Early Postoperative Takotsubo Cardiomyopathy: A Case Report

Barbara Bradbury, CRNA, MSN
Ferne Cohen, CRNA, MSN

Takotsubo cardiomyopathy (TC), also called stress-induced cardiomyopathy, is a rare type of nonischemic cardiomyopathy accompanied by a transient ballooning and akinesis of the left ventricle. It has been reported mainly in the Japanese literature, although awareness is increasing in the United States and Europe. The reason for the increased incidence in postmenopausal women remains unclear, as does why the apex and left ventricular midcavity are commonly affected. The degree of symptom presentation and severity varies widely and is usually accompanied by an unremarkable medical history. The most frequent presentation is left ventricular dysfunction with or without pulmonary edema. Identifying signs and symptoms and its correlation with physical or emotional stress will lead to early diagnosis and intervention. This case report describes a premenopausal woman in whom TC developed in the immediate postoperative period following general anesthesia.

Keywords: "Broken heart syndrome," left ventricular apical ballooning, reversible ventricular dysfunction, stress-induced cardiomyopathy, Takotsubo cardiomyopathy.

Takotsubo cardiomyopathy (TC), also termed transient left ventricular (LV) apical ballooning syndrome, stress-induced cardiomyopathy, or "broken heart syndrome," is a rare cardiac condition with symptoms resembling acute coronary syndrome (ACS). Takotsubo cardiomyopathy was first identified as a clinical entity in the Japanese literature by Dote and colleagues,[1] who reported 5 cases. The appearance of the left ventricle during systole resembles a Japanese fishing pot, which has a round bottom and narrow neck. In Japanese, *tako* translates as octopus and *tsubo* as pot, hence the name.[2] Because TC occurs mostly in the Japanese population, a genetic component may be involved, although case reports have emerged in non-Asian populations in the United States and Europe.[3-6] It is common among postmenopausal women precipitated by sudden emotional and/or physical stressors,[3,4,7,8] with a correlation rate of 74%.[4] Patients typically have a benign or unremarkable past medical history, and the degree of symptom severity and presentation varies.[3] The etiology remains unclear, but several proposed pathophysiological mechanisms attempt to explain the unusual features of this syndrome.

A large systematic review by Gianni and colleagues[7] indicates that TC accounts for approximately 1.7% to 2.2% of all suspected cases of ACS, with limited reports associated with anesthesia. In the perioperative period, the clinical presentation may include acute LV dysfunction, pulmonary edema, electrocardiogram (ECG) changes consistent with myocardial infarction and/or ischemia, elevation of cardiac enzymes, or cardiogenic shock.[3,9] This case study describes the development of TC in the immediate postoperative period following general anesthesia given for a laparoscopic gynecological procedure.

Case Summary

A 47-year-old premenopausal white woman, ASA 2 classification, with a weight of 63.6 kg and height of 180.3 cm was scheduled for a hysteroscopy, dilation and curettage, diagnostic laparoscopy, and right oophorectomy in an outpatient surgical center. Her medical history included migraines, anxiety, endometriosis, and ovarian cysts. The patient reported a surgical history of laparoscopic procedures and 2 previous cesarean deliveries without any adverse anesthetic complications. Her current medications included ethinyl estradiol and norgestrel birth control, fluoxetine, sumatriptan, calcium, and multivitamins, but no reported herbal medications. Physical examination revealed clear lung sounds on auscultation and regular cardiac rhythm without murmurs or gallops. No preoperative imaging or testing was ordered, although a fingerstick hemoglobin was performed immediately prior to surgery, with a result of 10.5 g/dL. Baseline vital signs were within normal limits (Table 1).

In the operating suite, standard noninvasive monitors were applied and included a pulse oximeter, noninvasive blood pressure cuff, a 3-lead electrocardiogram (ECG), and a peripheral nerve stimulator. Intravenous (IV) access was obtained, and preoxygenation was administered via mask with 100% fraction of inspired oxygen (FIO_2) for 3 minutes. Then general anesthesia was induced with IV administration of 2 mg of midazolam, 100 µg of fentanyl, 60 mg of lidocaine, 4 mg of dexamethasone, and 200 mg of propofol. Once manual

Vital sign	Baseline	After induction	In Trendelenburg position	After metoprolol dosing	On PACU arrival
Temperature (°C)	36.1	36.1	36.2	36.2	36.2
Heart rate (min)	66	75	157	90	117
Respiratory rate (min)	22	10	12	12	10
Blood pressure (mm Hg)	113/75	100/50	220/100	160/80	142/97
Oxygen saturation (%)	100	99	100	100	66
Peak inspiratory pressure (cm H_2O)	–	23-24	28-30	27-30	–
End-tidal carbon dioxide (mm Hg)	–	31	36	35	–

