

Acute left ventricle failure on induction of anesthesia: a case report of reverse stress cardiomyopathy—presentation, diagnosis and treatment

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Abstract Reverse takotsubo cardiomyopathy (TCM) is a less common variant of classic TCM that presents within a different patient profile and with its own hemodynamic considerations. A 46-year-old woman was admitted to our hospital for laryngoscopy and possible balloon dilatation for tracheal stenosis under general anesthesia. One year prior to this admission, the patient was admitted after a motor vehicle accident with subdural hematoma, subarachnoid hemorrhage, and fracture of the eighth thoracic vertebra. She underwent uneventful anesthesia for thoracic spine surgery and tracheostomy to help her wean from the ventilator during that admission. Since her previous admission, she developed posttraumatic anxiety and depression (a neuropsychiatric disorder triggered by subdural hematoma and subarachnoid hemorrhage) and was treated with anti-anxiety and antidepressant medication. At this admission, the patient developed acute left ventricle failure on induction of anesthesia secondary to reverse TCM. We report a case of reverse TCM, where posttraumatic emotional stress of

a neuropsychiatric disorder combined with physical stress from anesthesia and laryngoscopy triggered TCM in a patient with previous uneventful anesthesia 1 year earlier.

Keywords Anesthesia · Reverse stress cardiomyopathy · Neuropsychiatric disorder · Management

Introduction

Reverse takotsubo cardiomyopathy (TCM) is a syndrome that clinically mimics acute coronary syndrome (ACS) but is not secondary to obstructive coronary artery disease [1]. Patients with reverse TCM have different clinical manifestations, demographics, and laboratory values compared with classic type TCM. This report describes a case of reverse TCM in a woman who had undergone an uneventful anesthesia 1 year earlier but developed reverse TCM on subsequent anesthesia during induction and laryngoscopy. This case report highlights the importance of a newly developed neuropsychiatric disorder that can trigger reverse TCM when, concomitantly, a patient is subject to physical stress from anesthesia and laryngoscopy [2].

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Case report

A 46-year-old Caucasian woman with subdural hematoma, subarachnoid hemorrhage, and fracture of the eighth thoracic vertebra was admitted to our hospital after a motor vehicle accident. She underwent an uneventful anesthesia for thoracic spinal fusion and had a tracheostomy to help her wean from the ventilator. One year later, she was readmitted as an outpatient for laryngoscopy and possible balloon dilatation under general anesthesia to treat

posttracheostomy tracheal stenosis. At this admission, her home medicines included alprazolam, bupropion, citalopram, and tizanidine for anxiety and depression. She had no previous history of cardiac disease or cardiac risk factors, and her functional capacity was adequate, with a metabolic equivalent of task (MET) value >4 .

At the start of the procedure, cardiac and respiratory monitoring was established as per American Society of Anesthesia guidelines. Patient's lungs were preoxygenated, and anesthesia was induced with midazolam (2 mg), lidocaine (100 mg), and etomidate (20 mg). Immediately after induction, the patient developed sinus bradycardia, with a heart rate of 35 beats/min. Glycopyrrolate (0.4 mg) was administered. The airway was secured with an oral endotracheal tube with internal diameter of 6 mm after administration of suxamethonium (100 mg). Equal breath sounds were confirmed and end-tidal carbon dioxide noted.

The patient's systolic blood pressure increased to 180 mmHg, and she developed sinus tachycardia with a heart rate of 140 beats/min. At this point esmolol hydrochloride (total of 80 mg) was administered intravenously. The patient's systolic blood pressure improved to 130–150 mmHg, and her heart rate settled at 120 beats/min. However, her oxygen saturation dropped to 88–90 % and pink frothy sputum was observed in the endotracheal tube. She then became hypotensive, with a systolic blood pressure of 70–80 mmHg with minimum response to phenylephrine (100 μ g) boluses. Phenylephrine infusion was started (250 μ g/min), arterial line and central venous catheters were placed, and the surgical procedure was aborted. The patient was transferred to the intensive care unit with a diagnosis of acute pulmonary edema, hypotension, and tachycardia.

