*Case Report*

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# Stress Induced Postoperative Takotsubo Cardiomyopathy

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**Abstract**

The Takotsubo cardiomyopathy is a rare hemodynamic dysfunction, only recently reported . While the diagnostic criteria have been established and the outcome is known as favourable, the pathophysiological mechanisms are not entirely understood. Here we present the case of a patient scheduled for laparoscopic hysterectomy, who postoperatively developed a Takotsubo cardiomyopathy, supposedly triggered by an acute hypertensive crisis and emotional stress.

**Keywords:** Takotsubo Cardiomyopathy; Transient Left-Ventricular Apical Ballooning Syndrome; Stress-Induced Cardiomyopathy And Broken Heart Syndrome.

## Introduction

Takotsubo or "stress induced cardiomyopathy" is a rare disorder which usually manifests as myocardial infarction. It is characterized by reversible left ventricular dysfunction with apical akinesia.

## A Case Report

A peri-menopausal woman (45 year old), ASA I, a diagnosed case of dysfunctional uterine bleeding (DUB) was posted for laparoscopic hysterectomy under general anaesthesia. During her preanesthetic check up, patient had no history of any medical illness or surgical procedure in the past. Her all laboratory investigations were within normal limits.

Preoperatively patient was anxious and was much worried about the surgery. The counselling of patient was done to relieve her anxiety. Tab. Alprazolam 0.5 mg was given orally a night prior the surgery. At operation theatre, after the checking of consent & case paper, Multipara monitor cables were connected to the patient. IV access was achieved. General anaesthesia was administered with IV Midazolam 1.5 mg, IV Fentanyl 80 mcg, IV Propofol 2 mg/ kg and IV Vecuronium 6 mg. The endotracheal intubation was done without any difficulty. General anaesthesia was maintained with N2O: Oxygen in 60: 40% & Sevoflurane 1.5-2% and intermittent doses of Inj. Vecuronium and Fentanyl. The positive pressure ventilation was done via anaesthesia ventilator and $etCO_2$ was maintained between 32-36 mm/ Hg. The pneumo-peritoneum insufflation pressure was constantly maintained at 12-14 mm/ Hg. Intra-operatively patient's vitals were stable except that there was a transient increase of Blood pressure i.e. 170/100 mm/ Hg (Highest reading during intraoperative monitoring). It was managed with Inj. Nitro-glycerine (NTG) drip for a period of 20 minutes.

On completion of surgery, the reversal of anaesthesia was done with Inj. Neostigmine & Inj. Glycopyrrolate. After reversal of anaesthesia, the skeletal motor power was not adequate. Hence patient was shifted with endotracheal tube in situ to post anaesthesia care unit (PACU) and respiration was supported with CPAP. After two hours of reversal of anaesthesia, patient regained adequate skeletal motor tone and she was extubated.

After one hour of tracheal extubation, patient suddenly complained of chest pain and pink frothy secretions were coming out of mouth. Patient became suddenly dyspnoeic. Within few minutes, the patient developed pulse less ventricular tachycardia. Cardio-version was done with 200 J, she was reintubated and mechanical ventilation was initiated. Inotropic support was started with Inj. Dobutamine and

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Inj. Noradrenaline in titrated doses. ECG showed ST elevation. CPK-MB and Troponin-T levels were found to be minimally raised. X ray chest showed bilateral pulmonary oedema.

After 24 hours, 2D-Echo was done which showed left ventricular ejection fraction of 20 % with left ventricular apical hypokinesia. Repeat 2D echo on 3rd day showed mild improvement in ejection fraction of up to 30 %. Coronary angiography was done on 3rd day which did not reveal any coronary blockage (Proximal/ distal). Mechanical ventilation and Inotropic support was continued for the next 2 days.

Patient was extubated on 5th post-operative day as her vital parameters and laboratory parameters were satisfactory. Unfortunately after 6 hours of extubation, patient developed hypotension and cardiac arrest.

