

Case Report

A case of stress induced cardiomyopathy with extracorporeal membrane oxygenation after total colectomy

Sanjay Madhukar Surwade^{1*}, Tapas Mandal², Hemant Mehta², Shaunak Mangeshkar³

¹Department of Anaesthesia, National University Hospital, Kent Ridge, Singapore

²Department of Anaesthesia, Sir HN Reliance Foundation Hospital, Raja Rammohan Roy Road, Prarthana Samaj, Girgaon, Mumbai, Maharashtra, India

³MBBS Student, King Edward Memorial Medical College, Mumbai, Maharashtra, India

Received: 29 July 2019

Revised: 09 September 2019

Accepted: 13 September 2019

*Correspondence:

Dr. Sanjay Madhukar Surwade,

Email: sanjay.surwade17111983@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Takotsubo cardiomyopathy or stress-induced cardiomyopathy is a cardiac syndrome of a reversible, transient left ventricular dysfunction that is caused by emotional and/or physical stress and surgery. Its clinical manifestations are similar to acute myocardial ischemia in absence of coronary artery lesion. It is more common in middle-aged women, and the prognosis is favourable. We report the case of a 32-year-old male patient who underwent a total colectomy, developed stress-induced cardiomyopathy and had poor outcome despite extracorporeal membrane oxygenation (ECMO).

Keywords: Cardiomyopathy, Extracorporeal membrane oxygenation, General anesthesia, Stress

INTRODUCTION

Stress-induced cardiomyopathy is a deterioration of the myocardial function caused by stress such as emotional or physical that results in momentary, reversible ventricular dysfunction.^{1,2} It was first reported by Dote et al. 1991 is also known as 'takotsubo cardiomyopathy' or 'apical ballooning syndrome,' because its electrocardiographic (ECG) changes and clinical manifestations are similar to myocardial infarction but coronary arteries shows no lesion.^{3,4}

The exact pathophysiology of stress-induced Cardiomyopathy still remains elusive. There are studies reporting that stress-induced cardiomyopathy can occur during or after surgery with full recovery.^{5,6} Rarely ECMO is needed for recovery of these patients.^{7,8} But there are to the best of our knowledge there are no reports of this condition

causing death despite ECMO support. Authors report a case of stress-induced cardiomyopathy that developed after a total colectomy who suffered unfavorable outcome even with the use of ECMO.

CASE REPORT

A 32-year-old, 55 kg, 154 cm height male, known case of ulcerative colitis, Crohn's disease and low grade adenocarcinoma of colon presented to our hospital with loose stools, nausea, pain in abdomen for 15 days. Preoperative investigations revealed hemoglobin (Hb) of 6.8 g/dl white blood cell (WBC) count of 2450 per microliter, and albumin of 2.2 g/dL. The preoperative chest x-ray, electrocardiogram (ECG), echocardiography and renal function tests were normal.

Patient was optimized using three packed red cells, albumin and G-CSF over 3 days following which Hb was

9.8, WBC count 12270 per microliter and albumin of 3.3 g/dL.

