Takotsubo Cardiomyopathy: A Case Study of Stress Induced Transient Left Ventricular Apical Ballooning Syndrome

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Abstract

Takotsubo cardiomyopathy, also known as transient left ventricular apical ballooning syndrome, is an increasingly recognized differential diagnosis among postmenopausal women with symptoms of an acute myocardial infarction following an abrupt psychological or physical stressor. Although pathophysiology remains unclear, suspicion of catecholamine mediated myocardial stunning is highly favored as myocardial function returns to normal within days to a few weeks. Treatment is aimed at supportive measures including reducing anxiety, alleviating pain, maintaining heart contractility, monitoring fluid balance, and preventing and treating complications.

INTRODUCTION

Takotsubo cardiomyopathy also known as transient left ventricular apical ballooning syndrome, ampulla cardiomyopathy, stress-induced cardiomyopathy, hypocatecholamine-induced acute myocardial stunning, and broken heart syndrome is an acute reversible cardiomyopathy characterized by transient regional wall motion abnormalities of the left ventricular mid segments with or without apical involvement and hyperkinesis of the basal segments.(1) Takotsubo cardiomyopathy is an increasingly recognized differential diagnosis among postmenopausal women with suspected acute coronary syndromes following an abrupt psychological or physical stressor.(2) Takotsubo cardiomyopathy was first described in the early 1990s in Japan and named for the Japanese fishing pot with a narrow neck and a wide base used to trap octopus (Figure 1). Under fluoroscopy during cardiac catheterization, the left ventricle shows the peculiar appearance of a rounded bottom and a narrow neck on end-systolic left ventriculogram.(2) Transthoracic echocardiography confirms dysfunctional left ventricular contractile pattern with initial ejection fractions ranging from 20–40%.(3) Left ventricular wall motion abnormalities rapidly normalize to 60–75% within days to weeks without revascularization.(3) The pathophysiology remains unclear, but catecholamine mediated myocardial stunning is the most favored explanation to transient left ventricular apical ballooning syndrome. The article will present a case of transient apical ballooning syndrome following an acute episode of stress in a postmenopausal woman while teaching a Sunday school lesson.

Figure 1

CASE STUDY

Ms. L, 79 year old Caucasian female presented to the emergency department (ED) after sudden onset of severe

midsternal, nonradiating chest pain occurring about 45 minutes prior to arrival. While teaching Sunday school, the patient reportedly became quite “flushed” and “excited” during an intense religious discussion. She reported dizziness, slight nausea, diaphoresis, and palpitations (“I thought I would faint”). She acknowledged having had these similar symptoms prior to hospitalization. Medical personnel reported chest pain resolution just prior to ED arrival. The patient’s past medical history included hypertension, supraventricular tachycardia and paroxysmal atrial fibrillation with failed ablation, transient ischemic attack, hyperlipidemia, gastroesophageal reflux disease and frequent anxiety reactions. Home medications were warfarin, lisinopril, amiodarone, omeprazole, hydrochlorothiazide, simvastatin, tramadol, carvedilol, calcium and multiple vitamins. Her three children and families did not visit but one to two times yearly, and had not done so since her husband passed away six months prior. She was immediately supported only by her “church family” and friends.

Ms. L was admitted to the intensive care unit. Routine cardiac care was initiated for an acute coronary syndrome. Serial cardiac biomarkers elevated with troponin I (4.2) and creatinine kinase/MB (469/48). Serial electrocardiograms revealed sinus rhythm with first degree atrioventricular block and nondiagnostic T-wave inversion in lead III. Chest x-ray was normal. Home medications were continued except warfarin. Intravenous vitamin K was administered to reverse an international normalized ratio of 2.7. The following morning she was taken for cardiac catheterization. Coronary angiography with left ventriculogram revealed no obstructive coronary artery disease (Figure 2), no mitral regurgitation, left ventricular end-diastolic pressure 22 mmHg, ejection fraction 40% and a large apical akinetic segment (Figure 3). The patient was diagnosed with Takotsubo cardiomyopathy.

