Transient left ventricular apical ballooning, also known as takotsubo cardiomyopathy, is an unusual abnormality that may be the underlying cause of signs and symptoms of acute myocardial infarction (AMI) in a small number of patients. The signs and symptoms include chest pain, ST-segment changes, and the release of cardiac biomarkers. Dyspnea and hypotension may also occur. Although these signs and symptoms are suggestive of AMI, they are not caused by ischemic coronary artery disease. This abrupt onset of extensive “ballooning” or dilatation of the left ventricle occurs most often in postmenopausal women after a traumatic psychosocial or physical stressor.

Although its onset is sudden and dramatic, apical ballooning is transient and reversible. Its cause is not known. It is increasingly recognized and reported in the medical literature. Because apical ballooning mimics the signs and symptoms of AMI, emergency department and critical care nurses may care for patients with this interesting and uncommon abnormality. In this article, I review the pathophysiology, clinical features, management, complications, and prognosis of apical ballooning. Three case reports are included to illustrate the growing experience with this abnormality.

Apical ballooning was first described in the Japanese literature in the early 1990s and was attributed to simultaneous spasm of multiple coronary arteries. The original name given to apical ballooning was takotsubo cardiomyopathy, which was derived from the shape of the narrow-necked bulging “takotsubo” container used by Japanese fisherman to trap octopus. The shape of the takotsubo pot resembles the distorted ballooning ventricle (Figure 1). Table 1 lists several other terms used in the medical literature to describe this ballooning. Additional reports of apical ballooning have come from many areas of the world, including Europe, Australia, South America, and the United States. Because of the increased recognition of this abnormality, in the International
Among women with signs and symptoms of AMI, 6% have apical ballooning.\textsuperscript{19} Most patients with apical ballooning have experienced a marked psychosocial or physical stress that precipitates their signs and symptoms.\textsuperscript{5,13-15,18-20} Examples of reported stressors are listed in Table 2.

**Pathophysiology**

Patients with apical ballooning have marked systolic ballooning of the ventricular apex, often associated with hypercontractility of the base of the heart. Although ballooning of the left ventricle is most common, the right ventricle can also be affected.\textsuperscript{2,32} In the initial reports from Japan, the alterations in ventricular function were attributed to simultaneous multivessel coronary artery spasm.\textsuperscript{1,2} The pathophysiological mechanism that initiates apical ballooning is not fully understood, but it is now thought to be related to stunning of the myocardium related to excessive catecholamines.\textsuperscript{15,16}

In most patients, the onset of signs and symptoms is preceded by increased psychosocial or physical stress, suggesting an association with increased activity of the sympathetic nervous system. Catecholamines can have a toxic effect on the myocardium.\textsuperscript{19} Markedly elevated serum levels of catecholamines have been reported in patients with apical ballooning, and it is postulated that these elevated levels lead to toxic myocardial effects and stunning of the ventricle. In one series of patients,\textsuperscript{16} the levels of plasma catecholamines were several times higher than the levels in a group of patients who had AMI. Other suggested mechanisms include rupture of a nonobstructive plaque followed by spontaneous thrombolysis,\textsuperscript{11} microvascular coronary spasm or dysfunction,\textsuperscript{16,23} transient obstruction to left ventricular outflow,\textsuperscript{1} and acute myocarditis.\textsuperscript{23}

Why apical ballooning occurs most often in postmenopausal women is not known. The role of sex hormones and their impact on the sympathetic nervous system and catecholamine metabolism are not well understood. Postmenopausal alterations of endothelial function are also not well understood.\textsuperscript{5} More research is necessary. In addition, a genetic component may be involved; apical ballooning has been reported in sisters. Some patients have had single or multiple recurrences of this abnormality.\textsuperscript{15,16,21,33}

Anatomically, an abnormally long left anterior descending artery that courses along the diaphragmatic surface of the left ventricle has been reported, but this finding is not a consistent one in apical ballooning.\textsuperscript{34} Elevated levels of circulating
delayed enhancement with gadolinium contrast medium was apparent, indicating that the myocardial injury was reversible.