Table 1. Vital Signs
PACU indicates postoperative anesthesia care unit.

ventilation was confirmed, 30 mg of rocuronium was administered IV. Laryngoscopy was performed and endotracheal intubation was accomplished with a size 7 endotracheal tube without difficulty. General anesthesia was maintained with a F_{IO_2} of 50%, 2 L of oxygen and air, and desflurane with an expired concentration of 5% to 6.8%. After induction of anesthesia and prior to insufflation of the abdomen, the patient was hemodynamically stable (see Table 1). Shortly after abdominal insufflation the patient was placed in a steep Trendelenburg position. The ECG displayed a tachyarrhythmia, with a rate of 157/min, and blood pressure increased to 220/110 mm Hg, which was immediately treated with metoprolol (2 mg IV). The surgical procedure was continued without further events of hemodynamic instability or tachyarrhythmias (see Table 1). Rocuronium (10 mg) was given 30 minutes after induction based on a train-of-4 (TOF) of 2 of 4 twitches, and a total of 1 mg hydromorphone IV and 30 mg IV of ketorolac was administered prior to the end of surgery. The patient received 2 L of IV crystalloid solution during the 90-minute procedure, with minimal blood loss of approximately 25 mL.

At the end of the surgical procedure, the patient demonstrated a TOF of 2 of 4 twitches, and neuromuscular blockade was reversed with neostigmine (3 mg IV) and glycopyrrolate (0.4 mg IV). Upon emergence, the patient was awake with spontaneous respirations, arterial oxygen saturation (Sa_{O_2}) of 100%, and following commands but was unable to maintain a sustained head lift for greater than 5 seconds and demonstrated a weak hand grasp despite a TOF of 4 of 4 twitches. The decision was made to maintain tracheal intubation, and the patient was transported supine to the postanesthesia care unit (PACU), manually ventilated with 100% F_{IO_2} via a disposable resuscitator.

On arrival to the PACU, the oxygen saturation was 66%, and a copious amount of pink frothy sputum was noted in the endotracheal tube. The patient's oxygen saturation increased to 77% after the head of the bed was

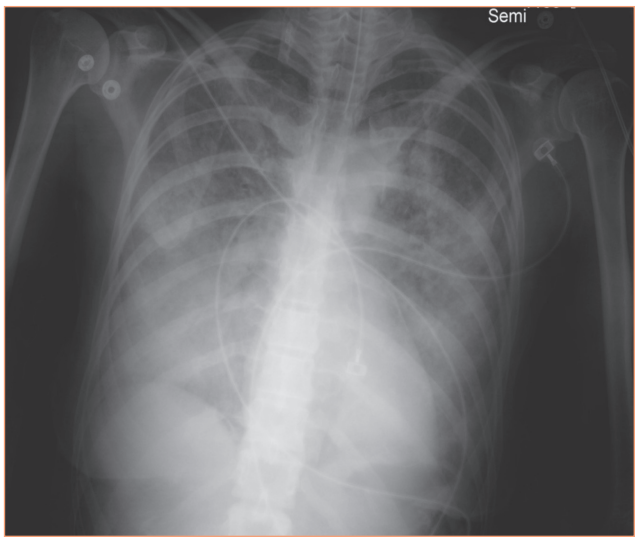

Figure 1. Chest Radiograph Obtained 30 Minutes Postoperatively

elevated, and she was placed on a controlled ventilation regimen with 100% F_{IO_2}, positive end-expiratory pressure (PEEP) of 10 cm H_2O, tidal volume of 600 mL, and assisted controlled ventilation or respiratory rate of 10/min (see Table 1). Considering the patient's clinical presentation, the anesthesia care team implemented the following interventions: multiple tracheal suctioning, 20 mg IV of furosemide, and laboratory and diagnostic testing included complete blood cell count, a comprehensive metabolic panel, cardiac markers, arterial blood gas analysis, portable chest radiograph, and 12-lead ECG. The chest radiograph revealed extensive symmetrical bilateral infiltrates consistent with pulmonary edema (Figure 1). The 12-lead ECG revealed changes consisting of ST-segment elevation in leads V1 and V2, loss of R wave in precordial leads, and inferolateral ST-segment depression (Figure 2). Laboratory results revealed modest elevations of troponin levels, creatine kinase muscle/brain (CK-MB) fraction, CK-MB relative

