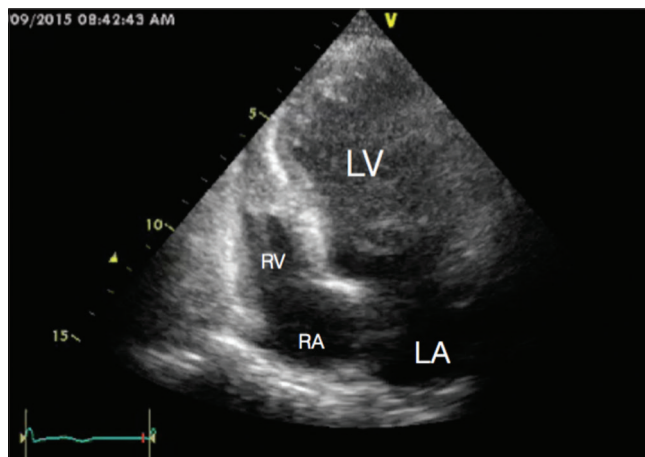


## Anesthetic management of a patient with Takotsubo cardiomyopathy presenting for surgical clipping of intracranial aneurysm

Takotsubo cardiomyopathy (TC) or stress-induced cardiomyopathy is a rare but devastating condition, with high mortality and morbidity which affects the patient outcome. Initially described in patient with subarachnoid hemorrhage (SAH) and trauma, the disorder has been recognized to occur in a variety of other conditions.<sup>[1]</sup> The hallmark of the condition is the electrocardiogram changes of myocardial ischemia, severe left ventricular dysfunction with normal coronary angiogram. Increased incidence has been reported in the perioperative period, especially after anesthetic induction. Literature search showed little about the anesthetic management of patients with documented TC.

A 47-year-old female was referred to our hospital with documented SAH (World Federation of Neurosurgical Surgeons Grade 2, Fisher Grade 3). Computed tomography angiogram showed anterior communicating artery (ACoM) aneurysm. She was a hypertensive and diabetic on irregular treatment. She had no prior history of cardiac disease. The preoperative transthoracic echocardiogram (TTE) showed an ejection fraction (EF) of 38% with concentric left ventricular hypertrophy, dilated mid and apical portions [Figure 1] with Grade 1 diastolic dysfunction, and extensive regional wall motion abnormality (RWMA) of the apical and mid left ventricle (LV) with sparing of the basal region of LV. Troponin T was positive (> 100 ng/ml), B-type natriuretic peptide 590 pg/ml preoperatively. The echo was suggestive of TC. Since the patient was in poor neurological condition, urgent clipping of the aneurysm was planned.

No premedication was given. In the operating room, under standard monitoring lines including invasive blood pressure, the anaesthetic induction and intubation were planned under echocardiographic guidance, initially transthoracic and later transesophageal (TEE) route after intubation. Anesthetic induction consisted of titrated dose of fentanyl (150 mics), Propofol in 10mg aliquots, a total of 70 mg which showed loss of verbal response. Vecuronium was administered to facilitate endotracheal intubation. Within 2 min postintubation, EF dropped to 15%, increasing RWMA, fall in BP and a bolus of phenylephrine was given to increase the blood pressure. Within



**Figure 1:** Dilated apical and mid-portion of left ventricle in the 4-chamber view in transthoracic echocardiography (LV = Left ventricle, LA = Left atrium, RV = Right ventricle, RA = Right atrium)

minutes, the EF returned to 38%. Anesthesia was maintained with O<sub>2</sub>/air with sevoflurane inhalation 0.8–1 minimum alveolar concentration. TEE echo probe was introduced after intubation, and the cardiac status and volume and hemodynamic status were managed under TEE guidance. Surgical clipping was uneventful. During the closure, there were episodes of hypotension, and noradrenaline infusion at 0.05 mics/kg/min was started. At the end of the surgery, the patient was responding to commands and trachea was extubated. On the 2<sup>nd</sup> postoperative day, patient developed weakness of left upper and lower limb. Transcranial Doppler showed features of vasospasm (middle cerebral artery peak systolic flow velocity > 185 cm/s). The noradrenaline dose was increased to 0.15 mics/kg/min to increase the systolic pressure from 120 to 160 mmHg with TTE guidance. In few hours, patient's neurological condition improved. Rest of the postoperative period was uneventful. Repeated TTE showed marginal improvement in the RWMA and EF to 42% at 14 days postsurgery.

Our patient satisfies the criteria suggested by the Mayo clinic cardiology for diagnosis of TC.<sup>[2]</sup> Limited literature exists in the anesthetic management in TC. In a small series of 8 patients with documented TC who underwent aneurysmal clipping or coiling, two died and five had pulmonary edema, only one was uneventful.<sup>[3]</sup> However, no details of anesthetic management were described. We had also published a case of ACoM aneurysm clipping with documented TC who succumbed in the postoperative period due to vasospasm and malignant cerebral infarct.<sup>[4]</sup> We came across a case report of intraoperative occurrence of TC during the end of spine surgery which was managed with beta-blockers.<sup>[5]</sup> Reports show that surgeries were canceled in patients where TC occurred, following induction of anesthesia.<sup>[6]</sup> It is important to understand the

pathophysiology in the management of these patients. Since TC is caused by stress, it may be tempting to keep the patient under deeper plane of anesthesia. However, such a procedure would be potentially harmful due to limited cardiac reserve, poor compensation for hemodynamic fluctuations, as well as suppressant action of anesthetic drugs. TC is thought to be caused by high level of circulating catecholamines. It is obvious that management of hemodynamic instability by the use of inotropes in a patient having already high level of catecholamines may worsen the existing myocardial damage. Hence, the anesthetic goal would be to protect the unaffected limited myocardium while maintaining the hemodynamic stability. Hence, vasopressors would help for maintaining blood pressure while maintaining the myocardial function. The goals of the anesthetic management would be maintenance of sinus rhythm, avoidance of tachycardia, or excessive bradycardia. Preload must be optimized individually as excessive preload in a poorly compliant LV can lead to pulmonary edema. Unlike the patient with coronary artery disease where diastolic augmentation of blood pressure improves the coronary blood flow, in patients with TC, such effects cannot improve the myocardial performance as they have normal coronaries. After load augmentation to improve, the coronary flow can cause reduction in cardiac output. Early use of intra-aortic balloon pump is a very useful option in managing acute cases of TC.

The options for management depend on the acuteness of the TC and the surgery. Majority of cases of TC resolves in days to weeks. If the TC develops following anesthetic induction before the start of surgery which is not urgent, then the choice would stabilize the patients in the intensive care unit. Recently, levosimendan has been found to have a favorable effect on the initial management of these patients.<sup>[7]</sup> If the TC occurs during the middle of the surgery and if not life-threatening, the goals would be to maintain the hemodynamics with control of stress response using opioids and beta blockers. Centrally acting alpha 2 agonist-like dexmedetomidine use also has been suggested due to its favorable hemodynamic actions, but no report exists on the benefits of its use. In case of documented case of TC (acute or resolving stage) presenting for urgent/emergency surgery, the procedure should be done in places where intraoperative echocardiographic monitoring is available. Echocardiography helps in guiding the optimal management.

Our case report suggests that use of echo in monitoring systolic, diastolic LV function and preload in the intraoperative period from baseline, induction, intubation, and maintenance in the postoperative period aids in the successful management of a patient with TC.

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### Conflicts of interest

There are no conflicts of interest.

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