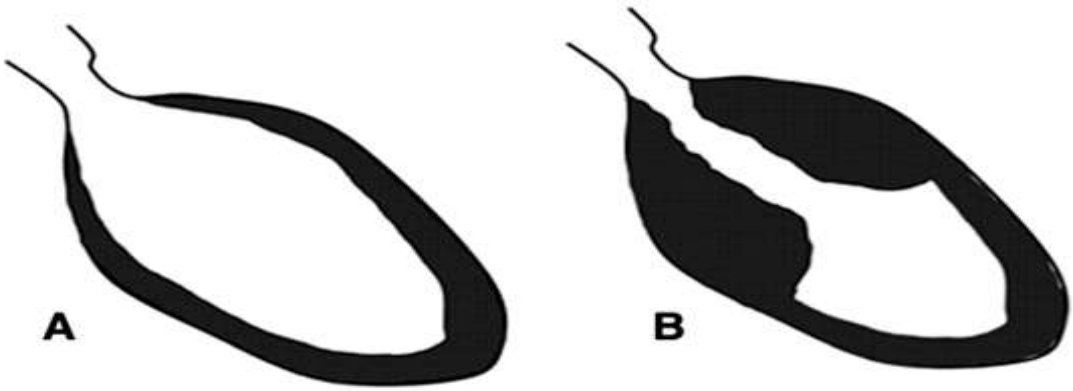


**Fig. 1:** Normal heart (A) and Tako-Tsubo sign (B) with apical ballooning (end-systolic view).

Inspite of all efforts of resuscitation, patient could not be revived and succumbed to death.

**Discussion**

Takotsubo cardiomyopathy (TCM) or "broken heart syndrome" is a clinical entity characterised by transient left ventricular dysfunction and apical dyskinesia with minimal increases in cardiac enzymes in the absence of coronary blockage/ occlusion.

Dote et al, first described this syndrome in Japanese patients; the name relates to the peculiar shape of left ventricle, which resembles an octopus-fishing pot called Takotsubo and can be visualised by end-systolic ventriculography [1].

Although the world wide incidence is unknown, TCM accounts for approximately 1% of admission for suspected for acute myocardial infarction in Japan; most cases are reported in post menopausal woman aged 60 -75 years [2-7]. Psychological, emotional (fear, grief, anger) & physical stresses (surgery, asthma, chemotherapy, stroke) are known triggers for onset of TCM. Initial signs and symptoms are similar as that of with acute coronary syndrome i.e. chest pain, dyspnoea and ST segment changes on ECG. The most common ECG finding in Takotsubo cardiomyopathy is ST segment elevation (as seen in our patient ), but ECG may be normal or may show T or Q wave abnormalities, diffuse often deep T wave inversion in the right pre-cordial leads mimicking STEMI can occur. In classic MI, size of infarct corresponds to the amount of myocardium supplied by obstructed artery. In Takotsubo, area of affected myocardium is much larger than normal distribution of a single coronary artery [8].

*Pathogenesis*

Although the exact aetiology of Takotsubo cardiomyopathy is remain unclear. Various mechanisms proposed is: 1) Multi vessel coronary artery spasm 2) Impaired cardiac micro vascular function and 3) Endogenous catecholamine induced myocardial stunning and micro-infarction. In TCM, catecholamine level reaching 7-34 times as high as published normal values and 2-3 times as high as level in patient with MI have been reported. Possibility of genetic predisposition may also exist [9].

*Clinical Features of Takotsubo Cardiomyopathy:*

*1)* Presentation of acute chest pain, or dyspnoea after emotional and physiological stress, 2) ECG abnormalities that mimic an acute MI, 3) Transient akinesia or hypokinesia of left ventricular apex and mid ventricle with basal hyperkinesias, 3) Absence of obstructive coronary lesion on coronary angiography, 4) Absence of other catecholamine-surge state, including Pheochromocytoma, recent head trauma and intra cranial bleeding. Recovery of

left ventricular function may occur within 2 - 4 wks of presentation.

*Treatment*

It is mainly supportive with diuretics, beta blockers, ACE inhibitors and if necessary, mechanical ventilation and intra-aortic balloon pump. Prophylactically warfarin may be used to prevent LV apical thrombus, which may be formed due to stasis of blood in akinetic segment. Although most patient of TC recover without any complication, 15-45% may develop life threatening complications like Cardiogenic shock, ventricular fibrillation, cardiac arrest and even death (RARE) .

In the present case anxiety and acute hypertensive crisis might have triggered TCM. We could not rule-out the possibility that $CO_2$ insufflation (Carbo-peritoneum) might have been the trigger factor of TC. Anderson et al. [10] found that increased catecholamine blood levels five minutes after peritoneal insufflations with CO2 may trigger Takotsubo Cardiomyopathy. This is less likely, considering that our patient's et $CO_2$ was low (30-31 mmHg) throughout the whole procedure [10]. Although this patient showed mild improvement in ejection fraction within 72 hours but she could not recover completely and succumbed to death due to cardiac arrest, which is a rare complication of reversible TCM.

## Conclusion

Takotsubo syndrome (Stress induced Cardiomyopathy) is an adverse event whose relationship with anaesthesia and surgical stress should be studied because of its specific course, which is different from typical myocardial ischemia [11].

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