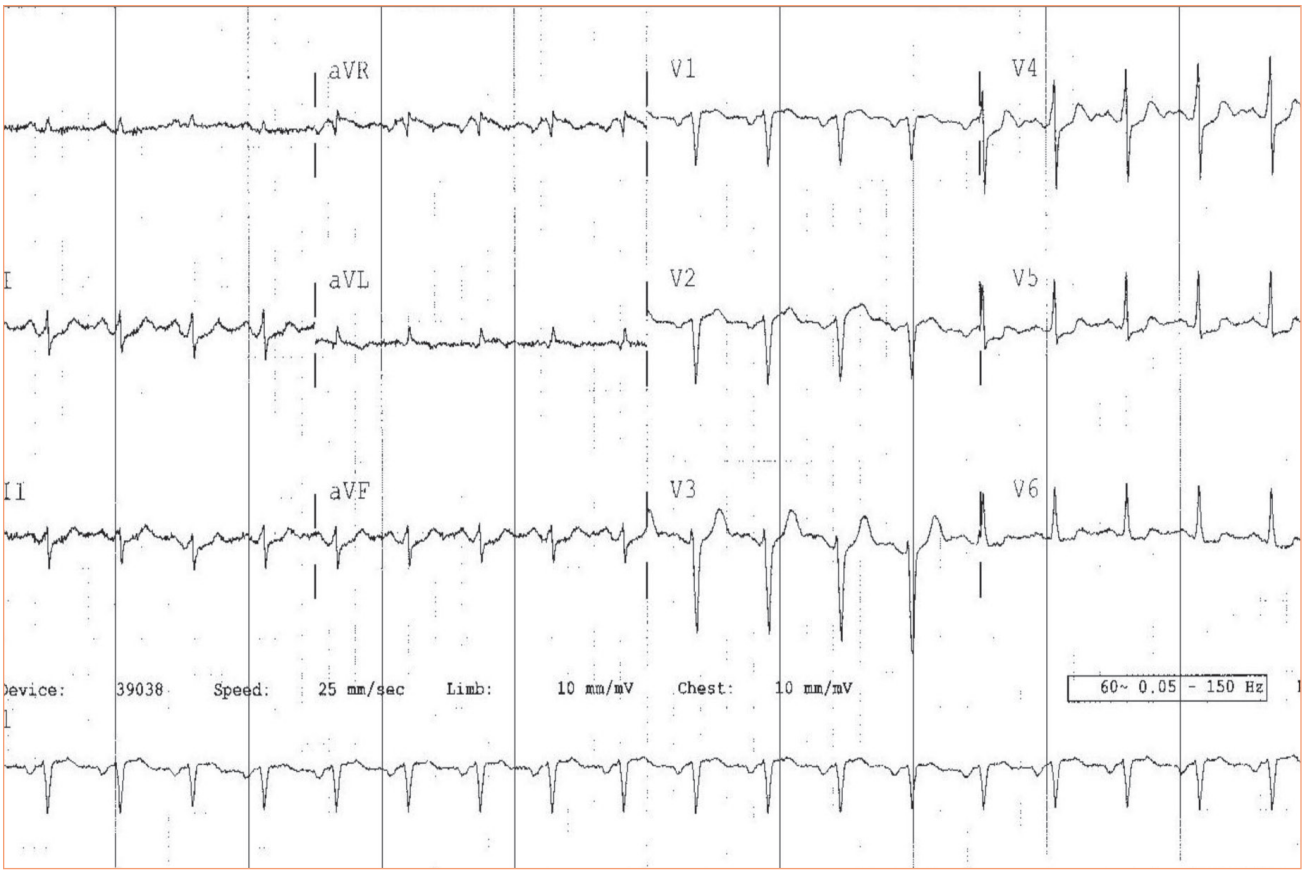

Figure 2. Twelve-Lead Electrocardiogram Immediately Postoperatively
ST segment elevation in leads V1 and V2, loss of R wave in precordial leads, and inferolateral ST segment depression.

index, and lactate dehydrogenase levels (Table 2). A cardiologist was consulted, and after evaluation of the abnormal diagnostic findings and the patient's clinical presentation, an urgent portable transthoracic echocardiogram (TEE) with 2-dimensional views was performed to evaluate cardiac function.

The TEE documented inferior and septal wall akinesis, moderate dilated LV, severely reduced LV systolic function with an ejection fraction of 20% to 25%, and elevated pulmonary artery pressures of 65 mm Hg. Because it was unclear whether these findings were diagnostic of an acute myocardial infarction with an unusual coronary distribution, the cardiologist ordered an urgent coronary angiogram. The patient was transferred intubated to the angiography suite, located in the level I trauma center connected to the outpatient surgical center. The coronary angiogram revealed normal coronary anatomy and unremarkable coronary calcifications but severe basal segmental ventricular wall hypokinesis. The left ventriculogram demonstrated circumferential basal dilation and akinesis with a hyperdynamic apex (Figure 3). Based on the abnormal ECG changes, nonobstructive coronary arteries, modest elevation of cardiac markers, and LV systolic dysfunction, the diagnosis of TC was made. Table 3 contains the diagnostic inclusion criteria for TC.

