

Takotsubo Cardiomyopathy Induced Intraoperative and Postoperative Cardiac Arrests

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Introduction

Takotsubo cardiomyopathy (TC) is a transient left ventricular apical ballooning syndrome which results in an acute, reversible, left ventricular dysfunction. Unlike a myocardial ischemia, TC is not due to coronary artery stenosis. Initially the syndrome was described by a series of five Japanese patients in 1991 [1] and there have been multiple subsequent reports. The syndrome received its name from an octopus trap (tako-tsubo) which is a wide pot that has a narrow opening. This trap resembles the left ventricle of a patient with TC during systole as the apex is rounded and balloons out despite a hypercontractile and narrow base. TC has also been called “broken heart syndrome” because it is usually associated with a stressful event. Although recurrent cases of TC have been reported, this is the first presentation of a patient that experienced recurrent TC induced cardiac arrest in the same perioperative period.

Case Report

Takotsubo cardiomyopathy (TC) is a transient left ventricular apical ballooning syndrome which results in an acute, reversible, left ventricular dysfunction. Unlike a myocardial ischemia, TC is not due to coronary artery stenosis. Initially the syndrome was described by a series of five Japanese patients in 1991 [1] and there have been multiple subsequent reports. The syndrome received its name from an octopus trap (tako-tsubo) which is a wide pot that has a narrow opening. This trap resembles the left ventricle of a patient with TC during systole as the apex is rounded and balloons out despite a hypercontractile and narrow base. TC has also been called “broken heart syndrome” because it is usually associated with a stressful event. Although recurrent cases of TC have been reported, this is the first presentation of a patient that experienced recurrent TC induced cardiac arrest in the same perioperative period.

A 26 year old, 56 kg female with bilateral hearing loss presented for cochlear implant. The patient was ASA 1 and she denied alcohol or illicit drug use. Her urine pregnancy test was negative. The patient received midazolam 2 mg IV in the pre-anesthesia area and she was taken to the operating room. General anesthesia was induced with fentanyl, lidocaine, propofol, and succinylcholine and the patient was intubated. General anesthesia was maintained with desflurane (6%) in air and oxygen. The patient was turned 180° and prepped and draped. Clindamycin was started 15 minutes after induction.

Four minutes later and prior to surgical incision, the patient's rhythm suddenly changed to sinus tachycardia at 150 bpm and her blood pressure increased to 170/107. End-tidal desflurane was 5.9%. Esmolol 50 mg iv was titrated to decrease the patient's heart rate. Blood pressure was unobtainable by automated cuff despite a heart rate of 90

bpm and the femoral pulse was only faintly palpable. Phenylephrine was given with no response. Epinephrine 1 mg iv was given in divided doses with a continued weak pulse and the blood pressure was still not obtainable. Chest compressions were performed for approximately 1 minute to assist in circulating a 1 mg iv bolus of epinephrine. Blood pressure returned to 100/50 and chest compressions were stopped. Central venous access was obtained and a radial arterial line was placed. Blood pressure was maintained with fluid boluses, epinephrine 0.1 mcg/kg/min, and dopamine 5 mcg/kg/min. Frequent arterial blood gases and electrolytes were obtained and replacement of bicarbonate, potassium, and calcium were given. Transesophageal echocardiography (TEE) revealed a left ventricular ejection fraction of 20-25% with poor contractility of the apex and a hypercontractile base. An intraoperative cardiology consult concluded that the TEE was consistent with TC. The patient's oxygen saturation decreased to <90% and she was found to be in pulmonary edema. The PEEP was increased to 14 cm H₂O and furosemide 20 mg iv was administered to obtain an acceptable oxygen saturation. The case was cancelled and the patient was taken to the ICU intubated, sedated, and on dopamine. Subsequent transthoracic echocardiogram (TTE) revealed an extensive LAD distribution wall motion abnormality and apical ballooning (Figure 1). Given the possibility of spontaneous coronary dissection or thrombus, the patient was taken to the cardiac catheterization lab for further evaluation. Coronary angiogram revealed a severely reduced LVEF (18%) with clean coronary arteries and elevated LVEDP (24 mmHg). The patient improved and was extubated the following day and maintained on low dose dopamine.

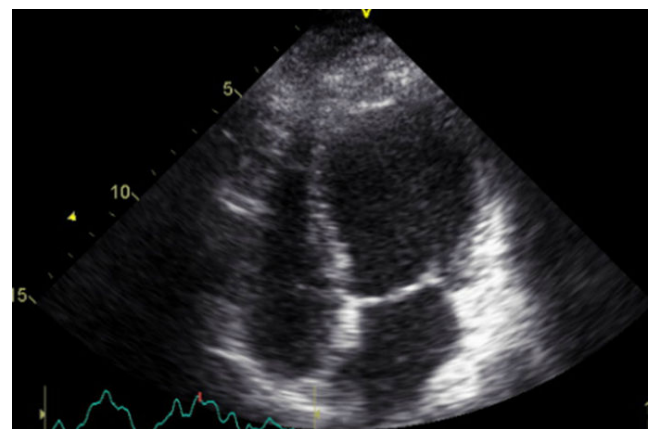


Figure 1: Apical ballooning during systole.