Clinical Features and Findings
Consensus criteria for diagnosing apical ballooning do not yet exist. 

Table 2
Reported triggers of apical ballooning

- Unexpected death of a spouse, child, or close family member
- Anniversary of death of a family member
- Having a spouse leave for war
- Witnessing an accident, being in an accident
- House fire
- Armed robbery
- Excessive alcohol intake or alcohol/drug withdrawal
- Surprise party, surprise reunion
- Losing money in a casino, loss of life savings, financial instability
- Court appearance, legal proceedings
- Public performance, public speaking
- Hypothermia
- Loss of job, occupational stress
- Lightning strike
- Earthquake
- Quarreling, fierce arguments
- Domestic abuse
- Vigorous exercise
- Cerebrovascular accident, witnessing a family member have a cerebrovascular accident
- Epilepsy, grand mal seizure
- Acute dyspnea, asthma, pneumothorax
- Acute abdomen, acute cholecystitis
- Sepsis and hypoxemia with respiratory failure
- Hip fracture, hip surgery
- Severe pain
- Severe hypoglycemia
- Noncardiac surgical procedures
- Fear of impending procedure
- Receiving news of serious diagnosis

Table 3
Clinical features of apical ballooning

- Onset of signs and symptoms often preceded by emotional/physical stressor
- Most common in postmenopausal women
- ST-segment abnormalities that mimic those of acute myocardial infarction
- Signs and symptoms similar to those of acute myocardial infarction
- Mild to moderate increase in levels of cardiac enzyme compared with the increases in acute myocardial infarction
- No significant epicardial coronary artery disease to account for the left ventricular dysfunction
- Left ventricular “ballooning” wall motion at the apex with hypercontractility at the base
- Transient and reversible left ventricular changes with favorable prognosis

catecholamines and plasma brain natriuretic peptide have been reported. In one report, endomyocardial biopsy showed no significant inflammatory process. In another report, biopsy did not suggest any specific heart disease, and myocarditis was ruled out as a causative factor. Some patients have a pressure gradient within the left ventricle, but this finding is not consistent.

Results of thallium nuclear scanning are usually normal or may show a small area of defect. In a study by Sharkey et al, magnetic resonance imaging showed diffuse wall motion abnormalities of the ventricle that could not be explained by spasm of any single coronary artery. In addition, the scarring normally associated with AMI was not present, and no delayed enhancement with gadolinium contrast medium was apparent, indicating that the myocardial injury was reversible.

Clinical Features and Findings
Consensus criteria for diagnosing apical ballooning do not yet exist.

Table 3 provides a summary of clinical features consistent with this abnormality. Patients most often have chest pain mimicking that of AMI. They may also have dyspnea. The pain characteristics are not well delineated. Other reported signs and symptoms include syncope or near syncope, fatigue, malaise, and palpitations. A subset of patients may also have marked hypotension, pulmonary edema, cardiogenic shock, and/or lethal ventricular arrhythmias. These patients require short-term vasoressors, temporary pacemakers, and intra-aortic balloon pump support while the left ventricle recovers.

Electrocardiographic (ECG) findings are variable and cannot be used to diagnose apical ballooning. ECGs show ST-segment elevation or depression, usually in the precordial leads, particularly V2 to V5. Reciprocal changes in the inferior leads may not occur, and in most patients, Q waves do not develop. Deeply inverted T waves are common during the recovery phase. Many patients have a markedly prolonged QT interval (normal, 440 milliseconds). Compared with findings in AMI, the cardiac biomarkers troponin, creatine kinase, and creatine kinase
MB are only moderately elevated in apical ballooning, and they do not follow the same rise-and-fall patterns associated with AMI.22 The reason for this difference is unclear. The characteristic systolic ballooning of the ventricle is clearly evident on echocardiograms and in cardiac catheterization. Ejection fraction may be markedly decreased in the acute phase of the ballooning. Ejection fractions as low as 15% to 40% have been reported.4,13,14,16,18 Coronary angiography reveals no significant epicardial coronary artery disease to account for the marked left ventricular dysfunction.12,18 Table 4 gives associated diagnostic findings.

