Surviving a Broken Heart: A Case of Intraoperative Cardiac Arrest and Takotsubo Cardiomyopathy

LT Devon Dan, BSN, RN, NC, USN
CDR Chad Moore, DNP, CRNA, NC, USN

Takotsubo cardiomyopathy manifests as global myocardial hypokinesis, a rare challenge for anesthesia practitioners. This report discusses a case in which a seemingly healthy patient presented for open abdominal hysterectomy and experienced intraoperative cardiac arrest requiring cardiopulmonary resuscitation. Takotsubo cardiomyopathy was diagnosed following resuscitation. This case examines risk factors and the intraoperative and postoperative management of a patient with Takotsubo cardiomyopathy.

Keywords: Broken heart syndrome, intraoperative cardiac arrest, stress cardiomyopathy, Takotsubo cardiomyopathy.

Takotsubo cardiomyopathy (TC) is a reversible, sudden-onset cardiomyopathy, thought to be precipitated by high serum catecholamine levels. Clinically resembling acute myocardial infarction, TC may be differentiated from infarction or other cardiomyopathies on an echocardiogram. Left ventricular apical ballooning will be notable during both systole and diastole. This effect may be transient with variable wall-motion abnormalities.

The diagnostic criteria for TC (Table) notably exclude findings consistent with coronary artery disease. The overall incidence is unknown, but an international registry using the diagnostic criteria shown in the Table included 1,750 patients in whom TC was diagnosed between 1998 and 2014. This report describes a case of Takotsubo cardiomyopathy with an onset shortly after incision and retraction for abdominal surgery.

Case Summary
A 47-year-old woman with uterine fibroids presented for a total abdominal hysterectomy. Determined to be otherwise healthy by the surgical team, she was not seen by an anesthesia practitioner until the day of surgery. On the day of surgery, she weighed 51 kg, had a height of 157.5 cm (62 in) and a body mass index of 20 kg/m², and reported that she had no allergies and was not receiving any medications. Her medical history was remarkable for dysmenorrhea and menorrhagia. She was an active runner without functional limitations or known cardiovascular disease. Her surgical history included an uncomplicated dilation and curettage. She reported no family history of anesthetic complications. Her social history included a 30 pack-year smoking history and no regular alcohol use, and her spouse was away for military deployment.

The patient presented the day of surgery, extremely anxious by self-report, a point reinforced by her family members present. Pre-procedural vital signs included a blood pressure of 114/77 mm Hg, heart rate of 86/min, respiratory rate of 12/min, and 100% oxygen saturation by pulse oximetry on room air. Results of her most recent serum chemistry panel and complete blood cell counts were unremarkable. Her airway was favorable for intubation, lungs sounds were clear bilaterally, and heart tones were normal without murmur. Intravenous (IV) induction and general anesthesia were planned, to be followed by pre-procedural ultrasound-guided transversus abdominis plane (TAP) block.

Sublingual ondansetron, 8 mg, and IV midazolam, 2 mg, were given for antiemetic prophylaxis and anxiolysis. General anesthesia was induced uneventfully with IV fentanyl, 50 μg; propofol, 90 mg; rocuronium, 30 mg; and dexamethasone, 4 mg; it was maintained with 2% sevoflurane. The patient was intubated with a 7.0-mm endotracheal tube and given pressure-controlled ventilation. Cefazolin, 2 g IV, was administered. The TAP block was performed bilaterally under ultrasound guidance using 10 mL of saline for hydrodissection, 10 mL of 1.3%

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<th>Table. Mayo Clinic Diagnostic Criteria for Takotsubo Cardiomyopathy</th>
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- Transient left ventricular wall-motion abnormality
  1. Beyond a single zone of epicardial coronary artery perfusion or
  2. Within a single zone and matching all other criteria, with death during acute phase before wall-motion recovery.
- Absence of occlusive coronary artery disease
- Presence of new electrocardiographic abnormalities or elevation in cardiac biomarkers
- Absence of pheochromocytoma and myocarditis

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liposomal bupivacaine, and 20 mL of 0.25% bupivacaine for each side.

