy is due to weakening of the heart muscle or to an abnormality of the heart structure. This progressive disease process reduces the ability of the heart to pump blood effectively. Traditionally, cardiomyopathy is classified as ischemic or nonischemic. Ischemic cardiomyopathy is precipitated by coronary artery disease and/or prior myocardial infarctions. Nonischemic cardiomyopathy is categorized as hypertrophic, dilated (congestive), or restrictive. Each term refers to the type of muscle damage involved. Cardiomyopathy often develops as

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the heart muscle becomes abnormally thick, rigid, or scarred. These changes then progress to irreversible heart failure.

In takotsubo cardiomyopathy, rapid severe weakness of the heart muscle develops into cardiomyopathy. Treatment often requires short-term options to reduce contributory conditions and relieve signs and symptoms. Therapies may include medical management, use of a ventricular assist device (eg, an IABP or a percutaneous device), or placement of a temporary pacemaker. The goal of care is to prevent further progression of cardiomyopathy. Takotsubo cardiomyopathy is a stress-induced cardiomyopathy that is self-limiting, resolving, and not prone to recurrence.1-8,10,12-15 Although takotsubo cardiomyopathy is potentially life threatening, if it is treated appropriately, even the most critically ill patient can make a quick and complete recovery.

Etiology
Takotsubo cardiomyopathy appears to be precipitated by an intense emotional or physical condition.1,4,10-12 This intense response may be caused by extreme stress triggered by emotional reactions.1,4,6 Emotional stressors can include a recent death in the family, a catastrophic medical diagnosis such as cancer, terminal illness, or a life-changing treatment. Physical conditions such as exacerbation of COPD or fear related to domestic abuse have been documented as stressors.1,2 Environmental factors such as earthquakes or other destructive weather conditions are also examples of recognized triggers.3 Additional documented stressors include intense emotions stimulated during psychotherapeutic treatments, excessive gambling, and tension related to financial difficulties.1,4

Compared with a physical condition stressor, an emotional trigger occurs in twice as many cases of takotsubo cardiomyopathy.5 Premenopausal women less than 50 years old seem to have increased responses to emotional triggers, whereas postmenopausal women more than 50 years old have increased responses to physical triggers.5

Pathophysiology
The exact mechanism of takotsubo cardiomyopathy remains unknown. It is suspected that high circulating levels of catecholamines produce myocardial stunning and wall motion abnormalities in the heart. Stress activates the classic sympathetic response of fight or flight, producing rapid release of catecholamines. The key marker of takotsubo cardiomyopathy is the inappropriate decreased responsiveness of the apical myocardium to catecholamine overload.2,24 For unknown reasons, the apex of the heart relaxes and dilates, leading to a negative inotropic response to the increased catecholamine levels.2,7 This response results in decreased cardiac contractility and grossly reduces the ability of the heart to pump and circulate blood volume. The left ventricular outflow tract responds appropriately to the catecholamine overload by narrowing, a change that obstructs blood flow. When this change is coupled with the balloon-shaped apex of the left ventricle, it becomes difficult for the ventricle to fully empty. Consequently, blood pools and increases left ventricular end-diastolic pressure. As volume in the heart increases, tension against the ventricular wall increases. The increased tension leads to a decreased contractile state, reducing cardiac output and increasing myocardial oxygen demands. Increased oxygen demand alters the demand to supply ratio. As this ratio increases, more myocardial oxygen is needed. The oxygen demand of the heart quickly exceeds the body’s capability. Aerobic metabolism soon ceases, causing the body to rely on anaerobic metabolism in an attempt to meet the body’s needs. Anaerobic metabolism soon generates increased lactate, as indicated by elevated blood lactate levels. The increased lactate level is an indicator of tissue hypoxia.