Patient was posted for laparoscopic total colectomy after three days. Patient's blood pressure (BP) and heart rate (HR) after arriving in the operating room was 106/60 mmHg and 81 beats/min. Anesthesia was induced with fentanyl 100 micrograms and propofol 120 mg intravenously. Muscle relaxation was achieved with atracurium 50 mg after confirming the loss of consciousness, and endotracheal intubation was performed with 8 number cuffed endotracheal tube and position confirmed. Anesthesia was maintained with O₂ 0.5 L/min, air 0.5 L/min, desflurane maintained at 1-1.5 MAC. Central venous catheter was placed under ultrasonography guidance in right internal jugular vein. As patient did not have major co-morbidities invasive blood pressure monitoring was not considered. Mechanical ventilation was maintained with a tidal volume, respiratory rate and end-tidal carbon dioxide of 450 ml, 12/minute and 30-35 mmHg, respectively. Antacid prophylaxis in the form of pantoprazole 40 mg and appropriate antibiotic was administered within 30 minutes of surgical incision. Abdominal insufflation done and procedure started. Due to complexity of surgery, after about 8 hours, laparoscopic procedure was converted to open and completed. Intraoperative problems faced were hypotension and hypothermia. Hypotension began immediately after the induction of anaesthesia and was persistent throughout the surgery. Intravenous fluid boluses were given to build up central venous pressure and intermittent phenylephrine injections were used as a measure to maintain blood pressure. Despite this, blood pressure was still on the lower side, so left radial artery was cannulated and noradrenaline infusion started and was titrated to sustain blood pressure within normal limits. Continuous temperature monitoring was done using nasopharyngeal temperature probe. Temperature noted immediately after induction of anaesthesia was 35.5°C which showed decreasing trend despite use of in line intravenous fluid warmer and forced warming device throughout intraoperative period. It reached its nadir of 31.5°C after about 7 hours and 15 minutes of surgery. Patient's heart rate and oxygen saturation during surgery were maintained at 60-70 beats/min and 100%. The total surgical duration was about 12 hrs. The total amount of fluids administered was 3,000 ml of Hartman's solution, 1500 ml plasmalyte, 1000 ml of normal saline, 500 ml gelofuscin and 499 ml packed cells while the urine output and blood loss were 950 ml and 1000 ml, respectively. Patient was not reversed and not extubated in view of prolonged duration of surgery, persistent hypotension, high inotropic support and hypothermia. The patient was transported to the intensive care unit, with blood pressure, heart rate, et CO₂ and temperature of 100/64 mmHg, 117/minute, 27 mmHg and 32.7°C respectively, where he was kept on Pressure-regulated volume control mode of mechanical ventilation. Injection noradrenaline was running at the infusion rate of 0.2 mcg/kg/min. In the ICU, to keep patient sedated a mixture of injection

midazolam 25 mg and injection fentanyl 200 mcg started at 5 ml/hr. Arterial blood gas was done which showed pH of 7.17, PCO₂- 39, HCO₃⁻ 13.9, lactates-140.6 mmol/L. Sodium bicarbonate correction of 100 cc was given. Noradrenaline was stepped up at the infusion rate of 0.4 mcg/kg/min to maintain the blood pressure of 100/77 mmHg. Simultaneously, vasopressin infusion was also started at 2.4 units/hr. After 4.30 hours post -surgery, reason to find out the cause of persistent hypotension despite high inotropic support, a cardiology reference was raised. A 12 lead electrocardiogram was performed, which was suggestive of q waves in anterior leads. Subsequently, a screening echocardiography was performed by a cardiologist, and it revealed inferior vena cava fullness, ejection fraction of 15-20%. Blood investigations revealed troponin-I levels of 460.7 pg/ml. A provisional diagnosis of Takatsubo's cardiomyopathy was made. Infusion of levosimendan 0.05 mic/kg/min was also added as per cardiologist's advice. On subsequent day troponin-I level was 10545.9 pg/ml.

Twelve hours post-surgery patient developed marked tachycardia of 172/min, and blood pressure of 71/50 mmHg on noradrenaline, vasopressin and levosimendan infusions. Patient also developed edema over body, with fever of 100.1°C All subsequent arterial blood gases revealed persistent metabolic acidosis in the range of 7.19-7.26, very high accumulation of lactates to the extent of 230 mmol/L. Patient also had frequent episode of hypocalcemia. It was treated with 50% glucose administration and frequent glucose monitoring. During next 4 hours patient became anuric. Due to rapidly worsening condition of the patient, veno-arterial extracorporeal membrane oxygenation device (ECMO) was put by a cardiac surgeon. This procedure involved 750 ml blood loss, which required transfusion of 9 units packed cells, 4 units random donor platelets, 5 units fresh frozen plasma and 5 units of cryoprecipitate. In addition, normal saline of 2000 ml and 1500 ml volulyte was also given. ECMO was initially started at 1.3 litres/min, which had to be stepped up to 2.2 litres/min due to persistent hypotension. Patient continued to be anuric and was finally put on sustained low efficiency dialysis (SLED).