Following the TAP block and during surgical setup, phenylephrine, 50 μg IV, was administered on 3 occasions for mild hypotension, without a substantial reduction in heart rate. Her vital signs remained stable without intervention for another 17 minutes. After surgical incision and during retraction (Alexis O retractor, Applied Medical), a precipitous drop in heart rate was observed. When the heart rate reached 37/min, the surgical team was notified to release any traction in the abdomen while glycopyrrolate, 0.2 mg, was prepared; however, the heart rate continued to decline rapidly. Less than 5 seconds before glycopyrrolate administration, asystole was observed on a 3-lead electrocardiogram (ECG), pulse oximetry was not detectable, and a right carotid pulse was not palpable. Sevoflurane administration was stopped, and 100% oxygen was administered at 12 L/min. Chest compressions were started, and IV epinephrine, 1 mg, was administered. Support staff arrived with a defibrillator, but defibrillation was not indicated with the rhythm interpreted as pulseless electrical activity. During compressions, a second IV catheter and arterial line were placed, end-tidal waveform capnography was more than 20 mm Hg, and oxygen saturation stayed at 100%. Following the first round of chest compressions, a palpable pulse returned.

After the return of spontaneous circulation, the 3-lead ECG displayed sinus tachycardia with a rate of 160/min, unquantified ST-segment elevation was visible in lead II, and a 12-lead ECG was requested. Midazolam, 10 mg IV, was administered in divided doses to maintain amnesia. The surgery was canceled, and her incision was closed. The critical care physician arrived to consult before the patient was admitted to the intensive care unit (ICU). Neuromuscular blockade was reversed with IV sugammadex, 200 mg. She was able to follow commands and was extubated with appropriate extubation criteria met. The 12-lead ECG showed diffuse ST-segment elevation, not confined to a perfusion zone of a single coronary vessel.

The patient was transported to the ICU, where thoracic echocardiograms revealed global hypokinesis and apical ballooning. Takotsubo cardiomyopathy was confirmed, and no cardiac vessel occlusion was found following transfer to a nearby facility performing cardiac catheterization. The patient recovered without further incident.

**Discussion**

Before initial imaging or diagnostic testing, the anesthesia team considered differential diagnoses including local anesthetic systemic toxicity, intraoperative myocardial infarction, and vagally mediated cardiac collapse. Takotsubo cardiomyopathy, also called broken heart syndrome, was the provisional diagnosis on initial images that demonstrated global hypokinesis with apical ballooning of the left ventricle. Later cardiac catheterization findings, which showed no evidence of occlusive coronary artery disease or plaque rupture, confirmed the TC diagnosis. The patient had no neurologic deficits following the event, although after the event she disclosed a history of 2 or 3 unexplained syncopal episodes. Her surgery, severe anxiety, and prolonged absence of her spouse were possible sources of physical and emotional stress. Stress does increase the risk of TC, but it is unknown which of these sources of stress was the most physiologically impactful.

Takotsubo cardiomyopathy is largely thought to be a state of myocardial cell hypercontraction, resulting from progressive damage caused by circulating catecholamines. Hypercalcified dense eosinophilic transverse bands form on the sarcomeres; then, actin and myosin have more available calcium to bind continuously in a state of hypercontraction. Release of reactive oxygen molecules then results in diffuse cardiac cell death. In the presence of these cellular changes, the excessive discharge of catecholamines stuns the myocardium. Individuals at elevated risk of TC may be older than 75 years, female, and have a systolic blood pressure less than 110 mm Hg, a history of pulmonary edema, unexplained syncope, ventricular fibrillation, ventricular tachycardia, left ventricular ejection fraction less than 35%, left ventricular outflow greater than 40 mm Hg, mitral valve regurgitation, apical thrombus, new ventricular septal defect, or contained left ventricular wall rupture. Vagally mediated cardiac collapse, autonomic dysregulation due to smoking, and chronic emotional stress may have contributed to this patient’s event.

**Vagally Mediated Cardiac Collapse.** Vasovagal reflex bradycardia is not uncommon during intraabdominal surgery. Peritoneal stretch stimulates mechanical receptors, sending afferent vagal signals to the brain, which trigger an efferent response of bradycardia and hypotension. Bradycardia (heart rate < 60/min) with systolic blood pressure less than 70 mm Hg may occur in 28.4% to 69.0% of open abdominal procedures, and cardiac arrest resulting from reflex bradycardia has been reported. Release of surgical traction on the abdomen and anticholinergic medications may resolve the bradycardia; however, in cardiac-compromised patients, profound bradycardia can lead to cardiac collapse. Vagally mediated cardiac collapse was likely a factor for cardiac arrest in this patient.