Discussion

The etiology of TC is unclear; however, several proposed pathophysiological mechanisms leading to the LV wall motion abnormalities have been postulated and include a wraparound left anterior descending coronary artery anatomy, microvascular coronary spasm, microvascular dysfunction, transient excessive levels of catecholamine, and abnormal stress response to catecholamines, specifically epinephrine and norepinephrine.[2,7,8] Wittstein and colleagues[5] measured catecholamine plasma levels in women with transient LV apical ballooning syndrome and discovered that levels were markedly higher than in women presenting with an acute myocardial infarction. The relationship between stress-induced catecholamine release and myocardial dysfunction suggests a link to the development of TC.[10] Multiple case reviews also report an abnormal fatty acid metabolism with impaired glucose metabolism, transient abnormal catecholamine response, or coronary microvascular dysfunction.[3] The myocardium uses fatty acid metabolism during both rest and aerobic conditions, but during ischemia the glucose metabolism becomes impaired.[8] Insulin resistance is associated with high levels of catecholamines, but the etiology of impaired glucose metabolism remains unclear.[8]

A literature review by Gianni and colleagues[7] in 2006

Laboratory value	Immediate postoperative period (12:01 PM)	Next day (12:16 AM)	Reference range
White blood cells ($\times 10^9$/L)	31	28.4	3.5-11
Hemoglobin (g/dL)	12.7	11.6	11.7-15.7
Hematocrit (%)	41.2	37.3	35-47
Platelets ($\times 10^9$/L)	554	375	150-400
Glucose (mg/dL)	196	179	70-120
Sodium (mmol/L)	140	140	136-146
Potassium (mmol/L)	4	4.5	3.5-5
Chloride (mmol/L)	101	101	100-112
Total carbon dioxide (mmol/L)	24	21	24-32
Serum urea nitrogen (mg/dL)	15	20	8-22
Creatinine (mg/dL)	1	1.4	0.5-1
Magnesium (mEq/L)	2	1.5	1.7-2.4
Phosphorus (mg/dL)	6.7	5	2.5-4.5
Calcium (g/dL)	8.6		8.4-10.3
Anion gap (mmol/L)	15	18	4-12

	Early postoperative period		Next day		
Cardiac enzymes	12:45 PM	8:57 PM	5:39 AM	7:16 PM	Reference range
Troponin (µg/L)	0.51	1.06	0.64	0.39	0-0.03
Creatine kinase (CK) (U/L)	118	240	340	335	30-120
CK muscle/brain (CK-MB) fraction (µg/L)	6.5	22.6	27.3	15.7	0-3.8
CK-MB relative index	5.5	9.4	8	4.7	0-4.2
Lactate dehydrogenase (U/L)	724				310-620

	Early postoperative period			
Arterial blood gas	12:01 PM	1:25 PM	11:50 PM	Reference range
pH	7.16	7.25	7.38	7.35-7.45
Pco_2 (mm Hg)	60.5	48.4	36.3	35-45
Po_2 (mm Hg)	59	72	138	75-100
Total co_2 (mmol/L)	24	23	23	24-32
Hco_3 (mmol/L)	22	21.3	21.7	22-27
Base excess (mmol/L)	–7	–6	–3	–2-3
o_2 saturation (%)	83	91	99	96-99
Fio_2 (%)	100	100	60	

Table 2. Laboratory Results
Pco_2 indicates partial pressure of carbon dioxide; Po_2, partial pressure of oxygen; co_2, carbon dioxide; Hco_3, bicarbonate; o_2, oxygen; and Fio_2, fraction of inspired oxygen.

revealed a marked gender discrepancy in TC and found that of 286 patients, 254 (89%) were female and most were postmenopausal, with a mean age range from 58 to 77 years. It is proposed that postmenopausal women may demonstrate altered endothelial function in response to reduced estrogen levels after exposure to sudden, unexpected emotional or physical stress.[7] The occurrence of TC increased substantially in elderly women living near the epicenter of the Niigata earthquake in Japan.[10]

A large systematic review by Dorfman and Iskandrian[8] revealed that physical or emotional stress was the precipitating factor in 87% of 789 patients with TC. In 2009, Regnante and colleagues[11] conducted a prospective study of 70 patients diagnosed with TC, and the authors identified that 37% (26) of these patients has a preceding physical stressful trigger and 30% (21) had an emotional trigger. Ninety-five percent (67) of the patients were postmenopausal women and 87% (61) were white