In a study by Goldstein et al,20 baseline levels of the catecholamine epinephrine normally circulating in the body without injury were 37 pmol/L. In contrast, catecholamine levels are about 376 pmol/L in patients with acute myocardial infarction and about 1264 pmol/L in patients with takotsubo cardiomyopathy. Epinephrine also triggers the hepatic release of glucagon, which is broken down into glucose,20 and decreases the production of insulin by suppressing the α-adrenergic response. With less active insulin to break down glucose, a hyperglycemic state develops.

The main source of epinephrine is the adrenal medulla. During the stress response, the medulla releases epinephrine, which normally causes increased myocardial contraction and a positive chronotropic effect. Noncardiac effects are venous vasodilatation, increasing preload,
and skin constriction that augments central blood flow.

Shock is defined as a condition in which the cardiovascular system fails to perfuse tissues adequately. During takotsubo cardiomyopathy, the catecholamine overload causes the left ventricle to become overwhelmed, resulting in severe spasm of the heart muscle. Blood remains in the ventricle after ejection, reducing circulating volume and increasing the risk for clots. Clots can quickly develop into a large thrombus, and small clots can enter the circulation and cause embolism. The longer cardiac tissue remains poorly oxygenated, the greater is the chance that areas of cardiac necrosis may develop. Such necrosis can sometimes form a ventricular septal defect or a ventricular rupture.

Patients with COPD or an enlarged ventricle in the area of ballooning may have an increased risk for cardiac arrhythmias. Both COPD and an enlarged ventricle often are associated with an increased QT segment, reflecting the increased time the ventricle requires to depolarize and then repolarize. A normal QTc is less than 460 ms. A QTc greater than 460 ms indicates that the ventricle is taking a prolonged time to repolarize, increasing the possibility of R-on-T phenomenon and the development of ventricular tachycardia and torsades de pointes.

Complications

Complications occur in about 18.4% of cases of takotsubo cardiomyopathy. Common complications are shock, thrombus formation, heart failure, cerebral vascular accident, and ventricular tachycardia. More severe complications include mechanical issues such as left ventricular rupture and/or ventricular septal rupture. Race or ethnic background may play a role in mortality. Donohue et al. reported a 6% chance of death among Asian women and a 1.7% chance among white women. However, in that study, the Asian women were a mean of 8 years older than the white women.

Diagnostic Studies

Electrocardiography

Interpreting the ECGs of patients with takotsubo cardiomyopathy is a challenge because multiple distinct findings are possible. ST-segment and T-wave inversion are indicators of myocardial ischemia. Because prolonged ischemia can lead to myocardial injury, a rapid diagnosis is required to identify takotsubo cardiomyopathy as the cause.

The following examples are 3 common ECG patterns found in takotsubo cardiomyopathy:

1. Convex ST-segment elevation in leads V1 through V3 (Figure 2)
2. T-wave inversion in all leads, with the largest inversions in V3, V4, and V5 (Figure 3)
3. Q waves in leads V2 through V6 (Figure 4)

A common ECG finding in most patients with takotsubo cardiomyopathy is a prolonged QTc greater than 460 ms.

ECG findings also differ between white and Asian women. White women usually have T-wave inversion,
whereas Asian women predominantly have ST-segment elevation.

**Laboratory Values**

Serum troponin levels (normal, <0.1 ng/mL) are useful in the diagnosis of acute myocardial infarction, and they also play a key role in the diagnosis of takotsubo cardiomyopathy. Elevations in troponin I in patients with takotsubo cardiomyopathy are mild (up to 14 ng/mL) compared with those in patients with acute myocardial infarction (0-125 ng/mL), but the degree of elevation is directly related to the severity of the cardiomyopathy. Decreases in troponin I levels indicate that the syndrome is resolving. The peak phase should occur within 3 days of the onset of symptoms. Once decreases in troponin I levels are noted, catecholamine levels probably are also decreasing.

