

Stress-induced Takotsubo Cardiomyopathy Following Traumatic Brain Injury: A Critical Diagnostic Challenge

Puteri Amira Mohd Hassan^{1,2}, Normalinda Yaacob², Mohd Zailani Abd Shariff¹,
Azlizawati Azil¹, Balarajan Nagaraja¹, Jahlelawati Zul¹, Adi Osman¹

¹ Emergency and Trauma Department, Hospital Raja Permaisuri Bainun, Jalan Raja Ashman Shah, 30450 Ipoh, Perak, Malaysia

² Department of Emergency Medicine, School of Medical Sciences, Universiti Sains Malaysia,, 16150 Kubang Kerian, Kelantan, Malaysia

Abstract

Takotsubo cardiomyopathy (TC), also known as “broken heart syndrome” or “stress cardiomyopathy”, is characterised by transient cardiac dysfunction with left ventricular apical ballooning triggered by physiological or physical stress. We present a case of traumatic brain injury (TBI)-induced TC, which highlights the importance of point-of-care ultrasound (POCUS) and transoesophageal echocardiogram (TOE) as real-time diagnostic tools for identifying TC in TBI patients. These tools are pivotal in the early detection of TC and in guiding interventions for the management of this condition in trauma patients.

Keywords: *takotsubo cardiomyopathy, traumatic brain injury, POCUS, TOE*

INTRODUCTION

Takotsubo cardiomyopathy (TC), also referred to as “broken heart syndrome” or “stress cardiomyopathy”, is a unique and uncommon condition characterised by transient cardiac dysfunction with left ventricular apical ballooning triggered by physiological or physical stress and is more common in females. TC can be caused not only by intense emotional stress but also by factors such as head injuries, strokes, medical illnesses (such as sepsis), surgery, excessive natural catecholamines, or certain medications.¹

Early identification guides clinical decisions, optimising the timing of surgical interventions and reducing the risk of complications. Left ventricular wall motion abnormalities in the TC typically resolve spontaneously within hours to weeks. The incidence rates of TC have been reported to be 10% among individuals with traumatic brain injury, 18.4% among individuals with intense physical activity or trauma, and 15% among individuals with perioperative myocardial damage and hip fracture.² It has also been reported that significant brain injuries, such as subarachnoid, account for 1.2 to 28% of cases but are infrequent in patients with intracerebral

haemorrhage, ischemic stroke, and traumatic brain injury (TBI).³ We report a case of severe traumatic brain injury in a patient who developed unexplained hypotension and evidence of TC within hours after injury.

CASE REPORT

A 21-year-old Malay woman with no known medical illness and alleged high-impact motor vehicle accident presented to the Emergency Department (ED) of the district hospital with a Glasgow Coma Scale (GCS) score of E2V1M4 and unequal pupils. The primary survey revealed a laceration wound over the left parietal region with a scalp hematoma and swelling over the right mandibular region. She was intubated for cerebral protection. Post intubation, her blood pressure (BP) dropped to 71/54mmHg, with a heart rate (HR) of 98 beats per minute. She was resuscitated with 20cc/kg normal saline and required inotropic support. She was transferred to a tertiary hospital for further treatment. The patient had received 1 litre of normal saline, packed cell transfusion, and inotropic support. The BP was picked up to 119/65mmHg but the HR increased to 119 beats per minute. Hence, E-

Fast was repeated, and free fluid was noted over the pouch of Douglas.

A computed tomography (CT) scan of the head revealed acute intraparenchymal haemorrhage in the left frontal and left parietal lobes, with multiple punctate haemorrhages in the bilateral frontal and right temporal lobes. The CT image of the face showed right angle and left parasymphysis of the mandible fracture, and the CT image of the cervical showed no fracture. The patient had undergone CT of the abdomen and pelvis on the same day and was reported to have no solid intra-abdominal organ injury but presence of a fracture in the bilateral superior pubic rami.