Cardiology and anesthesia intensive care services were consulted. Bilateral diffuse infiltrates were seen on a chest X-ray. Heart rate was recorded as 133 beats/min, and an abnormal left axis deviation was noted via a 12-lead electrocardiograph (EKG). Serial cardiac troponins I were elevated, with values of 0.2, 0.6, and 1.2 ng/ml at 1, 6, and 12 h, respectively. Echocardiogram indicated akinetic mid and basilar left ventricular segments with dynamic apex and a calculated ejection fraction of 23 %, suggestive of reverse TCM (Fig. 1). The patient remained unstable on arrival in the intensive care unit, requiring sedation and paralysis to manage hypoxia resulting from pulmonary edema. The patient was weaned off phenylephrine and started on epinephrine and dobutamine infusions. She was also started on aspirin and heparin for akinetic left ventricular segments.

On postanesthesia day 2, EKG continued to show normal sinus rhythm, troponin I level trended down, and all other laboratory values remained within normal range. On day 3, the patient was weaned off the inotropes and started on a beta-blocker (carvedilol). The inspired oxygen

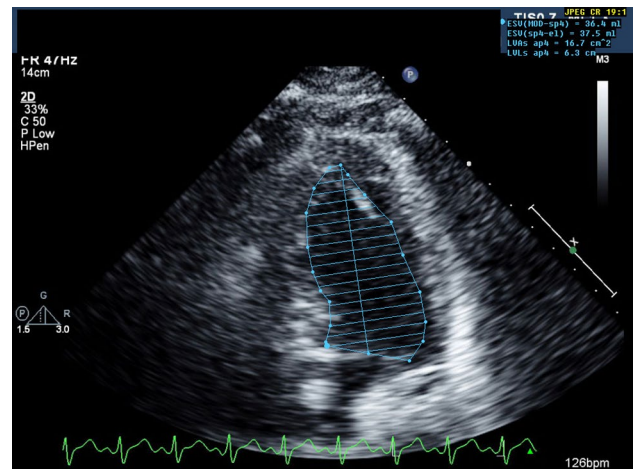


Fig. 1 Systole (immediate postoperative day 1). Akinetic mid and basilar left ventricle segments with dynamic apex. Calculated ejection fraction of 23 %

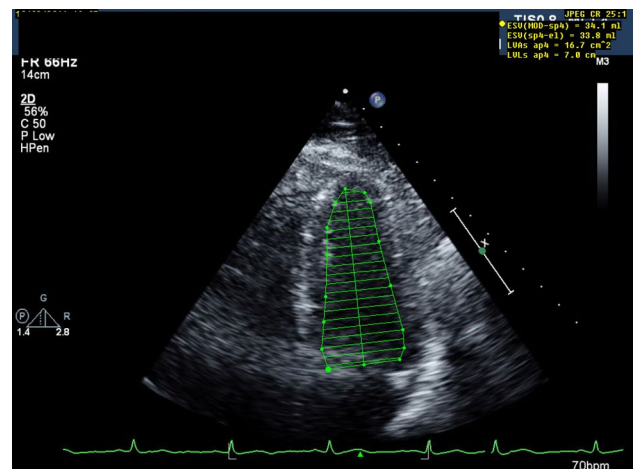


Fig. 2 Systole 4 chambers (postoperative day 4). Recovering myocardium. Calculated left ventricle ejection fraction of 55 %

requirement decreased from 100–40 %, and the patient was extubated. Postextubation, the patient's oxygen saturation dropped to mid-80 %, and noninvasive ventilation with bi-level positive airway pressure (BiPAP) was initiated. She was started on an angiotensin-converting enzyme (ACE) inhibitor and a diuretic. On day 4, the patient was weaned from BiPAP to 2 L oxygen via nasal cannula, and heparin was replaced with subcutaneous enoxaparin. On day 5, the patient was ambulated, the indwelling Foley catheter was removed, and the patient was started on a normal diet. A repeat echocardiogram on postoperative day 4 showed recovering reverse TCM with calculated left ventricle ejection fraction of 55 % (Fig. 2). On postoperative day 9, the patient was discharged home on aspirin, β -blocker, and

ACE inhibitor, with instructions to follow up with the cardiology team.

