Hemodynamic support with TandemHeart™ in tako-tsubo cardiomyopathy – a case report

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Submitted: 8 July 2009
Accepted: 13 July 2009

Arch Med Sci 2010; 6, 6: 971-975
DOI: 10.5114/aoms.2010.19311

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Abstract

Tako-tsubo cardiomyopathy is characterized by chest pain, electrocardiographic abnormalities mimicking acute myocardial infarction, akinesis or dyskinesis of apical or mid left ventricular segments, and the absence of obstructive coronary artery disease. Tako-tsubo cardiomyopathy is usually a potentially reversible form of cardiac dysfunction. A careful literature search revealed no previous report of a patient requiring mechanical circulatory support in tako-tsubo cardiomyopathy. We report a patient with tako-tsubo cardiomyopathy, ventricular fibrillation, and hemodynamic instability requiring a left ventricular assist device (TandemHeart™) followed by improvement of left ventricular ejection fraction to 45%.

Key words: tako-tsubo cardiomyopathy, TandemHeart™

Introduction

Tako-tsubo cardiomyopathy is a unique syndrome characterized by chest pain, electrocardiographic abnormalities mimicking acute myocardial infarction, akinesis or dyskinesis of apical or mid left ventricular segments, and the absence of obstructive coronary artery disease. Tako-tsubo cardiomyopathy is also called transient left ventricular apical ballooning syndrome and is usually a potentially reversible form of cardiac dysfunction. Initial reports of tako-tsubo cardiomyopathy did not include clinical heart failure, ventricular fibrillation, or sudden cardiac death. Refractory ventricular arrhythmias are now reported in up to 15% of patients with this syndrome. A careful literature search revealed no previous report of a patient requiring mechanical circulatory support in tako-tsubo cardiomyopathy. We report a 51-year-old patient with tako-tsubo cardiomyopathy, ventricular fibrillation, and hemodynamic instability requiring a left ventricular assist device (TandemHeart™) followed by improvement of left ventricular ejection fraction to 45%.

Case report

The patient is a 51-year-old woman with a past medical history of hypertension and breast cancer, status post right mastectomy with radiation therapy. A few days prior to presentation, the patient had a stressful argument with family members. On the day of presentation,

the patient developed a sudden onset of chest pain and collapsed a few minutes later. Cardiopulmonary resuscitation was initiated in the field. When emergency medical services arrived, she was found to have ventricular fibrillation and was successfully defibrillated with 200 J. The patient was transferred to the emergency department of a nearby community hospital. On arrival, she was hemodynamically stable, but had another episode of ventricular fibrillation. The patient was again successfully resuscitated and endotracheal intubation with mechanical ventilation was instituted.

A 12-lead electrocardiogram showed diffuse ST-segment elevations. The patient underwent emergency cardiac catheterization which revealed normal coronary arteries except for a 20% nonobstructive narrowing of the distal left anterior descending coronary artery. Ventriculography showed severe mid and apical hypokinesis with an estimated left ventricular ejection fraction of less than 10%. During the procedure, the patient became hypotensive and was started on norepinephrine, epinephrine, and vasopressin. An intraaortic balloon pump was inserted. The chest roentgenogram showed bilateral pulmonary vascular congestion and a right lower lobe opacity. The patient was treated with an acute respiratory distress syndrome protocol and was also started on inhaled nitric oxide at 20,000 ppm.

The patient was transferred to the tertiary medical center Westchester Medical Center/New York Medical College for further management. On arrival, the blood pressure was 88/50 mmHg, the pulse was regular with a rate of 102/min, and the respiratory rate was 24/min. Physical examination was normal except for diffuse rhonchi heard throughout both lung fields, a regular cardiac rhythm with a ventricular rate of 102 beats/minute, diminished intensity of the first heart sound, and a left ventricular third heart sound gallop heard at the point of maximum apical impulse.

A 12-lead electrocardiogram (Figure 1) showed sinus tachycardia and diffuse ST-segment elevations in numerous leads. A chest roentgenogram showed bilateral pulmonary vascular congestion and a right lower lobe opacity.