The blood investigation showed no drop in haemoglobin (Hb) compared to the investigation done upon arrival with Hb 10.7g/dL, white blood cells were $14.1 \times 10^9/L$, haematocrit was 32.2%, and platelets were $301 \times 10^9/L$. The cardiac marker troponin I (4396.8 H ng/L) was significantly raised. An electrocardiogram (ECG) showed T inversion at leads V2 and V3. Transthoracic echocardiography (TTE) revealed apical ballooning of the left ventricle with low ejection fraction (EF 20%). A bedside transoesophageal echocardiogram (TOE) was performed to look for thoracic aorta injury. The TOE showed that the left ventricle and the left atrium were dilated, the EF as before with global hypokinesia, apical ballooning seen, mild to moderate mitral regurgitation, the right ventricle and right atrium were not dilated, normal tricuspid annular plane systolic excursion (TAPSE), no pericardial effusion and the left ventricular outflow tract (LVOT) was not dilated, and no intramural flap was observed (Figures 1, 2 and 3). The descending aorta was not dilated, and no intramural flap was seen.

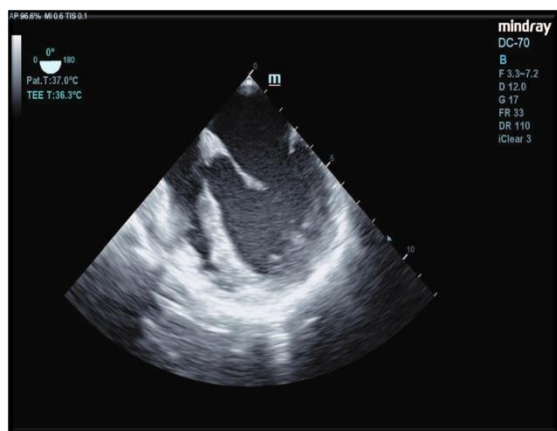


Figure 1: Bedside TOE with an EF of 20% and apical ballooning



Figure 2: Video of TOE (scan QR for the video)



Figure 3: Video of TOE with colour Doppler (scan QR for the video)

All possible causes of hypotension in trauma patients were ruled out, and the patient was diagnosed with extreme stress-induced cardiomyopathy precipitated by trauma. Inotropic support was continued, and the patient was put on cerebral protection protocol.

The patient was admitted to the intensive care unit (ICU). She was treated with double inotropic support: noradrenaline and dobutamine. Her blood pressure gradually increased, and she was able to wean off the inotropes on day 5 of injury. She was extubated, and tracheostomy was performed on day 9 after the injury. No repeated ECHO was done, and her cardiac troponin I result was down-trended. She was fully awake and kept well. The patient was transferred to the general neurosurgical ward with stable hemodynamic.

DISCUSSION

Cardiac dysfunction is one of the complications in individuals who experience TBI, and TC is a common manifestation of this complication. TC is a rare condition that has been reported in severe traumatic brain injury. In a longitudinal cohort study done by Cinotti R. et al., TC was reported in 10% of traumatic brain injuries.⁴ "Takotsubo" comes from a Japanese word that means "octopus pot", and this disease is characterised by temporary dysfunction in both the systolic and diastolic phases of the left ventricle, resulting in diverse wall motion abnormalities. The exact mechanisms behind TC are not entirely clear, but available evidence indicates that the development of this condition is influenced by various factors. These

include the brain–heart axis with excessive sympathetic system activation, abnormalities in microvascular and myocardial tissue metabolism, and coronary artery vasospasm.²

Patients with brain injuries demonstrating ST-segment changes on their electrocardiogram (ECG) and a significantly reduced left ventricle ejection fraction on echocardiography warrant a heightened index of suspicion for transient TC.⁵

In the revised Mayo Clinic criteria of 2008, the diagnostic criteria for TC are as follows: (1) transient hypokinesia, akinesia, or dyskinesia observed in the mid segments of the left ventricle, with or without apical involvement; (2) regional wall motion abnormalities extending beyond a single epicardial vascular distribution, often associated with a stressful trigger, though not consistently; the absence of obstructive coronary disease or angiographic evidence of acute plaque rupture; (3) new ECG abnormalities such as ST-segment elevation and/or T-wave inversion, or a modest elevation in cardiac troponin; and (4) the exclusion of pheochromocytoma and myocarditis. All four criteria must be met for a definitive diagnosis.⁶