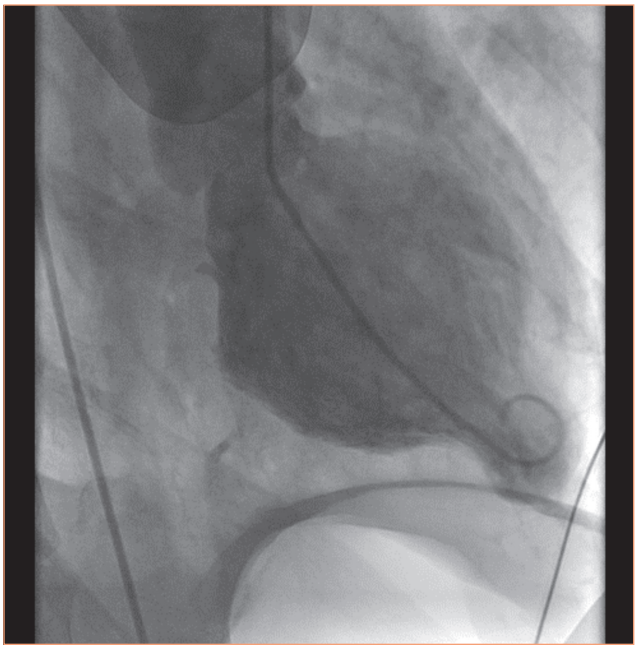

Figure 3. Left Ventriculogram
End systolic phase demonstrates circumferential basal dilation and akinesis with a hyperdynamic apex.

1. Transient left ventricular hypokinesis, akinesis, or dyskinesis with or without apical involvement. The regional wall motion abnormalities typically extend beyond a single epicardial coronary distribution. It is frequently, but not always, a stressful trigger.
2. Absence of obstructive coronary artery disease or angiographic evidence of acute plaque rupture
3. New electrocardiogram changes: ST-segment elevation and/or T-wave inversion or modest elevation in cardiac troponin levels
4. Absence of pheochromocytoma and myocarditis

Table 3. Mayo Clinic Diagnostic Inclusion Criteria of Takotsubo Cardiomyopathy
All 4 criteria are required for the diagnosis.[10,17]

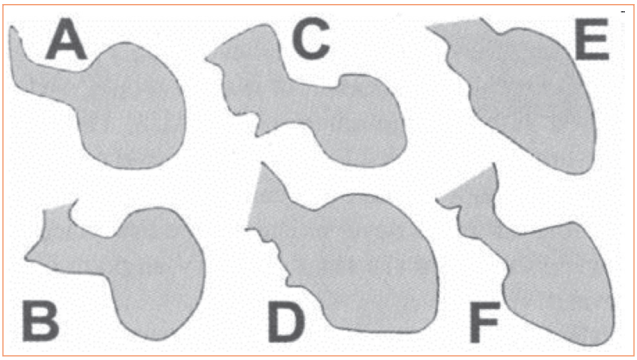

Figure 4. Diversity of End-Systolic Left Ventricle Silhouettes
(Reprinted with permission from *Mayo Clinic Proceedings*.[2])

women, which are consistent findings among other case series.[11] The study also revealed a seasonal variation, with an increase in occurrence of TC during the summer, in contrast to the National Registry of Myocardial Infarction, which demonstrates a peak incidence for acute myocardial infarctions in the winter.[11]

The review of literature about TC related to anesthesia and the perioperative period reveal a limited number of case reports. In 2006, Gavish and colleagues[12] reported the first case of a general anesthesia-related transient LV apical ballooning in a non-Japanese patient undergoing cataract extraction, who experienced acute transient myocardial dysfunction without substantial coronary pathology. Gavish et al[12] suspected that preoperative psychological stress, hypoxemia, laryngoscopy, and reintubation may have contributed to the catecholamine surge and the consequential development of TC. Littlejohn and colleagues,[13] in 2008 presented 3 anesthesia-related case studies of women in whom TC developed postoperatively. All 3 patients received an accurate diagnosis, were appropriately managed, and had good outcomes.[13]

The literature describes only a few cases of TC in younger patients.[14] A noteworthy case report involves a 31-year-old healthy woman scheduled for an elective "repeat" cesarean delivery under spinal anesthesia.[15] Fifteen minutes after the spinal anesthetic was placed, the patient became hypotensive and bradycardic, with a heart rate of 36/min, and was treated with atropine, ephedrine, and phenylephrine.[15] After the second dose of atropine, the ECG showed sinus tachycardia (heart rate, 150/min), and the patient complained of chest pressure and blindness and demonstrated a deteriorating neurological condition.[15] The patient was administered general anesthesia, and 4 hours after extubation reversible LV systolic dysfunction developed.[15] Although the cause is unclear, it is postulated that the hemodynamic instability coupled with the pharmacologic intervention may have induced a stress response and precipitated the development of TC.[15]