Figure 2

Figure 2: Normal right coronary artery

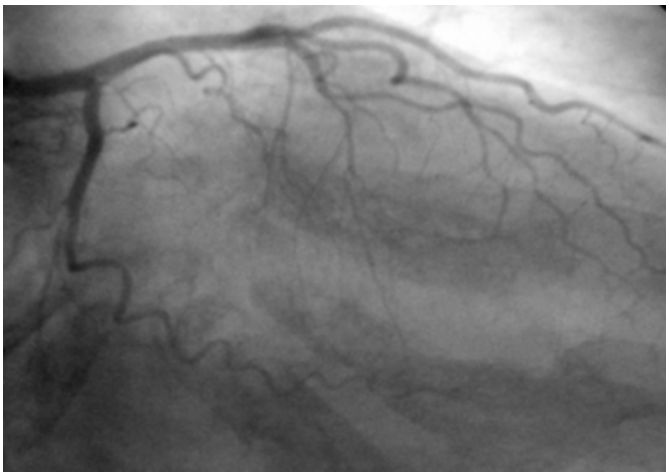


Figure 3

Figure 2: End-diastole during left ventriculogram appears essentially normal

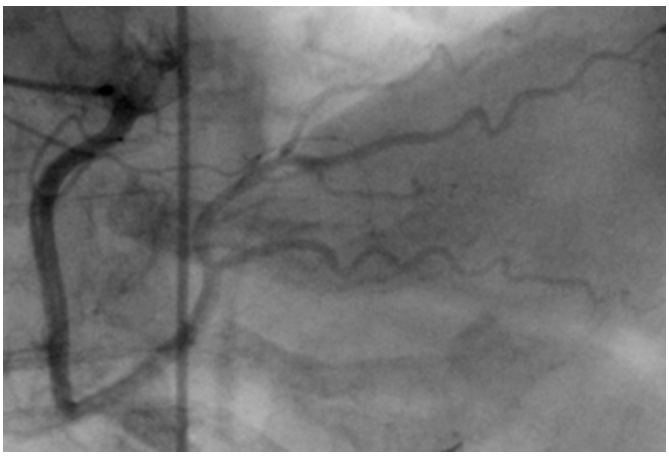


Figure 4

Figure 3: Normal left coronary artery

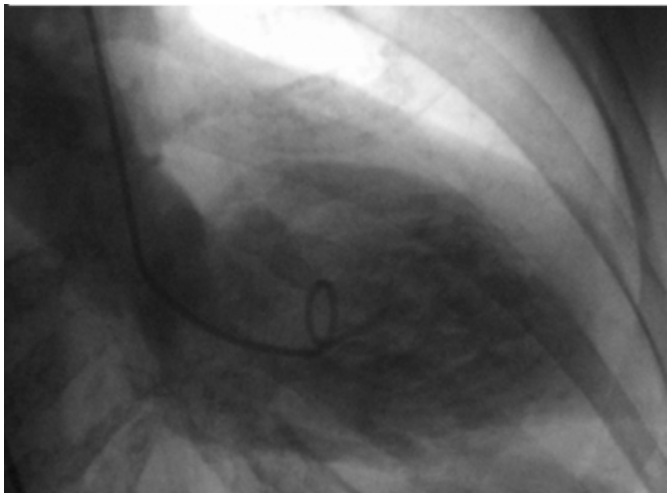
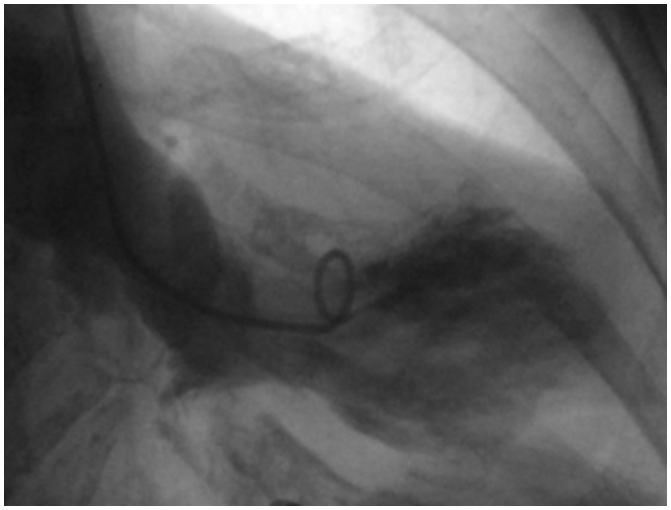


Figure 5

Figure 3: End-systole during left ventriculogram depicting apical ballooning syndrome.