**Cardiac Imaging**

Various imaging techniques can be used to detect the abnormality in the left ventricular cavity in takotsubo cardiomyopathy. Common methods include noninvasive transthoracic echocardiography and invasive transesophageal echocardiography. Cardiac magnetic resonance imaging and percutaneous coronary angiography may also be used. Figure 1 shows a...
ventriculogram of a patient with takotsubo cardiomyopathy with normal left ventricular filling during diastole. During systole the narrowed neck and the apex balloon deformity are apparent. Figure 5 shows magnetic resonance images of the left ventricle during acute and resolved phases of Takotsubo cardiomyopathy. Part A shows the narrowed neck and apex balloon deformity of the acute phase; part B shows resolution of the cardiomyopathy.

**Echocardiography**

Ejection fraction can be measured by using either echocardiography at the bedside or angiography in the cardiac catheterization laboratory. Ejection fractions are often decreased by 15% to 40% from baseline during the acute phase of takotsubo cardiomyopathy. After recovery, ejection fractions are usually normal.

**Diagnostic Criteria for Takotsubo Cardiomyopathy**

The Mayo Clinic has developed 4 diagnostic criteria for the diagnosis of takotsubo cardiomyopathy:

1. New ECG findings (not evident on previous ECGs or are acute changes) such as T-wave inversion or ST-segment changes
2. Absence of any coronary artery disease
3. Transient akinesis of the left ventricular apical and midventricular segment, including wall motion abnormality
4. All other possible causes of the changes have been ruled out (eg, head trauma, intracranial bleeding, pheochromocytoma, coronary occlusion, myocarditis, hypertrophic cardiomyopathy)

**Patient Management**

No guidelines currently exist for management of patients with takotsubo cardiomyopathy. Widely held practice is to use the common guidelines for ST-elevation acute myocardial infarction. These guidelines include use of oxygen, aspirin, nitroglycerin, β-blockers, and heparin. Medications such as morphine for pain control, nitrates for vasodilatation, and inotropes for improved contractility all play important roles in management of patients with takotsubo cardiomyopathy. Vasopressors along with an IABP may be needed to support hemodynamic status. The use of stents, thrombolitics, and long-term coronary heart disease medications should be excluded from treatment of patients with takotsubo cardiomyopathy once the diagnosis has been made.

**Case Study**

**Pre-ICU**

A 55-year-old white woman was admitted to the hospital for mediastinal lymph node biopsy, which required general anesthesia. She had a history of severe COPD and emphysema and had chronic atrial fibrillation that was treated with warfarin sodium.

Preadmission workup included a 12-lead ECG (Figure 6), which showed normal sinus rhythm with a rate of 87/min; first-degree atrioventricular block with a PR interval of 206 ms (normal, 120-200 ms); and a prolonged QTc interval of 558 ms (normal, <460 ms), possibly...
related to the COPD. She was highly anxious about her current health and apprehensive about the biopsy.

She tolerated the planned procedure, but respiratory distress developed after extubation (see Table). Arterial blood gas analysis of a sample obtained while she was receiving 100% oxygen indicated a severely acidemic state, with pH 6.92, PaCO₂ 115 mm Hg, and PaO₂ 77 mm Hg (bicarbonate level was not reported). The patient required reintubation.

ECG showed a normal sinus rhythm of 60/min; blood pressure was

![Figure 6](image) Preoperative electrocardiogram shows a normal sinus rhythm with a PR interval of 206 ms and a QTc of 558 ms.