Management

When a patient’s onset of signs and symptoms, especially in a postmenopausal woman, coincides with a significant psychosocial or physical stressor, apical ballooning should be considered. Early echocardiography or coronary angiography showing ballooning of the ventricle will be most helpful in differentiating this abnormality from AMI. Prompt recognition of apical ballooning prevents the unnecessary administration of fibrinolytic agents to patients with apical ballooning who have ST-segment elevation.

Complications and Prognosis

The most commonly reported complication of apical ballooning is left ventricular failure.22 Arrhythmias have been reported, including sinus bradycardia, atrial fibrillation, ventricular tachycardia, and ventricular fibrillation.14,22 Heart block requiring temporary and permanent pacing has been described.9 Hypotension, pulmonary edema, cardiogenic shock, mitral regurgitation, mural thrombus, left ventricular free-wall rupture, and death have also been reported.1,19,37,38

Apical ballooning occurs most often in postmenopausal women who have experienced a physical or psychosocial stressor. Left ventricular function improves rapidly, often within 7 to 30 days of the onset of signs and symptoms.9 The ECG changes may be slower to resolve.36 The prognosis is generally favorable; a mortality of 0% to 8% has been reported.14,22

Implications for Nursing Care

Patients with apical ballooning typically have the same signs and symptoms as patients with AMI and are admitted to a critical care unit. Goals of care are similar to those for patients with AMI: alleviating pain, reducing anxiety, maintaining contractility, and preventing and

---

**Table 4** Diagnostic findings common in apical ballooning

| Laboratory tests: Elevated serum/plasma levels of | creatine kinase, creatine kinase MB, troponin, norepinephrine, brain natriuretic peptide, dopamine |
| Electrocardiography: ST-segment elevation or depression, T-wave inversion, and/or prolonged QT interval |
| Nuclear stress testing: Evidence of reversible myocardial injury |
| Echocardiography: Marked apical ballooning with systolic dysfunction; akinetic or dyskinetic left ventricle |
| Left ventriculography: Marked apical ballooning with systolic dysfunction; akinetic or dyskinetic left ventricle |
| Coronary angiography: No significant coronary artery disease |
| Cardiac magnetic resonance imaging: Diffuse wall motion abnormalities |
treatment complications, such as arrhythmias, heart failure, and cardiogenic shock.39-41

Because ST-segment changes, prolonged QT intervals, heart block, and lethal ventricular arrhythmias have been reported, continuous cardiac monitoring is essential. Many patients also have shortness of breath and pulmonary congestion that requires frequent assessment of heart and lung sounds to monitor fluid status. Diuretic therapy is often indicated and can lead to alterations in fluid and electrolyte balance, so regular monitoring of laboratory values and electrolyte replacement is essential.

During the acute phase, contractility is affected by the severe dysfunction of the left ventricle, resulting in decreases in ejection fraction, cardiac output, and cardiac index. For some patients, inotropic support with low-dose dopamine or dobutamine may be required to improve cardiac output and cardiac index. A pulmonary artery and/or an arterial catheter may be placed for monitoring hemodynamic parameters and titrating vasoactive agents during the acute phase of the illness. On a short-term basis, intra-aortic balloon counterpulsation may be indicated to decrease left ventricular workload, decrease afterload, improve contractility, and increase stroke volume.

**Case 1**

A 66-year-old woman came to the emergency department of a small hospital because of the sudden onset of severe substernal non-radiating chest pain and shortness of breath. An ECG showed marked T-wave inversion in leads V3 to V6. The QT interval was prolonged at 604 milliseconds (Figure 2). The patient’s serum troponin level was elevated at 3.35 ng/mL (normal, 0.00-0.04 ng/mL). She had a history of hypothyroidism and no major risk factors for coronary disease. She was the primary caregiver for her husband, who had advanced, rapidly progressing Alzheimer’s disease. The diagnosis was non-ST-segment elevation myocardial infarction. She was given a weight-based bolus of eptifibatide, and a continuous infusion of the drug at a rate of 2.0 μg/kg per minute was started. She was transferred to a tertiary care facility for urgent cardiac catheterization and treatment.