PATHOPHYSIOLOGY

The pathogenesis of the Takotsubo cardiomyopathy is still unknown. Many explanations have been proposed including multi-vessel coronary vasospasm, abnormalities in coronary microvascular function and catecholamine mediated cardio-toxicity provoked by emotional or physical stress.(4) Bybee et al. (1) reported on patterns of abnormal coronary flow in the absence of obstructive coronary artery disease in patients with stress-related myocardial dysfunction. Wittstein et al. (3) noted multi-vessel coronary vasospasm on cardiac catheterization in 70% of Takotsubo cardiomyopathy patients. Sharkey et al. (5) could not explain diffuse wall motion abnormalities by vasospasm of any single coronary artery. Magnetic resonance imagery has not revealed scarring normally associated with acute myocardial infarction.(5) Gadolinium enhancement has not indicated any reversible ischemia.(5)

The frequent occurrence in postmenopausal women has not been explained, but changes in autonomic control of the cardiovascular system and in baroreceptor sensitivity have been suspected.(6) Lavi et al. (7) added that cardiac vagal tone and baroreflex sensitivity decreased significantly in postmenopausal women. Furthermore, cardiovascular beta-adrenoreceptor responsiveness decreased and alpha 1-adrenoreceptor responsiveness increased yielding sympathetic dominance to replace parasympathetic dominance as the main regulator of the cardiovascular system.(7) Changes would be associated with acute stress and include heart rate increases and vasoconstriction.(6)

Wilkinson et al. (8) noted epinephrine was released from the heart at rest and during spontaneous panic attacks. Epinephrine and norepinephrine samples obtained from the coronary sinus during panic episodes have demonstrated reduced extraction of norepinephrine and epinephrine during transit through the heart.(9, 10) Norepinephrine transporter impairment limits catecholamine effects on the heart and has the potential to increase cardiac responses to catecholamine surges during panic attacks and other stressful events. Patients with Takotsubo cardiomyopathy have local release of catecholamines in myocardial tissue.(6)

Therefore, the most favorable pathogenesis to Takotsubo cardiomyopathy development is direct myocardial injury caused by catecholamine medicated myocardial stunning.(3) Direct myocyte injury occurs through formation of oxygen-deprived free radicals which decrease myocyte viability. Free radicals interfere with sodium and calcium transporters increasing calcium influx, thus cellular calcium overload.(3) Mononuclear inflammatory infiltrates and myocyte necrosis occurs and can be depicted by myocardial tissue biopsy. Adrenoceptor density is higher in the cardiac apex compared with other areas of the myocardium accounting for apical and mid left ventricular contractile abnormalities. Basal segments are spared giving the distinctive shape noted on ventriculography.(3)

INCIDENCE, PREVALENCE AND MORTALITY

The prevalence of Takotsubo cardiomyopathy is estimated to range between 0.7 to 2.5%.(11) Female predominance is 90.7%. The mean age of 62 to 76 years.(11) Hospital mortality rates range from 0–8% (1) and are lower than for

myocardial infarction.(12) To date, the largest patient series (n=88) had an overall hospital mortality rate of only 1%.(1) Long-term survival is similar to an age-matched and gender-matched population.(13) Regnante et al. (14) observed a trend in the time of year when Takotsubo cardiomyopathy was most often diagnosed. In contrast with myocardial infarction occurrences which typically peaked in the winter months, Takotsubo cardiomyopathy occurrences distinctly spiked during the summer months.(14) Thus the Takotsubo cardiomyopathy syndrome does not appear to follow the same typical seasonal pattern as the myocardial infarction as documented in the National Registry of Myocardial Infarction.(14)