A more recent case involving a 31-year-old premenopausal woman who experienced an atypical presentation of stress-related cardiomyopathy after major breast surgery was reported by Pfister et al.[16] Despite not having the takotsubo feature on left ventriculogram, she did present with other characteristics commonly found which included pulmonary edema, diffuse T wave abnormality, severe LV dysfunction, and elevated troponin levels. The authors postulate that prolonged surgery with catecholamine release may have contributed to the development of LV dysfunction along with postoperative pain, length of surgery, anesthesia withdrawal, emotional stress related to diagnosis of invasive, aggressive breast cancer, or intravascular fluid volume shifts.[16] The patient was found to have normal coronary anatomy and recovered quickly.[16]

Atypical presentation of transient LV apical ballooning without involvement of the apex may be present in TC,

Patient presents with	Treatment
ST elevation	Urgent cardiac catheterization for primary percutaneous coronary intervention (PCI). If TC, angiographic findings will demonstrate no critical coronary disease and apical ballooning or midwall hypokinesis on left ventricular angiogram.
ST elevation, but urgent angiography and PCI are not available	Initiate fibrinolytic therapy
No ST elevation	Early (less than 48 hours) cardiac catheterization should suggest the correct diagnosis
Contraindication to cardiac catheterization and fibrinolytic therapy	Pattern of wall motion abnormality may differ between TC and acute obstruction of flow in left anterior descending coronary artery on echocardiogram. TC typically involves distribution of more than 1 coronary artery.

Table 4. Guide to Diagnosis and Intervention of Takotsubo Cardiomyopathy (TC)

Patient presents with	Treatment
Hemodynamically stable patient without left ventricular outflow tract (LVOT) obstruction	β-Blocker therapy
	Angiotensin converting enzyme inhibitor or angiotensin receptor blocker
	Diuresis if heart failure is present
	Aspirin therapy in presence of coexisting coronary atherosclerosis
Hypotension without LVOT	Intravenous inotropes—dopamine
	Intra-aortic balloon pump if no response to initial medical therapy and volume resuscitation
Severe systolic dysfunction or LVOT obstruction with hypotension and shock	Hypotension and shock present: urgent echocardiogram to determine if LVOT obstruction is present
	No intravenous inotropes; may worsen the degree of obstruction
	Phenylephrine
	β-Blocker therapy
	If β-blocker therapy is not tolerated, then add α-agonist with caution and closely monitor patient
	Intra-aortic balloon pump if no response to initial medical therapy and volume resuscitation
	Avoid dobutamine

Table 5. Management Based on Hemodynamics

which is thought to be due to a unique distribution of the adrenergic receptors.[13] Patients are also at risk of obstruction of the LV outflow tract, which is caused by the exaggerated contraction of the base of the ventricle when hypokinesis of the apex occurs.[7] Left ventricle presentation can include a variety of end-systolic LV silhouettes,[2] as seen in Figure 4.

• *Treatment and Prognosis.* Accurate diagnosis of TC with pharmacologic and hemodynamic support leads to rapid reversal of LV dysfunction and increases the rate of survival.[6] Although the optimal medical management of TC has not been established, supportive treatment leads to a good prognosis in patients without any major comorbidities, provided that they survive the acute severe heart failure period.[3] The most frequent complication is left-sided heart failure with or without pulmonary edema.[3] Infrequent complications include cardiogenic shock, ventricular arrhythmias, LV mural thrombus, mitral valve dysfunction, pulmonary embolism, and LV rupture,[3] with the in-hospital mortality rate ranging from 0% to 8%.[6,8,10,17,18] In the setting of LV outflow tract (LVOT) obstruction and inappropriate management, death is much more common.[8]

Patients presenting to the emergency department with TC have symptoms suggestive of ACS, which include chest pain, dyspnea, ECG changes, and small early increases in cardiac biomarkers.[17] Because the symptoms of TC closely mimic ACS, they must be carefully diagnosed as these 2 cardiac disorders have a different pathogenesis, management, and outcomes. Table 4 outlines the recommendations to guide diagnosis and medical treatment of TC.[18]