Normal laboratory test results included hemoglobin, hematocrit, platelet count, random blood glucose, serum creatinine, sodium, potassium, chloride, calcium, magnesium, bicarbonate, total bilirubin, alkaline phosphatase, and international normalized ratio. The white blood cell count was 16,500 cells/mm^3. Blood urea nitrogen was 20 mg/dl. Serum aspartate aminotransferase was 212 U/l. Serum alanine aminotransferase was 139 U/l. Serum total protein was 4.7 g/dl. Serum albumin was 3.3 g/dl. Partial thromboplastin time was 36 sec. Serum creatine kinase was 779 IU/l. Serum creatine kinase MB was 43.6 IU/l. Serum troponin I was 19.73 ng/ml. Serum lactate dehydrogenase was 544 U/l.

The patient was intubated with an endotracheal tube using a 7 French catheter. A Swan-Ganz catheter was inserted in the right internal jugular vein. The right atrial pressure was 12 mmHg, the right ventricular pressure was 22/10 mmHg, the pulmonary artery pressure was 25/16 mmHg, the pulmonary capillary wedge pressure was 8 mmHg, the cardiac output was 2.9 l/min, the cardiac index was 1.7 l/min/m2, the systemic vascular resistance was 780 dyne-sec^{-1} cm^{-5}, the pulmonary artery oxygen saturation was 41%, and the arterial oxygen saturation was 70%. The patient was treated with inotropic and vasopressor drugs.

The day after admission, a TandemHeart^{TM} was implanted. Hemodynamic values were obtained that day, 1 day after implantation, 2 days after

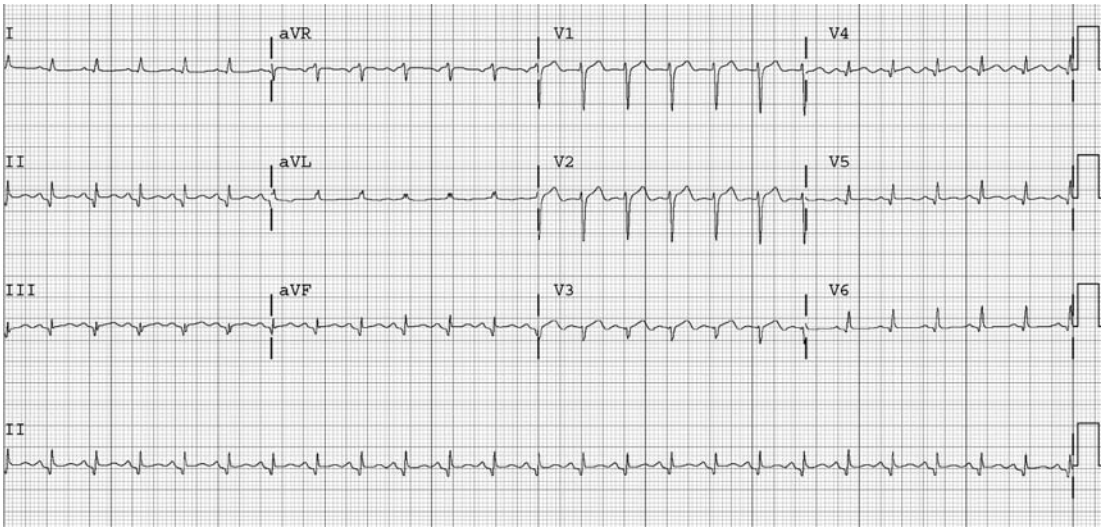

Figure 1. 12-lead electrocardiogram with diffuse ST-segment elevations

implantation, and 4 days after implantation. The right atrial pressures were 13, 10, 9, and 12 mmHg. The right ventricular pressure (peak/mean) was 22/10 mmHg. The pulmonary artery pressures (peak/mean) were 21/15, 29/18, 29/16, and 34/24 mmHg. The pulmonary capillary wedge pressure was 8 mmHg. The pulmonary artery oxygen saturation values were 72%, 72%, 74%, and 73%. The cardiac outputs were 5.8, 6.4, 6.8, and 7.0 l/min. The cardiac index values were 3.3, 3.7, 3.9, and 4.6 l/min/m^2. Systemic vascular resistance values were 857, 1048, 1028, and 747 dyne-sec^{-1} cm^{-5}. Left ventricular ejection fractions were 10% , 21%, and 42% 4 days after implantation. Tandem flow values were 3.4, 3.6, 3.09, and 2.6 l/min. Speed was 6350, 6950, 6350, and 5950 rotations per minute. The patient was treated with epinephrine, milrinone, and vasopressin on the day of TandemHeartTM implantation and with milrinone 1 day, 2 days, and 4 days after implantation.