Clinical presentation is diverse. Population bias is evident as most reported cases have occurred in high stress urban rather than rural settings.(12, 15) While apical ballooning occurs more often in postmenopausal women, the roles of sex hormones and their impact on the nervous system and catecholamine metabolism remain unclear.(16) Kumar et al. (17) suggested genetic predisposition through “familial clustering” as a risk factor for Takotsubo cardiomyopathy development. Mothers and daughters were observed to have similar presentations at diagnosis.(17) No data currently exists on frequency of patients with residual long-term left ventricular impairment.(18) Almost all patients experience a return to normal in myocardial function within a few weeks, the majority in 6+3 days.(19) However, since apical ballooning can be complicated by left ventricular rupture, Takotsubo cardiomyopathy is newly recognized as a cause of sudden cardiac death in up to 3% of patients.(19)

CLINICAL PRESENTATION

The diagnosis of Takotsubo cardiomyopathy is confirmed by presence of all four Mayo Clinic diagnostic criteria: transient contractile dysfunction of the mid-left ventricular segments with or without apical involvement, extending beyond a single epicardial vascular distribution; absence of obstructive coronary disease or angiographic evidence of acute plaque rupture; new electrocardiographic abnormalities (either ST-segment elevation, T-wave inversion, or both) or elevated cardiac troponin; and the absence of a pheochromocytoma or myocarditis.(12) The most common symptom associated with Takotsubo cardiomyopathy presentation is a severe sudden onset of substernal chest pain after an acute emotional or physical stressor.(12) Dyspnea, syncope and palpitations have also been described. Takotsubo cardiomyopathy mimics an acute coronary syndrome in 1-2% of all presentations.(12)

Electrocardiographic changes seen in transient left ventricular apical ballooning syndrome can be similar to ST elevation myocardial infarction including T wave inversions, new-onset bundle branch block or new, sometimes transient, Q waves. However, the Takotsubo cardiomyopathy electrocardiogram typically shows ST segment elevations in leads V4-V6, absence of reciprocal changes in inferior leads, and absence of abnormal Q waves.(20) Pathologic Q-waves are present in only 6–31% of patients.(3) Dib et al. (21) reported the absence of ST-segment elevation in two-thirds of patients investigated with Takotsubo cardiomyopathy. The remaining one-third had no significant electrocardiographic changes.(21) Clinical and echocardiographic characteristics as well as patient outcomes were similar.(21) Biomarkers (troponin and creatinine kinase) are often elevated at time of presentation but do not typically follow the same rise and fall patterns observed in conventional myocardial infarctions.(1)

TRIGGERS

Takotsubo cardiomyopathy has been documented following acute physiologic triggers such as acute medical conditions or surgery have been documented in 17–70% of patients.(1) Haghi et al. (22) suggested sudden hemodynamic deterioration in the intensive care unit requiring vasopressor support and/or electrocardiographic abnormalities consisting of ST-segment elevation, ST-segment depression or T-wave inversion may represent Takotsubo cardiomyopathy. Acute emotional stressors such as accidents, deaths, quarrels, and life-changing events have been implicated in 14–38%.(1) Watanabe et al. (23) diagnosed transient left ventricular apical ballooning syndrome as a result of emotional stress provoked by the earthquake in Niigata, Japan on October 23, 2004.

Depression and anxiety syndromes often coexist with heart disease.(24) Postmenopausal women have a high frequency of both depression and anxiety associated with episodic symptoms, including acute panic attacks.(6) Sun et al. (6) suggested Takotsubo cardiomyopathy may occur in postmenopausal women as a consequence of co-morbid psychiatric diagnoses and normal age-related changes in autonomic control of the cardiovascular system. Wassertheil-Smoller et al. (25) and Smoller et al. (26) reported surveys indicating 15.8% of postmenopausal women have depressed mood and 17.9% report either full-blown panic attacks or symptom limited attacks.