Emerging data concerning pharmacologic interven-

Preoperative care
Avoid psychological stress and minimize preoperative anxiety
• Anxiolysis with psychological and pharmacologic approaches
• Provide preoperative teaching and education
• Mentally prepare patient for surgery
Institute β-blocker therapy if no contraindications
Provide surgical care in hospital setting with full cardiac services
Consider regional anesthesia with sedation versus general anesthesia
Intraoperative care
Perform intra-arterial catheterization for monitoring arterial blood pressure
Closely monitor 5-lead electrocardiogram
Monitor transesophageal echocardiogram to assess left ventricular function continuously for general anesthesia cases
Minimize sympathetic stimulation and limit catecholamine discharge
• Brief laryngoscopy
• Smooth emergence and extubation
Avoid hemodynamic instability
Avoid residual neuromuscular blockade
Choose anesthetic agents with the least myocardial depressant effects for induction and maintenance
Avoid volume overload
Postoperative care
Admit to intensive care for vigilant monitoring
Provide adequate pain control

Table 6. Perioperative Management Goals and Guidelines

tions support the use of β-blockade to attenuate the transient LVOT obstruction, and treatment should be individualized based on intraventricular pressure gradients and LVOT obstruction with or without hypotension.[8] In patients with evidence of coronary artery vasospasm, dihydropyridine calcium channel blockers are recommended in place of β-blockers and phenylephrine.[8] The role of aspirin and angiotensin-converting enzyme (ACE) inhibitors remains unclear.[8] However, Regnante et al[11] found that long-term use of ACE inhibitors before the onset of TC protected against cardiogenic shock, sustained ventricular arrhythmia, and death.

If cardiogenic shock and acute decompensated heart failure develop, management includes positive inotropic medications, ventilatory support, an LV assist device, and an intra-aortic balloon pump.[3] Dopamine and dobutamine can be used in hypotensive patients without substantial LVOT obstruction.[18] However, it is recommended that dobutamine be avoided in patients with severe systolic dysfunction or LVOT obstruction with hypotension and shock.[8] Phenylephrine, an α-adrenoceptor agonist, attenuates transient outflow obstruction, intraventricular pressure gradients, and ejection velocity, which results in a higher LV end diastolic volume.[8] Phenylephrine, if used, should be administered with caution, and vigilant hemodynamic monitoring, tissue perfusion, and mentation should be monitored, because the vasoactive effects may be harmful in patients prone to coronary vasospasm.[18] In the presence of a prolonged QT interval on the ECG, selective anti-arrhythmic medication should be given.[3] See Table 5 for the management based on hemodynamic parameters.[8,18]

• *Anesthesia Management.* The correlation between physiological stress and cardiovascular events in the perioperative period is well known. The "stress response to surgery initiates a cascade of physiologic and metabolic events through direct activation of the sympathetic and somatic nervous system" which begins with the induction of general anesthesia and lasts 3 to 4 days postoperatively.[19] It is documented that cardiac morbidity is the most common cause of death after major surgical procedures.[19] Therefore, it is important to consider TC as a differential diagnosis in patients demonstrating myocardial dysfunction in the perioperative period, especially postmenopausal women. Because the recurrence rate of TC is approximately 3.5%, a patient with a history of TC may present for surgery.[7] The recommended perioperative anesthesia management of TC includes minimizing anxiety, maintaining normovolemia, close ECG monitoring of cardiac function, and supportive pharmacologic management based on hemodynamic parameters (Table 6).[9]

Conclusion

This case study documents a presentation of TC in the immediate postoperative period in a premenopausal white woman after general anesthesia. Her clinical presentation of distinctive LV wall motion abnormalities with resolution, absence of obstructive coronary artery disease, and ECG changes meet the diagnostic criteria for TC, which probably produced the pulmonary edema and reduced LV function. On postoperative day 1, the repeated echocardiogram revealed an improved ejection fraction of 40% with normal LV wall thickness and size; mild inferoseptal, anterior, and basal inferior wall hypokinesis; and normalization of pulmonary artery pressures to 20 to 25 mm Hg. The ECG demonstrated sinus rhythm with borderline prolonged QT interval. The patient was discharged on postoperative day 4 on a regimen of β-blockers, metoprolol (25 or 50 mg twice a day), and low-dose aspirin. It was recommended that follow-up echocardiograms be performed on an outpatient basis.

It is apparent that further research of TC is necessary to clarify the etiology, influence of genetic factors, relationship to postmenopausal women, correlation with emotional and physical stress, association with surgical procedures, optimal management strategies, and iden-

tification of any modifiable risk factors contributing to the development of TC. Because of the rarity of this syndrome, anesthesia providers must be cognizant of the recommended perioperative management for the surgical patient with a history of TC and consider a diagnosis of TC in a patient demonstrating myocardial dysfunction, as an early and accurate diagnosis leads to appropriate interventions and good patient outcomes.