The patient's left ventricular function improved, and the patient was weaned from the use of inotropic and vasopressor agents. After 5 days, the Tandem Heart was explanted. A 12-lead electrocardiogram (Figure 2) showed reduction of the ST-segment elevations. A 2-dimensional echocardiogram taken 6 days after admission showed a left ventricular ejection fraction of 45%. None of the 2-dimensional echocardiograms during hospitalization showed evidence of right ventricular involvement. The patient was discharged home without any neurological deficit. At 8-month follow-up, the patient is asymptomatic with a left ventricular ejection fraction of 55%.

Discussion

Tako-tsubo cardiomyopathy is a unique, newly recognized syndrome characterized by chest pain, new electrocardiographic abnormalities mimicking acute myocardial infarction, akinesis or dyskinesis of apical and mid-left ventricular segments, and absence of obstructive coronary artery disease. Tako-tsubo cardiomyopathy is also called transient left ventricular apical ballooning syndrome (TLVABS), a potentially reversible form of cardiac dysfunction. This syndrome was initially found in Japanese patients and was named for the fishing pot with a round bottom and narrow neck that is used for trapping octopi in Japan (octopus is "tako" and pot is "tsubo" in Japanese). Satoh *et al.* [1] were among the first investigators to describe this syndrome.

In a large proportion of persons with tako-tsubo cardiomyopathy, there were significant psychological stressors at the onset of symptoms (i.e., sudden accidents, death in the family, vigorous excitation, or other exacerbating systemic disorders such as cerebrovascular accident, epilepsy, bronchial asthma, renal or urinary tract disease, an acute abdomen, and noncardiac surgery or medical procedures) [2]. Enhanced sympathetic activity is thought to be one of the important mechanisms underlying TLVABS. Excessive adrenergic discharge has been demonstrated to cause transient spasm of the epicardial vessels, producing myocyte injury with subsequent ballooning of the myocardial apex [3]. This is further supported by reports of a tako-tsubo-like pattern in the endocrine crisis from pheochromocytoma [4]. Interestingly, an inverted-tako-tsubo pattern (ballooning of the base) rather than apical ballooning has been described commonly in pheochromocytoma [5-7]. Reversible left ventricular dysfunction with apical hypokinesis has also been described in hyperadrenergic states such as hyperthyroidism [8, 9].

The clinical features of TLVABS were studied in a systematic review of 28 series including