The implementation of TOE as an adjunct to point-of-care ultrasound (POCUS) in the resuscitation of acutely ill patients has generated interest. In the context of acutely injured patients, TOE plays a crucial role in delivering relevant diagnostic information to guide resuscitation efforts, particularly in the detection of life-threatening hidden thoracic injuries in scenarios involving patients with multi-system trauma, providing enhanced visualisation of cardiac anatomy, improved haemodynamic assessment, heightened diagnostic accuracy, and real-time evaluation during resuscitation, as well as for identifying evidence of shock with unclear aetiology.⁷

A hypotensive traumatic patient presenting with physiological symptoms might not all be suggestive of haemorrhage or bleeding. A comprehensive evaluation of trauma patients with unexplained hypotension emphasised that TC should be part of the differential diagnosis, particularly in patients with multi-system injuries. Accurate diagnosis during the initial assessment is crucial for the proper management of TC. A recommended strategy involves a trained physician conducting bedside TOE while concurrently implementing other critical resuscitative measures.

In our patient, TC was detected early, and inotropic support was initiated to improve hemodynamic stability and cardiac function. Adequate hemodynamic support is imperative for patients with severe

traumatic brain injury, aligning with current treatment principles that underscore the preservation of sufficient cerebral perfusion pressure (CPP). The cardiac function of individuals experiencing TC could be improved by considering interventions such as dobutamine, whether pharmacological or non-pharmacological, including extracorporeal life support.³

TC generally has good outcomes, and the resolution of cardiac function has been observed as fast as a few hours to 12 weeks.³ Unfortunately, despite her recovery of cardiac function, her neurological function remains poor, and she requires continuous 24-hour care.

CONCLUSION

This case proves the importance of POCUS and TOE in evaluating critically ill or injured patients in the ED. These diagnostic tools provide crucial insights that may not be accessible through other means, particularly in trauma patients with unexplained hypotension. The early detection of TC as a potential complication of TBI is vital, as it is often associated with impaired left ventricular function. Despite its rarity, TC is a reversible condition with a generally positive prognosis, highlighting its significance in clinical practice and the importance of recognising it in trauma care.

CONFLICT OF INTEREST

The authors declare that they have no competing interests and do not receive any financial support.

CORRESPONDENCE

Dr. Normalinda Yaacob
MD (UKM), M.Med (Emergency Medicine)
Department of Emergency Medicine,
School of Medical Sciences,
Kubang Kerian, 16150 Kota Bharu,
Kelantan, Malaysia.
Email: drnorma_yaacob@usm.my

REFERENCES

1. Wang F, Darby J. Case Report: Takotsubo Cardiomyopathy After Traumatic Brain Injury. *Front Neurol*. 2021 Sep 15;12.
2. Fernandez CA, Narveson JR, Walters RW, Patel ND, Veatch JM, Ewing KL, et al. Takotsubo Cardiomyopathy and Trauma: The Role of Injuries as Physical Stressors. *Cureus*. 2022

3. Cheah CF, Kofler M, Schiefecker AJ, Beer R, Klug G, Pfausler B, et al. Takotsubo Cardiomyopathy in Traumatic Brain Injury. *Neurocrit Care*. 2017;26(2):284-91.
4. Cinotti R, Le Tourneau T, Bach-Ngohou K, Le Courtois du Manoir M, Rozec B, Asehnoune K. A longitudinal cohort of stress cardiomyopathy assessed with speckle-tracking echocardiography after moderate to severe traumatic brain injury. *Crit Care*. 2020;24(1):216.
5. Gruhl SL, Su J, Chua WC, Tay KV. Takotsubo cardiomyopathy in post-traumatic brain injury: A systematic review of diagnosis and management. *Clin Neurol Neurosurg*. 2022;213:107119.
6. Contreras Gutiérrez VH. Takotsubo cardiomyopathy: A case-report. *Revista Médica del Hospital General de México*. 2018;81:41-6.
7. Girón-Arango L, D'Empaire PP. Is There a Role for Transesophageal Echocardiography in the Perioperative Trauma Patient? *Curr Anesthesiol Rep*. 2022;12(2):210-6.