REFERENCES

1. Dote K, Sato H, Tateishi H, Uchida T, Ishihara M. Myocardial stunning due to simultaneous multivessel coronary spasm: a review of 5 cases. *J Cardiol.* 1991;21(2):203-214.
2. Ibanez B, Benezet-Mazuecos J, Navarro F, Farre J. Takotsubo syndrome: a Bayesian approach to interpreting its pathogenesis. *Mayo Clin Proc.* 2006;81(6):732-735.
3. Brenner ZR, Powers J. Takotsubo cardiomyopathy. *Heart Lung.* 2008; 37(1):1-7.
4. Vidi V, Rajesh V, Singh PP, et al. Clinical characteristics of tako-tsubo cardiomyopathy. *Am J Cardiol.* 2009;104(4):578-582.
5. Wittstein IS, Thiemann DR, Lima JA, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. *N Engl J Med.* 2005;352(6):539-548.
6. Sharkey SW, Lesser JR, Zenovich AG, et al. Acute and reversible cardiomyopathy provoked by stress in women from the United States. *Circulation.* 2005;111(4):472-479.
7. Gianni M, Dentali F, Grandi AM, Sumner G, Hiralal R, Lonn E. Apical ballooning syndrome or takotsubo cardiomyopathy: a systematic review. *Eur Heart J.* 2006;27(13):1523-1529.
8. Dorfman TA, Iskandrian AE. Takotsubo cardiomyopathy: state-of-the-art review. *J Nucl Cardiol.* 2009;16(1):122-134.
9. Liu S, Bravo-Fernandez C, Riedl C, Antapli M, Dhamee MS. Anesthetic management of Takotsubo cardiomyopathy: general versus regional anesthesia. *J Cardiothorac Vasc Anesth.* 2008;22(3):438-441.
10. Akashi YJ, Goldstein DS, Barbaro G, Ueyama T. Takotsubo cardiomyopathy: a new form of acute, reversible heart failure. *Circulation.* 2008;118(25):2754-2762.
11. Regnante RA, Zuzek RW, Weinsier SB, et al. Clinical characteristics and four-year outcomes of patients in the Rhode Island Takotsubo Cardiomyopathy Registry. *Am J Cardiol.* 2009;103(7):1015-1019.
12. Gavish D, Rozenman Y, Hafner R, Bartov E, Ezri T. Takotsubo cardiomyopathy after general anesthesia for eye surgery. *Anesthesiology.* 2006;105(3):621-623.
13. Littlejohn FC, Syed O, Ornstein E, Connolly ES, Heyer EJ. Takotsubo cardiomyopathy associated with anesthesia: three case reports. *Cases J.* http://casesjournal.com/content/1/1/227. Accessed November 18, 2010.
14. Bybee KA, Prasad A. Stress-related cardiomyopathy syndromes. *Circulation.* 2008;118(4):397-409.
15. Crimi E, Baggish A, Leffert L, Pian-Smith MCM, Januzzi JL, Jiang Y. Acute reversible stress-induced cardiomyopathy associated with cesarean delivery under spinal anesthesia. *Circulation.* 2008;117(23): 3052-3053.
16. Pfister S, Wagar P, Casserly IP. Stress-related cardiomyopathy in a 31-year-old woman. *AANA J.* 2010;78(5):406-411.
17. Prasad A. Apical ballooning syndrome: an important differential diagnosis of acute myocardial infarction. *Circulation.* 2007;115(5):e56-e59.
18. Reeder GS, Prasad A. Stress-induced (takotsubo) cardiomyopathy. *UpToDate*. http://www.uptodate.com/home/content/topic.do?topicKey=hrt_fail/23221. Accessed November 18, 2010.
19. Moraca RJ, Sheldon DG, Thirlby RC. The role of epidural anesthesia and analgesia in surgical practice. *Ann Surg*, 2003;238(5):663-673.

AUTHORS

Barbara Bradbury CRNA, MSN, is a staff nurse anesthetist at Nanticoke Memorial Hospital in Seaford, Delaware. At the time this paper was written, she was a student at Drexel University, Nurse Anesthesia Program, Philadelphia, Pennsylvania. Email: bab49@drexel.edu or duckdive@verizon.net.

Ferne Cohen, CRNA, MSN, is assistant professor at Drexel University, Nurse Anesthesia Program. Email: fmc24@drexel.edu.

ACKNOWLEDGMENTS

We thank the following for their assistance: Elias Chua, MD, clinical site director, Christiana Health Systems Operating Room, and staff anesthesiologist, Anesthesia Services, P.A., New Castle, Delaware; Mark Mulvihill, MD, staff anesthesiologist, Anesthesia Services, P.A.; Paula Chua, CRNA, MS, staff anesthetist, Anesthesia Services, P.A.; and George Moutsatsos, MD, Cardiology Consultants, Wilmington, Delaware.