On dialysis, patient still required very high inotropic support of noradrenaline and vasopressin. All efforts to build up blood pressure and urine output failed. On post-operative day 2, patient developed one episode of ventricular tachycardia, which had to be defibrillated with 200 joules of shock. Repeat 2D echocardiography revealed failing right ventricle, pulmonary edema and ejection fraction of 10%. Injection frusemide was given to the patient. On the following day, patient developed hepatic enzymes derangement, acute respiratory distress syndrome, disseminated intravascular coagulopathy (deranged coagulation parameters with thrombocytopenia and low fibrinogen). SLED was abandoned and continuous veno-venous hemofiltration was started. One packed cell, 6 units cryoprecipitate and 1 single donor platelet were transfused to the patient to reverse

coagulopathy. Subsequent day, patient developed cardiac arrest, cardiopulmonary resuscitation was performed as per AHA guidelines, but patient could not be revived.

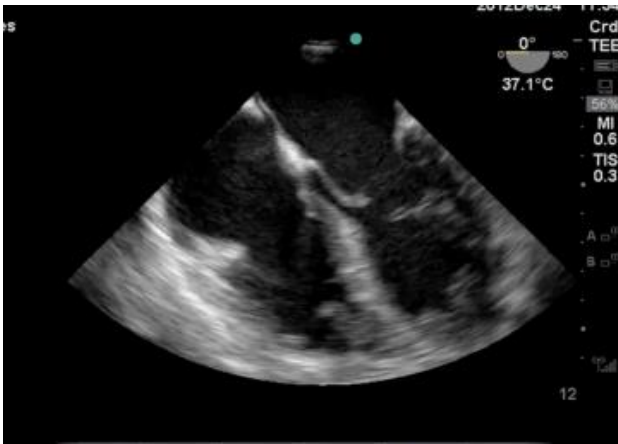


Figure 1: Normal preoperative 4-chamber view transesophageal echocardiography (TEE).

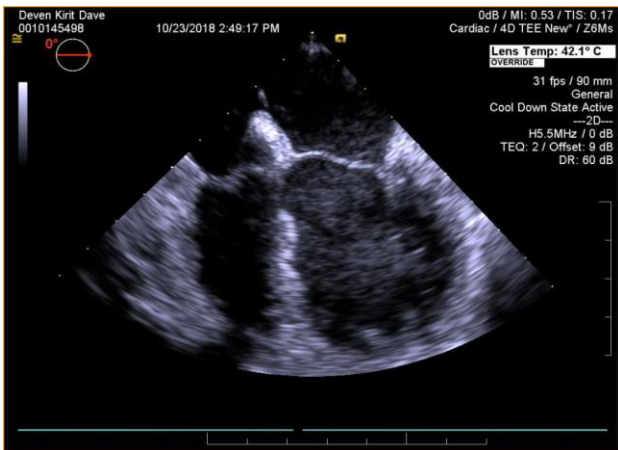


Figure 2: Postoperative TEE 4-chamber view shows severe left ventricular dysfunction with apical ballooning.

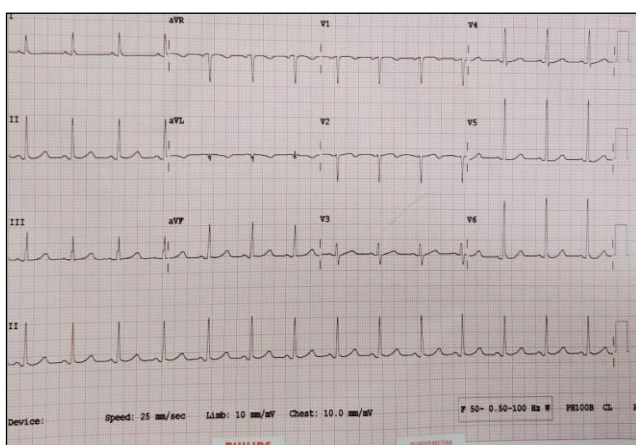


Figure 3: Normal preoperative electrocardiogram (ECG).