<table>
<thead>
<tr>
<th>Table</th>
<th>Vital signs, results of laboratory tests, and intravenous medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (OR)</td>
<td>B (CTICU)</td>
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<tr>
<td>Heart rate, beats per minute</td>
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</tr>
<tr>
<td>Rhythm</td>
<td>Normal</td>
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<tr>
<td>Blood pressure, mm Hg</td>
<td>65/30</td>
</tr>
<tr>
<td>Mean arterial pressure, mm Hg</td>
<td>42</td>
</tr>
<tr>
<td>Central venous pressure, mm Hg</td>
<td>13</td>
</tr>
<tr>
<td>Laboratory tests</td>
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<tr>
<td>Arterial blood gas analysis</td>
<td></td>
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<tr>
<td>pH</td>
<td>6.92</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>115</td>
</tr>
<tr>
<td>PaO₂</td>
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<tr>
<td>Bicarbonate, mmol/L</td>
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<td>Glucose, mg/dL</td>
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<td>Troponin, ng/mL</td>
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<tr>
<td>Lactate, mmol/L</td>
<td>7</td>
</tr>
<tr>
<td>Medications</td>
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<tr>
<td>Dobutamine, µg/kg per minute</td>
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<tr>
<td>Norepinephrine, µg/kg per minute</td>
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<td>Propofol, µg/kg per minute</td>
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<tr>
<td>Sodium bicarbonate, mEq/h</td>
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<tr>
<td>Amiodarone, mg/min</td>
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<tr>
<td>150-mg bolus</td>
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<tr>
<td>Epinephrine, µg/kg per minute</td>
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</tr>
<tr>
<td>Insulin, units/h</td>
<td>2</td>
</tr>
<tr>
<td>Milrinone, µg/kg per minute</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: AF, atrial fibrillation; cath lab, catheterization laboratory; CTICU, cardiothoracic intensive care unit; NA, not applicable; OR, operating room.
65/30 mm Hg. A triple-lumen central venous catheter was placed for vascular access. A portable chest radiograph showed no evidence of pneumothorax. The ECG tracing showed ST-segment elevation in lead V. Transthoracic echocardiography revealed severe left ventricular dysfunction. Propofol was started at 30 μg/kg per minute for sedation. Computed tomography of the head showed no evidence of cerebral hemorrhage. The patient was admitted into the cardiothoracic ICU.

**Cardiothoracic ICU**

After the patient’s arrival in the cardiothoracic ICU, a radial arterial catheter was placed for blood pressure monitoring. Vital signs at that time are given in the Table. Acute myocardial infarction was suspected because of the ST-segment changes and the results of the transthoracic echocardiography done in the operating room. Her serum level of troponin was elevated at 0.3 ng/mL (normal, 0-0.2 ng/mL). Another 12-lead ECG was obtained (Figure 7). This ECG showed atrial fibrillation, with ST-segment depression in the inferior leads and ST-segment elevation in V2 through V4. Q waves were also present in V1 through V3. The QTc was 549 ms. A strain pattern (Figure 8) was present in leads II and III. This strain pattern occurs when the QRS-T angle widens and there is a down slope from the R wave to the T wave in the opposite direction from the QRS. This pattern occurs in left ventricular enlargement and is associated with myocardial ischemia.23 Consultation with a cardiologist was requested in an attempt to identify the cause of the acute deterioration in the patient’s hemodynamic status.

Fluid replacement with 250 mL of 5% albumin was used as a first-line volume expander. The goal was to improve preload. This volume helps determine the force of contraction during systole and the effect of the contraction on cardiac output.

Simultaneously, a dobutamine infusion was started, at a rate of 5 μg/kg per minute, as first-line inotropic support. A repeat bolus of 250 mL of 5% albumin was given to further increase preload and improve cardiac output. The patient was not responsive to fluid replacement. The systolic blood pressure remained less than 90 mm Hg.

Phenylephrine hydrochloride was chosen for first-line vasopressor support, and several boluses were given with little effect. An infusion of the second-line vasopressor norepinephrine was started at a rate of 0.04 μg/kg per minute. Dobutamine provided no improvement in the patient’s vital signs and was replaced with an epinephrine infusion at a rate of 0.05 μg/kg per minute.

Because of the severe respiratory acidosis (pH 7.14), a sodium bicarbonate infusion, 30 mEq/h was started for buffering until the respiratory acidosis could be corrected. A solution of 5% dextrose was used.

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**Figure 7** Postoperative electrocardiogram shows a Q wave in V1 through V3, ST-segment depression in inferior leads, ST-segment elevation in V2, and ventricular slurring in II and III. QTc is 549 ms.