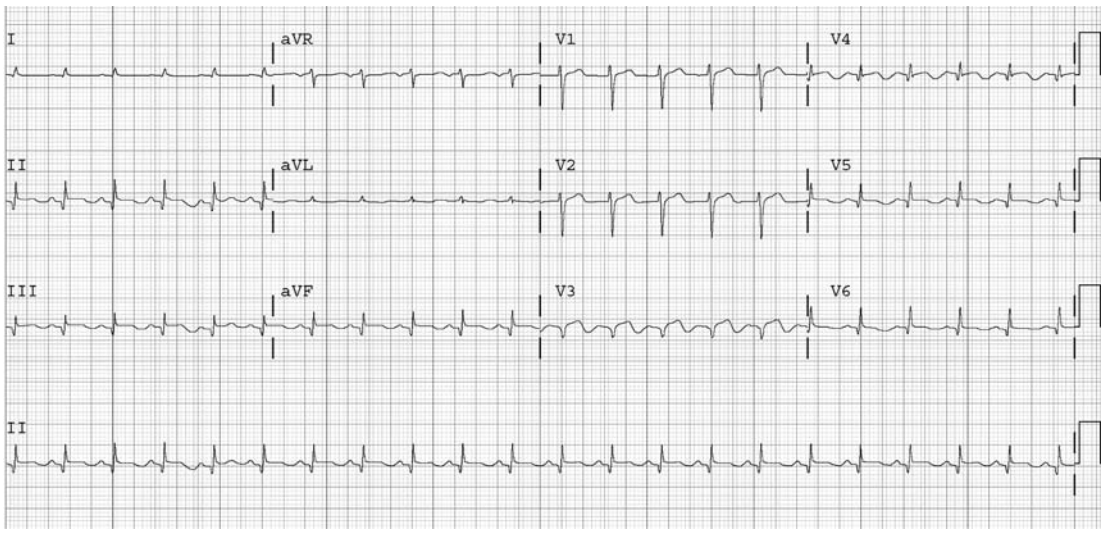

Figure 2. 12-lead electrocardiogram with reduced ST-segment elevations

563 patients [10]. TLVABS was observed in 0.7-2.5% of patients with suspected acute coronary syndrome. It was predominantly observed in postmenopausal women subsequent to a stressful event. It affects women in 90.7% of cases, with a mean age ranging from 62 to 76 years. The most common presenting symptoms include chest pain (83.4%) and dyspnea (20.4%) [10]. Initial presentations with syncope, palpitations, hypotension, ventricular fibrillation and cardiac arrest have also been described [11]. Preceding emotionally stressful events were recognized in 44% and physical stressors in 36.2% [10]. Common electrocardiographic changes include ST-segment elevation (71.1%), T-wave inversion (61.3%) and pathological Q waves (31.1%) [10]. Transient QT prolongation was consistently described in TLVABS [10]. The mean/median QTc ranged from 445.8 to 542 ms. Electrocardiography does not allow reliable differentiation between TLVABS and anterior ST-segment myocardial infarction with enough certainty to preclude coronary angiography. Cardiac biomarkers were elevated in 85% of patients [10]. Coronary angiography showed completely normal coronaries in 87.9% and noncritical luminal stenoses in the other patients [10]. Left ventricular function assessed by echocardiography, left ventriculography or cardiac magnetic resonance imaging typically showed dyskinesis of the left ventricular apical or mid-ventricular segments with a hyperkinetic basal region [10]. The mean left ventricular ejection on admission ranged from 20% to 49.4% and recovered over a period of 18 days on average (range 7 to 37 days) [10]. Transient intraventricular pressure gradients between the apex and the base reflecting dynamic left ventricular outflow tract obstruction have been described in 16.4% [10]. Identification of these clinical features has led to the proposal of Mayo criteria for the diagnosis of tako-tsubo cardiomyopathy [12].

Mayo criteria for the diagnosis of tako-tsubo cardiomyopathy are 1) transient akinesis or dyskinesis of the left ventricular apical and mid-ventricular segments with regional wall-motion abnormalities extending beyond a single epicardial vascular distribution, 2) absence of obstructive coronary disease or angiographic evidence of acute plaque rupture, 3) new electrocardiographic abnormalities (either ST-segment elevation or T-wave inversion), and absence of recent significant head trauma, intracranial bleeding, pheochromocytoma, obstructive epicardial coronary artery disease, myocarditis, and hypertrophic obstructive cardiomyopathy [12].

Cardiomyopathy may result years after treatment with radiation therapy [13] but has not been implicated in the pathogenesis of tako-tsubo cardiomyopathy. It is possible that prior radiation contributed to the malignant course in this patient, who had radiation therapy for breast cancer. Chemotherapy has been implicated in the pathogenesis of tako-tsubo cardiomyopathy [14, 15] but was not administered to this patient.

A variety of serious complications have been reported during the acute phase of tako-tsubo cardiomyopathy [10]. These complications include heart failure and pulmonary edema (15.9%), cardiogenic shock (10.3%) and life-threatening arrhythmias in 14.6%. Isolated cases of left ventricular free wall rupture have also been reported. The overall in-hospital mortality rate was 1.7%. Recurrence of the syndrome is rare (3.1%). Supportive measures are the mainstay of management during the acute phase of tako-tsubo cardiomyopathy. There are no specific therapies that lead to recovery from tako-tsubo cardiomyopathy. Management of patients with this syndrome includes medical management with aspirin, beta blockers, and angiotensin-converting enzyme inhibitors. Patients with severe congestive heart failure will require diuretics, inotropic drugs, and mechanical circulatory support if left ventricular dysfunction is severe. Interim anticoagulation has also been advised in patients with severe systolic dysfunction to prevent left ventricular mural thrombus formation. Severe left ventricular dysfunction warranted the use of inotropic agents in 23.6% of patients and hemodynamic support with intra-aortic balloon-pump counterpulsation in 11.2% of patients [10]. The use of a TandemHeart™ has not been previously reported.

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