Recurrence of Takotsubo Cardiomyopathy following Rigid Bronchoscopy: A Case Report

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Abstract

We report a case of a young male patient with a history of Takotsubo cardiomyopathy following polytrauma. On presentation, the patient had a tracheal stent placement for tracheal injury. The patient was again posted for removal of tracheal stent, by rigid bronchoscopy. During the procedure patient had a recurrence of cardiomyopathy, requiring inotropes and post-operative ventilatory support. 2D echo showed apical ballooning of the heart. The patient improved by inotropes and later on Lasix 10 mg IV. He was extubated after 2 days.

Keywords: Takotsubo cardiomyopathy; Recurrence; Rigid Bronchoscopy; General anesthesia; Physiological Stress Response; Complications.

Introduction

Takotsubo cardiomyopathy (TTC) or stress cardiomyopathy was first described in Hiroshima City Hospital in Japan in the 1990s. It is described as systolic dysfunction that occurs in response to stress and is usually transient in nature. Here we describe a case with a previous history of TTC after polytrauma, who developed a recurrent episode of TTC during rigid bronchoscopy two months later.

Case report

A 22-year-old male with a history of polytrauma two months back, during which he developed Takotsubo cardiomyopathy, diagnosed on echocardiography. The patient also suffered damage to the tracheal cartilages in the distal part resulting in airway collapse, for which initially endotracheal intubation was done. As the cardiomyopathy resolved, a tracheal stent was placed in view of distal tracheal
collapse. Two months later the patient was posted for rigid bronchoscopy for tracheal stent removal. All his routine investigations, including chest x-ray and echo, were normal.

After preoxygenation with 100% oxygen, the patient was induced with injection (inj.) fentanyl 100 mcg iv, Propofol 100 mg iv, and Succinylcholine 100 mg iv. As the rigid bronchoscope was inserted, the patient had an episode of hypertension (BP- 200/130 mm of Hg) and tachyarrhythmias (multiple ventricular ectopic, ventricular bigeminy). It was managed with inj. propofol 40 mg iv. That was followed by hypotension (BP-74/50 mmHg). Inj. Mephenetermine 6 mg iv were given, and iv crystalloid (normal saline) was given as 200 – 250 ml bolus. The rigid bronchoscopy lasted for about 5-10 minutes. That was followed by another episode of hypertension, and tachycardia. Inj. Fentanyl 30 mcg iv was given. An arterial cannula was placed, and started the patient on noradrenaline infusion, titrated to maintain a mean arterial pressure of 60 mm of Hg. The patient started desaturating and was intubated. He was maintaining an oxygen saturation of 96% with fraction of oxygen (FiO2) of 100%. Pink frothy sputum was noticed in the endotracheal tube and bilateral crepitations were present on auscultation. Echo showed global hypokinesia of left ventricles with an ejection fraction of around 20-30%.

The patient was shifted to the ICU and placed on ventilatory support. In view of compromised cardiac function, fentanyl and vecuronium infusions were continued, and low tidal volume ventilation (6 ml/kg), with FiO2 of 1, positive end-expiratory pressure (PEEP) titrated from 5-10 to maintain a saturation of > 92%. In view of hypotension, Noradrenaline infusion was continued with doses titrated according to response. The cardiologist later re-evaluated the patient and a diagnosis of a recurrence of Takotsubo cardiomyopathy was made based on apical ballooning seen on echocardiography. By the next day, the patient improved inotropes were stopped, injection of Lasix 10 mg iv was started (one dose or infusion?). As the chest condition improved, the patient was extubated after 2 days. Follow-up echo cardiography done on the 4th day showed an ejection fraction of 50-55% with no regional wall motion abnormality. Lasix was stopped and the patient was subsequently discharged the next day.

**Discussion**

Takotsubo cardiomyopathy (TTC) is a temporary dysfunction of the left ventricle, without the evidence of any coronary artery disease (CAD) on coronary angiography.[1] Satoh et al [2] first described this condition and it was named ‘Takotsubo’ due to the resemblance of apical ballooning of the left ventricle to the Japanese round neck pot called tako-tsubo. European Society of cardiology proposed the diagnostic criteria for TTC. Most cases have been attributed to underlying emotional or physical triggers causing blood-borne catecholamine-induced toxicity of the myocardium.[1]

The clinical picture resembles that of acute coronary syndrome. Patients present with chest pain, and dyspnea which is usually sudden in onset. An electrocardiograph (ECG) may show ST-segment elevation or
depression or the presence of a Q wave.[3] That is also associated with the elevation of cardiac enzymes. However, on coronary angiography, there is no occlusion of blood vessels. The condition is usually reversible and spontaneous recovery occurs in most cases.

TTC is associated with significant morbidity and mortality. A study by Templin et al,[4] found that the incidence of major cardiovascular events was 9.9% and mortality was 5.6% per patient-year. A systematic review[5] concluded that male sex, comorbidities like chronic kidney disease, malignancy, high body mass index, physical triggers, sepsis, anemia, and chronic obstructive pulmonary disease were associated with poor prognosis in TTC patients.

Recurrence has been reported in patients after recovery from the first episode of TTC. The recurrent TTC can be defined as the recurrence of LV dysfunction in a patient who had recovered from initial TTC, without any evidence of coronary artery disease. TTC may reoccur at an estimated rate of about 4% with most recurrences occurring in the first five years of the first episode.[6] They further divided the patients into recurrent and non-recurrent groups and compared the baseline characteristics and outcomes. They found that patients in the recurrent group had a higher incidence of hypertension. Further, there were no significant differences in terms of complications or outcomes except that patients with recurrence had a higher incidence of pulmonary edema.

In our case, the rigid bronchoscopy might have triggered an exaggerated sympathetic response due to overactivation of the hypothalamic-pituitary-adrenal axis. That would have resulted in an excessive release of epinephrine and nor-epinephrine causing arrhythmia and hypertension. Such high levels of catecholamines would have further precipitated myocardial dysfunction resulting from microvascular damage and coronary artery spasm. That would have caused global hypokinesia of myocardium, hypotension, and pulmonary oedema. We took timely corrective actions, resulting in rapid and complete recovery of our patient.

A systematic review studied the recurrence of TTC and found that the recurrence rate varies from 1 to 3.5% per year with a global recurrence rate of 3.8%. They also concluded that female gender, lower body mass index (BMI), LV middle third hypercontractility, and shorter time after the first episode were related to recurrence.[7] Our patient likely had a TTC recurrence as he initially had a complete recovery from TTC, and it reoccurred after a catecholamine-releasing stimulus and had typical echocardiography findings.

**Conclusion**

In conclusion, though TTC is a reversible condition, a high degree of awareness is required regarding the risk of recurrence. So that while anaesthetizing a patient with a history of Takotsubo cardiomyopathy, the team must remain vigilant and avoid any stressor that may precipitate another episode as the same which can be life-threatening.

**References**