**Figure 8** Slurring pattern is apparent in the RS segment and the S-T segment in lead II. The R wave slopes down slightly to the right, with a deep S-wave ending. The S wave slopes up at an angle up into the peaked T wave. This concave dipping in the S wave is consistent with ventricular strain pattern.
for the bicarbonate infusion, and the infusion rate was 150 mL/h. This infusion may have further elevated the blood glucose level, which was 304 mg/dL (to convert to millimoles per liter, multiply by 0.0555). An infusion of regular insulin was started for glucose control, with a goal of 90 to 120 mg/dL for blood glucose. At this time the cardiologist decided that cardiac catheterization was needed emergently to obtain angiograms and to open any occluded vessels to reduce damage due to the suspected myocardial infarction. Vital signs before the catheterization are given in the Table.

Cardiac catheterization showed no coronary artery blockage. A left ventriculogram revealed severe left ventricular dysfunction with an apical balloon deformity and an ejection fraction of 20%. The diagnosis was takotsubo cardiomyopathy. Now the goal was to reduce afterload. A reduction in afterload would improve contractility and allow increased tissue perfusion. Use of an IABP is currently a standard for reducing afterload in patients with cardiogenic shock. An IABP was placed in the right femoral artery. The epinephrine infusion was increased to 0.09 μg/kg per minute to improve left ventricular ejection.

When the patient returned to the ICU, her vital signs had stabilized (see Table). The patient’s right foot had no pulse and was cold to the touch. A vascular consultation was obtained to assess her leg. The decision was made to remove the IABP.

The patients’ hemodynamic status was further compromised when rapid atrial fibrillation developed, with pulse rates in the 140/min range (see Table). A biphasic synchronized cardioversion at 30 J was performed and then a second one at 150 J. Neither cardioversion was successful in converting or slowing the pulse rate; therefore, a 150-mg bolus of amiodarone was administered (see Table) followed by a maintenance infusion at 1 mg/min.

It was decided to avoid further invasive procedures, such as placing a pulmonary artery catheter. Venous oxygenation saturation, measured with a pulmonary artery catheter, would have been the best choice as an indicator of perfusion. The next best indicator of perfusion is level of lactate. Analysis of a blood sample indicated a lactate level of 7 mmol/L (normal, 1.7-2.1 mmol/L). Elevated lactate levels indicated that the tissues were not receiving adequate perfusion and anaerobic metabolism had started. The hypoperfusion was related to the patient’s poor ejection fraction of 20%.

An infusion of milrinone 0.25 μg/kg per minute was started as an adjunct inotropic agent to improve contractility and decrease afterload (see Table). The main goal of all therapy was to increase tissue perfusion. Serial measurements of lactate levels allowed continued evaluation of perfusion.

The next measured lactate level was 6.4 mmol/L, reflecting only a 0.6 decrease over the previous 5 hours. The milrinone was increased to 0.5 μg/kg per minute, resulting in further enhancement of contractility and a reduction in afterload (see Table). The patient’s vital signs improved with the infusion in use...
Chemotherapy treatments were started, and the takotsubo cardiomyopathy has not recurred.

Summary

Our patient had risk factors typical of patients with takotsubo cardiomyopathy. She was a woman more than 50 years old, had a catastrophic medical diagnosis (cancer was confirmed with the surgical biopsy), and had documented prolonged QTc and COPD. When the cardiomyopathy occurred, causative factors such as pneumothorax, intracranial bleeding, and coronary artery occlusion were ruled out. Treatment was aggressive without placement of a pulmonary artery catheter. Vital signs and laboratory results were reassessed after each intervention. The goal of treatment was tissue oxygenation, which was evaluated by monitoring the lactate level to achieve a goal of less than 2 mmol/L. Monitoring of laboratory levels was essential to evaluate use of intravenous inotropic support. This type of cardiomyopathy had not been witnessed in our cardiothoracic ICU before. Information obtained through Internet searches was used to help guide treatment. By analyzing this case, we learned that when this cardiomyopathy occurs, the consequences can be severe or even fatal if it is not diagnosed and treated promptly.