Coronary angiography showed no obstructive lesions (Figures 3 and 4). Testing with methylergonovine (Methergine) ruled out coronary artery spasm. Findings on the left ventriculogram (Figure 5) were markedly abnormal, with apical ballooning and a decreased ejection fraction of 45% (normal, 55%-70%). The left ventricular end-diastolic pressure was 29 mm Hg (normal, 5-12 mm Hg). Because angiograms showed no evidence of ischemic coronary disease, the eptifibatide was discontinued. The patient’s hospital course remained uneventful, and she was discharged home on day 4. The final diagnosis was takotsubo cardiomyopathy, or apical ballooning. Discharge medications included enoxaparin 80 mg subcutaneously twice daily for 5 days, warfarin 5 mg/d, hydrochlorothiazide 50 mg/d, and levothyroxine 0.125 mg/d.

**Case 2**

A 58-year-old woman went to an outlying hospital because she had
experienced intense chest pain when she found the body of her husband, who had committed suicide. She had a history of hypertension and smoking. The initial ECG showed nonspecific ST-T wave changes. The QTc interval was prolonged at 517 milliseconds, and the troponin level was elevated at 1.3 ng/mL. She was admitted to the intensive care unit for observation. Several hours after admission, severe crushing substernal chest pain developed; she described the intensity as 8 on a scale of 1 to 10. ECGs showed new T-wave inversion in leads V₂ to V₆ (Figure 6). Systolic blood pressure was 80 mm Hg. Transthoracic echocardiography indicated severe akinesis of the anterior wall of the heart and an ejection fraction of 20%.

She was transferred to another facility for emergent cardiac catheterization with coronary angiography. The examinations revealed no significant coronary artery disease. She had a markedly elevated left ventricular end-diastolic pressure of 35 mm Hg and severe akinesis and ballooning of the midanterior wall and midinferior wall with relative sparing of the proximal anterior and posterobasal and apical walls of the left ventricle. The diagnosis was takotsubo cardiomyopathy, or apical ballooning. She was transferred to the cardiac intensive care unit for continued monitoring and nursing care. Her remaining hospitalization was uneventful, and she was discharged home on day 4. Discharge medications included amlodipine 5 mg/d, ramipril 5 mg/d, aspirin 325 mg/d, rosuvastatin 10 mg/d, and carvedilol 3.125 mg twice daily.

**Case 3**

A 50-year-old woman went to an outlying rural hospital because she had shortness of breath and chest pain after intensely arguing with a family member about her (the patient’s) alcoholism. She had a history of type 1 diabetes mellitus and chronic obstructive pulmonary disease, and she was a 35-pack-year smoker. The ECG findings were markedly abnormal, with sinus tachycardia and ST-segment elevation in leads V₂ to V₅ (Figure 7). The QT interval was normal. The troponin level was elevated at 5.28 ng/mL. The patient was hypotensive and had bilateral crackles. Her clinical condition was stabilized in the emergency department. She was given 325 mg of chewable aspirin, and treatment with dopamine was started at a rate of 6 μg/kg per minute. She was transferred emergently to another facility for cardiac catheterization and possible percutaneous coronary intervention because of her continuing unstable hemodynamic status.

Coronary angiography showed no evidence of obstructive coronary artery disease. Findings on left ventricular angiography were markedly abnormal, with a large area of apical akinesis. The ejection fraction was 15%. Left ventricular end-diastolic pressure had shortness of breath and chest pain after intensely arguing with a family member about her (the patient’s) alcoholism. She had a history of type 1 diabetes mellitus and chronic obstructive pulmonary disease, and she was a 35-pack-year smoker. The ECG findings were markedly abnormal, with sinus tachycardia and ST-segment elevation in leads V₂ to V₅ (Figure 7). The QT interval was normal. The troponin level was elevated at 5.28 ng/mL. The patient was hypotensive and had bilateral crackles. Her clinical condition was stabilized in the emergency department. She was given 325 mg of chewable aspirin, and treatment with dopamine was started at a rate of 6 μg/kg per minute. She was transferred emergently to another facility for cardiac catheterization and possible percutaneous coronary intervention because of her continuing unstable hemodynamic status.