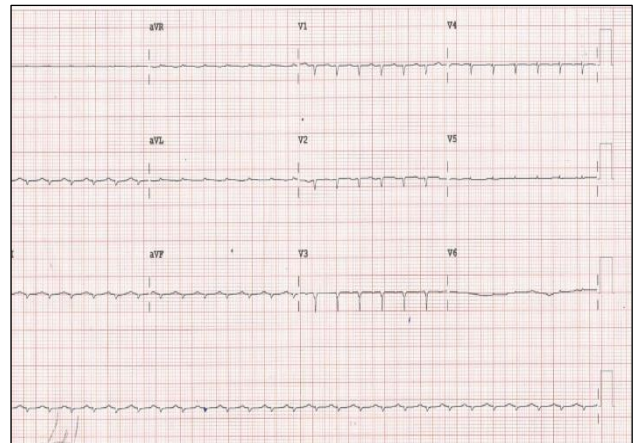


Figure 4: ECG showing low voltage QRS complexes, no ST segment changes despite severe left ventricular dysfunction.

DISCUSSION

Stress-induced cardiomyopathy is a likely complication in post-surgery patients.^{5,6,9} It is more commonly seen in post-menopausal patients.¹⁰ The precise etiology and pathophysiology is unclear. However, the most likely mechanisms are sympathetic hyperactivity related to stress and myocardial stunning due to an increase in the catecholamine level.^{4,6,10} In International Takotsubo Registry, physical and emotional stress preceded in about 64% of patients.¹⁰ The main clinical symptoms of this syndrome are chest pain or dyspnea. In the present case, the mechanism of stress-induced cardiomyopathy appears to be prolonged surgical stress.

Lyon AR et al, has recently defined Takotsubo cardiomyopathy as follows:¹¹

- Transient regional wall motion abnormalities of LV or RV myocardium occur and are frequently, but not always, preceded by a stressful trigger (emotional or physical).
- The regional wall motion abnormalities usually extend beyond a single epicardial vascular distribution and often result in circumferential dysfunction of the ventricular segments involved.
- There is an absence of culprit atherosclerotic coronary artery disease, including acute plaque rupture, thrombus formation, and coronary dissection or other pathologic conditions, to explain the pattern of temporary LV dysfunction observed (e.g., hypertrophic cardiomyopathy, viral myocarditis).
- New and reversible electrocardiography (ECG) abnormalities (ST segment elevation, ST depression, LBBB, T-wave inversion, and/or QTc prolongation) are seen during the acute phase (first 3 months).
- Significantly elevated levels of serum natriuretic peptide (BNP or NT-proBNP) are seen during the acute phase.

- A positive but relatively small elevation in cardiac troponin can be measured with a conventional assay (i.e., disparity between the troponin level and the amount of dysfunctional myocardium present).
- Recovery of ventricular systolic function is apparent on cardiac imaging at follow-up (3 to 6 months).

In this case, the patient presented with persistent hypotension during the surgery and it continued in intensive care unit which signify possible beginning of the myocardial stunning from intraoperative phase continuing perioperatively. In addition, in present case ECG showed pathological Q waves, seen in 40% of the patients, rise in cardiac enzymes, severe hypokinesia, ballooning of left ventricle with ejection fraction of 15% all pointed towards the stress induced cardiomyopathy.¹² Due to unstable condition of the patient, coronary angiography could not be performed. It would have further supported the diagnosis.

To this knowledge, there were no case reports where patient succumbed to death due to stress induced cardiomyopathy. In previous case reports ECMO has proved to be lifesaving in severe cases.¹³ In given case report, diagnosis of cardiomyopathy was within 10 hours and ECMO was initiated within 24 hours post-operative period. Despite early interventions spontaneous recovery did not occur. Although the cause of death was septic shock, we believe that cardiogenic shock secondary to cardiomyopathy was primary insult which lead to persistent hypotension and subsequent renal, hepatic failure coagulopathy and septic shock.