Discussion

The specific etiology of TCM is unknown, but hypotheses include elevated plasma catecholamine levels resulting from intense emotional or physical stress, leading to multivessel coronary artery vasospasm, microvascular dysfunction, impaired fatty acid metabolism within the myocardium, and myocardial stunning leading to transient hypokinesia of the left ventricular segments in all types of TCM [1]. Patients with neuropsychiatric disorders have less inhibition of corticotropin-releasing hormone/hypothalamic–pituitary–adrenal (CRH/HPA) axis with higher levels of stress hormones [3]. Even unconscious, a total peripheral blockade of the nociceptive system in a patient under general anesthesia may be impossible [4]. The stress of anesthesia can further stimulate the CRH/HPA axis and can trigger TCM in susceptible patients [5]. Our patient had a previous history of subdural hematoma and subarachnoid hemorrhage and was receiving treatment for anxiety and depression. We believe that the patient's newly developed neuropsychiatric disorder in conjunction with physical stress from anesthesia and laryngoscopy triggered reverse TCM in this individual [2].

In the International Takotsubo Registry (ITR), which includes 1750 patients, TCM is divided into four types, occurring in 82, 2.2, 14.6, and 1.5 % of patients, respectively [2]:

1. Apical or classic
2. Basal or reverse
3. Midventricular
4. Focal

Demographically, patients with reverse TCM present at an earlier age due to abundance of adrenoceptors at the base [6] compared with those with classic TCM, which affects postmenopausal older women with an abundance of adrenoceptors at the apex [7]. In reverse TCM, more myocardial tissue is involved, with greater elevation in cardiac biomarkers, but there are fewer incidences of reversible mitral regurgitation (MR) than the classic TCM type [8]. Furthermore, patients with reverse TCM have higher prevalence of triggering stress, either physical or emotional, than do patients with the classic type [6]. Our patient developed a neuropsychiatric disorder following her previous surgery and developed reverse TCM on physical stress she experienced during anesthesia and laryngoscopy.

Before induction of anesthesia, we used midazolam as anxiolytic and, subsequently, administered etomidate for induction to maintain hemodynamic stability. There is no evidence that etomidate triggers TCM [9], although

etomidate has been associated with a substantially increased risk for 30-day mortality and cardiovascular morbidity compared with propofol [10]. The bradycardia at induction of anesthesia was treated with glycopyrrolate, an anticholinergic with side effects of tachycardia and possible cardiac arrhythmias but that provides better hemodynamic stability than atropine [11]. Frequently, the stress response to laryngoscopy is blunted by opioids; however, opioids such as fentanyl can cause severe bradycardia or even asystole, especially in conjunction with the vagal stimulating effects of laryngoscopy [12]. We did not administer opioids to the patient, as she was already bradycardic on induction. In retrospect, induction with opioids and a titrating dose of propofol may have been a better option.

We administered suxamethonium for an anticipated short surgical procedure. Suxamethonium is associated with cardiac complications in certain medical conditions, such as spinal cord injury, peripheral nerve injury, and acute muscle wasting—none of which was present in our patient. We treated the postlaryngoscopy tachycardia and hypertension with titrating dosages of esmolol, as β blockers have been suggested in the treatment of TCM [9, 13]. To prevent the development of TCM among patients with neuropsychiatric disorders and for patients with a history of TCM presenting for surgery, β -blockers and ACE inhibitors initiated preoperatively are recommended to be continued in the perioperative period [13, 14]. Anxiety should be treated by psychological and pharmacological means such as benzodiazepines or an α_2 -adrenergic agonist (clonidine).

After an initial hypertensive period on induction, our patient developed hypotension. Inotropes can contribute to TCM development, but intractable hypotension should be treated with inotropes and vasopressors [9, 14]. Our patient developed left ventricular failure (LVF), and LVF should be treated with diuretics, ACE inhibitors, aspirin, and heparin [13–15]. In patients with poor response to inotropes, the options of intra-aortic balloon pump and left ventricular assist devices can be considered [9, 16]. In patients with documented coronary vasospasm, treatment with a nondihydropyridine calcium channel blocker, such as diltiazem or verapamil, should be considered [14, 17].

Perioperative monitoring for patients with a history of TCM should include continuous EKG monitoring. Intra-arterial catheterization should be used for blood pressure monitoring, and transesophageal echocardiography might be useful for continuous assessment of left ventricular function. In the event of significant left ventricular failure, pulmonary artery catheterization may be needed to optimize cardiac output.

In summary, we describe a patient who developed reverse TCM. While TCM is unpredictable, identifying patients who will develop TCM after posttraumatic neuropsychiatric disorder is even more challenging. The

management of these critically ill patients requires a team approach involving anesthesiologists, intensivists, and cardiologists.

Compliance with ethical standards

Conflict of interest The authors declare that they have no competing interests.

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