Coronary angiography showed no evidence of obstructive coronary artery disease. Findings on left ventricular angiography were markedly abnormal, with a large area of apical akinesis. The ejection fraction was 15%. Left ventricular end-diastolic pressure...
pressure was 28 mm Hg. The diagnosis was acute AMI due to coronary vasospasm. She was transferred to the cardiac intensive care unit for continued monitoring and nursing care. Her hemodynamic status remained unstable, and intubation was required. A pulmonary artery catheter was inserted at the bedside to assist in managing hemodynamic status and titrating vasoactive agents. Her hospital course continued to be slow and complicated. Brain natriuretic peptide was elevated throughout the hospitalization, indicating continued left ventricular dysfunction.

Echocardiography on day 17 showed dramatic improvements in left ventricular ejection fraction: an increase to 40%. Because of this significant change in left ventricular function, the diagnosis was revised from AMI to takotsubo cardiomyopathy, or apical ballooning. The patient continued to slowly progress and was discharged home on day 28. Discharge medications included carvedilol 3.125 mg twice daily, lisinopril 5 mg/d, aspirin 325 mg/d, nicotine patch, 21 mg/d topically, pantoprazole 40 mg/d, thiamine 100 mg/d, and folic acid 1 mg/d.

Conclusion

Transient left ventricular apical ballooning, or takotsubo cardiomyopathy, is an uncommon abnormality with signs and symptoms that mimic those of AMI. Making the diagnosis is difficult without early evaluation via echocardiography or cardiac catheterization. Apical ballooning should be considered as a possible diagnosis by emergency department physicians and cardiologists when patients, especially postmenopausal women whose onset of signs and symptoms coincides with some type of psychosocial or physical stressor, have chest pain and ST-segment elevation or depression. Critical care nurses need to be familiar with apical ballooning because some patients with the abnormality have altered hemodynamic parameters and require vasoactive agents and intra-aortic balloon pumping. No significant coronary lesions account for the markedly abnormal left ventricular akinetic and hypokinetic changes that occur. Apical ballooning is reversible; patients have dramatic improvements in left ventricular wall motion and ejection fraction by 30 days after onset of signs and symptoms. Treatment is empirical and supportive. The prognosis is generally favorable, although some deaths have been reported, usually the result of irrecoverable cardiogenic shock, refractory ventricular arrhythmias, or other catastrophic cardiovascular event.

Financial Disclosures

None reported.

Acknowledgments

I acknowledge the assistance of Jane Eymer, RN, BSN, University of California, Davis Medical Center, Sacramento, California; Siobhan Geary, RN, MS, CNS, Thomas Rhodes, RN, BSN, and Julie Chester Wood, RN, MS, CNS, Sutter Medical Center, Sacramento; and Evelyn Reilly, RN, MS, CNS, Sutter Roseville Medical Center, Roseville, California, in the development of this article.

References

3. Dote K, Sato H, Tateishi H, Uchida T, Ishihara M. Myocardial stunning due to simul-


1. What do the signs and symptoms of apical ballooning mimic?
   a. Sick sinus syndrome
   b. Acute myocardial infarction
   c. Mitral valve rupture
   d. Aortic stenosis

2. Which of the following statements is false?
   a. The signs and symptoms of transient left ventricular apical ballooning include chest pain, ST-segment changes, and the release of cardiac biomarkers.
   b. The symptoms of transient left ventricular apical ballooning are caused by ischemic coronary artery disease.
   c. The abrupt onset of extensive ballooning occurs most often in postmenopausal women after a traumatic stressor.
   d. Although the onset is sudden and dramatic, apical ballooning is transient and reversible.