This syndrome may complicate in the form of congestive heart failure and pulmonary edema in 3-46% of patients with mortality about 1-8%.¹³ Therefore, it is essential to consider early diagnosis and more aggressive therapy in case of severe reduction of left ventricular function. There is need of guidelines for the management of severe stress induced cardiomyopathy. The prognosis of stress-induced cardiomyopathy is considered favorable but present case also showed unfavorable outcome even if aggressive treatment was employed. Myocardial dysfunction on echocardiography may take several days or weeks to recover.⁵ The presented patient had no known risk factors leading to coronary artery disease or reversible cardiomyopathy. Moreover, unlike most cases reported to date, this patient required cardiac resuscitation and ECMO to maintain life, but it did not help because of several complicating factors.

CONCLUSION

Prolonged surgery with concomitant issues such as hypotension, hypothermia, and acidosis should be considered a potential cause of stress induced cardiomyopathy even for the patients with no cardiac risk factors. One must be cautious to consciously reduce the incidence of stress induced cardiomyopathy. Appropriate and timely diagnosis and early institution of ECMO in

selected patient might help to reduce the incidence of mortality in stress-induced cardiomyopathy.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

REFERENCES

1. Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. *Am Heart J.* 2008 Mar 1;155(3):408-17.
2. Gianni M, Dentali F, Grandi AM, Sumner G, Hiralal R, Lonn E. Apical ballooning syndrome or takotsubo cardiomyopathy: a systematic review. *Eur Heart J.* 2006 May 23;27(13):1523-9.
3. Dote K, Sato H, Tateishi H, Uchida T, Ishihara M. Myocardial stunning due to simultaneous multivessel coronary spasms: a review of 5 cases. *J Cardiol.* 1991;21(2):203-14.
4. Pilgrim TM, Wyss TR. Takotsubo cardiomyopathy or transient left ventricular apical ballooning syndrome: a systematic review. *Int J Cardiol.* 2008 Mar 14;124(3):283-92.
5. Littlejohn FC, Syed O, Ornstein E, Connolly ES, Heyer EJ. Takotsubo cardiomyopathy associated with anesthesia: three case reports. *Cases J.* 2008 Dec;1(1):227.
6. Lentschener C, Vignaux O, Spaulding C, Bonnichon P, Legmann P, Ozier Y. Early postoperative takotsubo-like left ventricular dysfunction: transient left ventricular apical ballooning syndrome. *Anesthesia Analgesia.* 2006 Sep 1;103(3):580-2.
7. Jo YY, Park S, Choi YS. Extracorporeal membrane oxygenation in a patient with stress-induced cardiomyopathy after caesarean section. *Anaesth Intensive Care.* 2011 Sep;39(5):954-7.
8. Pearson TE, Frizzola MA, Priest MA, Rochman MF, Froehlich CD. Pediatric Extracorporeal Cardiopulmonary Resuscitation Patient With Traumatic Subarachnoid Hemorrhage and Takotsubo Syndrome. *Air Med J.* 2018;37(1):64-6.
9. Bhojraj S, Sheth S, Pahlajani D. Postoperative Takotsubo cardiomyopathy. *Ann Card Anaesth.* 2014 Apr-Jun;17(2):157-60.
10. Dorfman TA, Iskandrian AE. Takotsubo cardiomyopathy: state of the art review. *J Nucl Cardiol.* 2009;16(1):122-34.
11. Lyon AR, Bossone E, Schneider B, Sechtem U, Citro R, Underwood SR, et al. Current state of knowledge on Takotsubo syndrome: a Position Statement from the Taskforce on Takotsubo Syndrome of the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail.* 2016 Jan;18(1):8-27.
12. Bybee KA, Kara T, Prasad A, Lerman A, Barsness GW, Wright RS, et al. Systematic review: transient left ventricular apical ballooning: a syndrome that

mimics ST-segment elevation myocardial infarction. *Ann Internal Med.* 2004 Dec 7;141(11):858-65.

13. Aggarwal N, Rai AK, Yizhak K, Saunders P, Tessler S. ECMO for cardiopulmonary rescue after near fatal Takotsubo cardiomyopathy. *Chest.* 2014 Oct 1;146(4):257A.

Cite this article as: Surwade SM, Mandal T, Mehta H, Mangeshkar S. A case of stress induced cardiomyopathy with extracorporeal membrane oxygenation after total colectomy. *Int J Res Med Sci* 2019;7:3959-63.