That evening, she had another acute episode of tachycardia and hypertension followed by hypotension and PEA arrest. The patient underwent CPR and she received two iv boluses of epinephrine 1 mg and was placed on epinephrine and dopamine infusions. An IABP was placed. There was clinical suspicion for a pheochromocytoma but an abdominal/pelvic CT was negative. Over the next 24 hours, the epinephrine and dopamine were weaned off, the patient was again extubated, and the IABP was removed. Repeat TTE four days following the initial arrest showed LVEF 55% and no regional wall motion abnormalities (Figure 2).

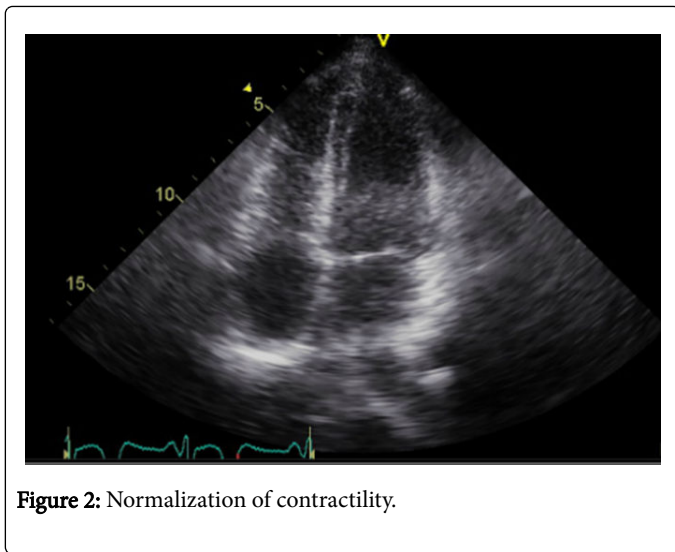


Figure 2: Normalization of contractility.

A cardiology consult recommended waiting at least one year to perform surgery. Urine metanephrines were obtained and ruled out pheochromocytoma. The patient returned 22 months later for cochlear implant surgery. Approximately six weeks prior to surgery the patient was started on metoprolol 25 mg daily which was increased to 50 mg daily three weeks prior to surgery. On the day of surgery, steps were taken to reduce the patient's anxiety including administering midazolam 2 mg IV and dexmedetomidine 1 mcg/kg iv in the preanesthesia area. A sign language interpreter and the patient's spouse were present with the patient at induction in the operating room. The general anesthesia with a laryngeal mask airway was uneventful and the patient was discharged from the hospital the same day.

Discussion

TC occurred in this patient as evident by transient left ventricular apical ballooning despite normal coronary arteries. Although TC usually occurs in postmenopausal women, possibly due to estrogen withdrawal and endothelial dysfunction [2], our patient was young. She noted feeling extremely anxious prior to surgery and she experienced perioperative catecholamine surges which likely led to myocardial stunning. While TC can be associated with EKG changes such as ST changes or QT prolongation, our patient's EKG was normal which occurs in 15% of patients [3]. Our patient's echocardiogram exhibited classic features of TC including decreased ejection fraction, hyperkinetic base, and apical and mid-ventricular akinesis in a non-coronary distribution. One study showed that compared with an acute myocardial infarction due to coronary artery occlusion, the rise in troponins in a patient with TC is only mildly elevated [4], as was the case with our patient. This mild increase in troponins in TC is thought to be due to increased levels of catecholamines due to damage to the

myocardium. Our patient responded to vasopressors and was extubated the following day. Her re-arrest was possibly caused by a repeat catecholamine surge which further raised the suspicion of a pheochromocytoma. CT scans of the chest, abdomen, and pelvis were used to rule out a pheochromocytoma because screening for metanephrines would not have been reliable in a patient receiving dopamine. The patient responded favorably to an IABP which has been described in patients with TC to decrease afterload and improve cardiac output. LVEF usually increases within a mean of 6 days [5] and in our patient TTE four days later showed normal LVEF and no wall motion abnormalities.

Although the exact mechanism of the catecholamine surge associated with TC is unknown, it appears that the excess epinephrine may induce a change in the beta-2 adrenergic receptor which inhibits contractility. Studies in animal models have shown that there is a greater concentration of beta-2 adrenergic receptors at the apex of the heart in TC [6]. This may contribute to wall motion abnormalities and negative inotropic effects in TC. To mitigate effects from catecholamine surges during her subsequent surgery, our patient was administered beta-blockers, dexmedetomidine, and a benzodiazepine. Although there is little evidence, long term beta blocker therapy is encouraged by some centers to help prevent recurrence of TC, and beta-blockers are believed to reduce LVOT obstruction by blocking basal segment hypercontractility [7]. The alpha-2 adrenergic agonist dexmedetomidine has been used in subsequent surgeries in TC patients in an effort to mitigate the perioperative stress response [8].

Conclusion

The stress of surgery may induce intraoperative and postoperative TC. Treatment with vasopressors and an IABP may be necessary. If the patient returns for surgery, one should consider beta-blockade as well as reducing anxiety with anxiolytics and dexmedetomidine.

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