3. What is the incidence of apical ballooning in women compared to men?
   a. 2 to 4 times higher
   b. 4 to 7 times higher
   c. 6 to 9 times higher
   d. 8 to 11 times higher

4. Which of the following mechanisms is thought to initiate apical ballooning?
   a. Spasm of the arteries related to lack of estrogen in the bloodstream
   b. Spasm of the arteries related to plaque rupture
   c. Stunning of the myocardium related to unknown toxin
   d. Ectopic foci in the right atrium

5. Which of the following statements is false?
   a. Electrocardiographic (ECG) findings of deeply inverted T waves are common in the recovery phase.
   b. ECG findings are variable and cannot be used to diagnose apical ballooning.
   c. Many patients have a markedly prolonged QT interval.
   d. ECG findings commonly show ST-segment elevation in leads V1 through V4.

6. Which 2 studies reveal the characteristic systolic ballooning of the ventricle?
   a. Cardiac biomarkers and ECG
   b. Echocardiogram and magnetic resonance imaging
   c. Cardiac catheterization and positron-emission tomography scan
   d. Echocardiogram and left ventriculography

7. What are the findings on coronary angiography for patients with apical ballooning?
   a. Occlusive lesion in left anterior descending coronary artery
   b. Occlusive lesion in first diagonal
   c. No significant disease
   d. First obtuse marginal spasm

8. Which of the following statements is false?
   a. Management of apical ballooning is largely empirical.
   b. Fibrinolytic agents are used to treat patients with apical ballooning.
   c. Antiarrhythmic drugs are administered for ventricular arrhythmias.
   d. Short-term anticoagulation may be given to prevent ventricular thrombus.

9. What is the most common complication of apical ballooning?
   a. Left ventricular rupture
   b. mural thrombus
   c. Left ventricular failure
   d. Heart block

10. What is the general outcome for patients with apical ballooning?
    a. High mortality rate because of ventricular rupture
    b. Left ventricular function improves rapidly
    c. ECG improves rapidly and resolves within 7 days
    d. Mortality rate of 15% is reported

11. Which of the following medications is never indicated for apical ballooning?
    a. Eptifibatide
    b. Dopamine
    c. Dobutamine
    d. Enoxaparin

12. What was the common factor in the 3 case studies presented in the article?
    a. All had an ejection fraction of less than 30%
    b. All were diagnosed correctly within 48 hours
    c. All had troponin levels greater than 2.0 ng/mL
    d. All were postmenopausal women

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

1. □ a □ b □ c □ d
   2. □ a □ b □ c □ d
   3. □ a □ b □ c □ d
   4. □ a □ b □ c □ d
   5. □ a □ b □ c □ d
   6. □ a □ b □ c □ d
   7. □ a □ b □ c □ d
   8. □ a □ b □ c □ d
   9. □ a □ b □ c □ d
   10. □ a □ b □ c □ d
   11. □ a □ b □ c □ d
   12. □ a □ b □ c □ d

Name ___________________________ Member # __________
Address __________________________
City __________ State ______ ZIP ______
Country __________ Phone ______
E-mail __________________________
RN Lic. 1/St ____________________ RN Lic. 2/St __________________
Payment by: □ Visa □ M/C □ AMEX □ Discover □ Check
Card # __________ Expiration Date ______
Signature _________________________

The American Association of Critical-Care Nurses is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center’s Commission on Accreditation. AACN has been approved as a provider of continuing education in nursing by the State Boards of Nursing of Alabama (#ABNP0062), California (#03036), and Louisiana (#ABN12). AACN programming meets the standards for most other states requiring mandatory continuing education credit for relicensure.
Transient Left Ventricular Apical Ballooning
Brenda McCulloch

Crit Care Nurse 2007;27 20-27
Copyright © 2007 by the American Association of Critical-Care Nurses
Published online http://ccn.aacnjournals.org/

Personal use only. For copyright permission information:
http://ccn.aacnjournals.org/cgi/external_ref?link_type=PERMISSIONDIRECT