Since this case, we have had a second patient with takotsubo cardiomyopathy, whose clinical features were much the same as those of the first patient. Milrinone, which inhibits the enzyme phosphodiesterase, had an inotropic effect as well as a vasodilating effect, but without the catecholamine effects. Afterload was reduced, and serum lactate levels

Figure 9  Electrocardiogram obtained on postoperative day 1 shows normal sinus rhythm. Strain pattern is absent, ST-segment changes have resolved, and QTc is 402 ms.
decreased. Because the second patient had a milrinone infusion started early, a second remarkable recovery occurred. If the risk factors and signs and symptoms of takotsubo cardiomyopathy are recognized and treatment is prompt, the clinical outcome is favorable.19 CCN

Financial Disclosures
None reported.

References
1. Why is the term “takotsubo” used to describe sudden onset cardiomyopathy?
   a. This octopus-fishing pot resembles the shape the left ventricle assumes during an episode.
   b. This disease has 8 symptoms, much like the legs of the octopus.
   c. This phenomenon was first described by Dr. Takotsubo, a Japanese physician.
   d. This disease was first diagnosed in a small octopus fishing village in Japan.

2. According to a retrospective study of patients in Hamburg, Germany, which of the following statements is true about patients with takotsubo?
   a. 68% had a myocardial infarction
   b. 32% had just experienced a stressful event
   c. 40% had chronic obstructive pulmonary disease
   d. 30% had acute respiratory failure

3. Which of the following types of cardiomyopathy is triggered by an acute myocardial infarction?
   a. Ischemic
   b. Hypertrophic
   c. Dilated
   d. Restrictive

4. What is takotsubo cardiomyopathy?
   a. Stress-induced cardiomyopathy that requires long-term therapy
   b. Stress-induced cardiomyopathy that is prone to recurrence
   c. Stress-induced cardiomyopathy that occurs rapidly
   d. Stress-induced cardiomyopathy that causes scarring of the heart muscle

5. When is a postmenopausal woman more likely to develop takotsubo cardiomyopathy?
   a. After an exacerbation of chronic obstructive pulmonary disease
   b. After a death in the family
   c. After an earthquake
   d. After financial difficulties

6. Which of the following is responsible for the decreased response to catecholamine overload that occurs in takotsubo cardiomyopathy?
   a. The apex of the heart relaxes and constricts
   b. The apex of the heart relaxes and dilates
   c. The apex of the heart contracts and dilates
   d. The apex of the heart expands and constricts

7. Which of the following causes the increase in lactate in patients with takotsubo cardiomyopathy?
   a. Decreased myocardial oxygen demand and increased oxygen supply
   b. Decreased myocardial oxygen demand and decreased oxygen supply
   c. Increased myocardial oxygen demand and increased oxygen supply
   d. Increased myocardial oxygen demand and decreased oxygen supply

8. Which of the following is a common electrocardiographic finding in a patient with takotsubo cardiomyopathy?
   a. Convex ST elevation in V4 to V6
   b. T wave inversions in V1 to V3
   c. QTc greater than 460 ms
   d. PR greater than 460 ms

9. In takotsubo cardiomyopathy patients, what do decreases in serum troponin I levels indicate?
   a. Increased myocardial damage
   b. Reinfarction of a myocardial infarction
   c. Decreased release of catecholamines
   d. The precipitating event was less than 3 days ago

10. The guidelines for treating a patient with takotsubo cardiomyopathy are similar to those used to treat patients with which of the following?
    a. ST depression
    b. ST elevation
    c. Acute ischemic stroke
    d. Chest pain without electrocardiographic changes

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

1. a  2. b  3. a  4. a  5. a  6. b  7. b  8. a  9. a  10. a

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Takotsubo Cardiomyopathy: A Nurse's Guide
Scott Griffin and Barbara Logue

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