**Compromised Automaticity in Long-term Smokers.** Long-term smoking increases cardiac vulnerability, compromises automaticity, and substantially reduces heart rate variability. Heart rate variability is used to help predict the risk of sudden cardiac death and arrhythmias in patients with myocardial infarction and congestive heart failure. Smoking may have reduced her ability to compensate when faced with the physiologic stress of surgery.
• **Emotional Stress and Myocardial Stunning.** Emotional stress has been shown to precipitate transient left ventricular dysfunction, although a definitive mechanism is not known. Causes such as epicardial coronary arterial spasm, exaggerated sympathetic mediation leading to microcirculatory irritation, and left ventricular outflow tract (LVOT) obstruction have been proposed. Excess catecholamines may cause direct myocyte injury. Although the Mayo Clinic diagnostic criteria for TC exclude pheochromocytoma, the European Society of Cardiology maintains that the excess catecholamines could cause secondary TC.

The clinical presentation of TC is similar to that of an acute myocardial infarction; however, the cardiac catheterization results will be negative for clinically significant occlusion, with minimal elevation in cardiac enzyme levels. The typical finding on echocardiograms is left-ventricular apical ballooning and global or midventricular hypokinesis. The midventricular hypokinesis and apical ballooning seen in this patient's echocardiogram may be partially attributable to her self-reported preoperative emotional stress related to her spouse being on military deployment and her fears about surgery.

• **Local Anesthetic Systemic Toxicity.** Liposomal bupivacaine is not equipotent to standard bupivacaine in terms of systemic release. Considering the dosing described earlier and adding first-hour kinetics of bupivacaine release from the liposomal form, the patient's dose was below her calculated maximum dose of 127.5 mg of free bupivacaine. Small doses of local anesthetic may still influence a compromised myocardium.

• **Anesthetic Implications.** Unusual symptoms such as unexplained syncope or limited functional capacity may give clues to underlying pathologic abnormalities. Risk scoring is used for determining the severity of failure in cases with a preexisting diagnosis of heart failure, but in patients without a preexisting diagnosis or active symptoms, risk scoring is not clinically useful. Anesthesia has a potential to exacerbate TC, considering the anxiety of surgery or sympathomimetic effects from inadequate depth of anesthesia. When a risk of TC can be identified preoperatively, empirical recommendations for perioperative management have been proposed. A slowly titrated IV anesthetic induction with concurrent echocardiographic monitoring has been described in a patient with known TC presenting for surgery. Proposed considerations also include achievement of effective preoperative anxiolysis, deep planes of anesthesia before stimulation, arterial line monitoring, avoidance of LVOT obstruction with adequate preload, and having transesophageal echocardiogram and intra-aortic balloon pump capability with a plan in place for crisis management. Additionally, α-blockade and/or β-blockade may aid in preserving normal cardiac function. Given the reversible nature of TC, if it is discovered in the preoperative phase, it is reasonable to delay elective surgery until regional wall abnormalities and ventricular function return to normal. Inotropes can worsen LVOT obstruction and potentially perpetuate cardiac collapse, and appropriate treatment is negative inotropy. Intraoperative echocardiography is helpful in detecting LVOT obstruction in such cases when cardiac instability is anticipated.

Practitioners may have a heightened suspicion for TC when given these risk factors. Without preoperative functional deficits, TC may not be possible to detect until the intraoperative cardiac event occurs. Given the course of perioperative treatment and diagnostic testing, we postulate that TC was present in our patient before the induction of anesthesia, but it was not clinically detectable with the usual screening for cardiomyopathies by her reported history. Furthermore, a predisposition to TC, smoking, and the stress of surgery limited her capacity to compensate during vagal stimulation, and finally the vagal stretch reflex treated with epinephrine also presumably led to the TC. This case serves to inform the broader anesthesia community of a potential diagnosis to consider in cases of intraoperative cardiac arrest.

**REFERENCES**


**AUTHORS**

LT Devon Dan, BSN, RN, NC, USN, is a student in the Nurse Anesthesia Program at the Daniel K. Inouye Graduate School of Nursing, Uniformed Services University of the Health Sciences at Naval Hospital Jacksonville, Florida. Email: devon.dan@usuhs.edu.

CDR Chad B. Moore, DNP, CRNA, NC, USN, is the phase 2 site director for the Nurse Anesthesia Program at the Daniel K. Inouye Graduate School of Nursing, Uniformed Services University of the Health Sciences at Naval Hospital Jacksonville, Florida. Email: chad.moore@usuhs.edu.

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