MANAGEMENT AND IMPLICATIONS TO ADVANCED PRACTICE NURSING

Supportive measures comprise the cornerstone of pharmacological and mechanical therapy in the treatment of Takotsubo cardiomyopathy. The majority of patients are initially treated as an acute anterior wall myocardial infarction or acute coronary syndrome, as cardiac biomarkers and electrocardiographic changes may be present.(1) Prompt recognition of left ventricular apical ballooning is needed to prevent unnecessary use of fibrinolytic agents, such as tissue plasmogen activators.(1) Fibrinolytic agents may cause serious bleeding disorders, cholesterol embolism, cardiogenic shock, heart failure, pericardial effusion, myocardial rupture and electromechanical dissociation.(36) Patients are typically prescribed aspirin, beta blockers, angiotensin converting enzyme inhibitors or angiotensin receptor blockers, and statins.(14) Calcium channel antagonists and nitrates are also prescribed.(14, 27)

Patient symptoms vary from mildly symptomatic to critically ill.(28) The advanced practice nurse (APN) must be prepared to address common complications of Takotsubo cardiomyopathy including heart failure, pulmonary edema, dynamic outflow tract obstruction and cardiogenic shock. (11, 12) Emergent coronary angiograms are frequently performed by the interventional cardiologist to definitively diagnose left ventricular apical ballooning syndrome. Twenty-percent of patients require the insertion of an intra-aortic balloon pump (27, 28) to decrease left ventricular workload, decrease afterload, improve contractility and increase stroke volume.(29) The APN may be required to initiate dobutamine or milrinone intravenous therapy as inotropic support of left ventricular failure.(28) Diuretics are given for heart failure and pulmonary edema. Pulmonary artery and/or arterial catheters may be inserted to assist in monitoring hemodynamic stability including cardiac output and cardiac index.(29) Left ventricular assist devices are implanted if the patient experiences severe cardiac pump failure.(27) The APN must closely monitor the patient for third-degree atrioventricular blocks and ventricular arrhythmias. Left ventricular thrombus formation and free wall rupture have also been reported. (1, 11)

Long term benefit to any treatment strategy is uncertain as patients have demonstrated favorable prognosis and outcomes regardless of the prescribed pharmacological therapy.(1, 3) Angiotensin converting enzyme inhibitors or angiotensin receptor blockers, indicated for left ventricular dysfunction are commonly continued until full recovery of systolic function.(12) Regnante et al. (14) asserted that patients on angiotensin converting enzyme inhibitors carried a protective effect against further development of critical illness. However, emerging data have shown limited beta blocker, calcium channel antagonist and nitrate efficacy to prevent Takotsubo cardiomyopathy occurrence.(30) Statins and aspirin are useful in preventing coronary artery disease, but fixed atherosclerotic plaques are essentially absent in Takotsubo cardiomyopathy. Vessel erosion or nonobstructive plaque rupture is an unlikely causation contributing to syndrome pathogenesis,(30) so statins and aspirin may be omitted when a definitive diagnosis of Takotsubo cardiomyopathy is made. The APN may choose to prescribe short-term anticoagulation with heparin or warfarin to prevent and/or treat mural thrombus formation in the setting of severe left ventricular systolic dysfunction.(1) Selective serotonin reuptake inhibitor antidepressants (SSRIs) are safe and effective for use in patients with heart disease and moderate, severe, or recurrent depression (32, 33) and may be considered in the treatment of post-menopausal women with Takotsubo cardiomyopathy. However, Sherwood et al. (34) warn of the unexpected association of antidepressant medications with worsening clinical outcomes.

CONCLUSION

Ms. L's warfarin was resumed and all previous home medications were continued at discharge. She followed two weeks later in the out-patient cardiology clinic with the APN. Transthoracic echocardiography was performed revealing a 65% ejection fraction and complete resolution of prior large apical akinetic segment. She denied any return of previous signs and symptoms of heart failure or chest pain. The left ventricular apical ballooning appearance of Takotsubo cardiomyopathy had resolved.

Takotsubo cardiomyopathy is an important entity to be recognized by the APN. (35) Differential diagnosis should be considered among postmenopausal women presenting with characteristic signs and symptoms of an acute coronary syndrome after an emotional or physical stressor. (2) ST-segment changes (21) and cardiac biomarker elevations (1) may or may not be evident. Coronary angiography typically reveals no significant coronary lesions to account for the marked left ventricular wall motion abnormalities.(12) The left ventricular apical ballooning is transient and the majority

of patient myocardial function returns to normal within days to a few weeks.(19) The APN should aim treatment goals at reducing anxiety, alleviating pain, maintaining contractility, monitoring fluid balance and preventing and treating complications.(29) With supportive care, prognosis is favorable.(1, 3)

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