

Case Report

Takotsubo Cardiomyopathy in Traumatic Brain Injury: A Case Report

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Abstract

Background: Takotsubo cardiomyopathy (TC) is a well-known complication of severe brain insult but has been rarely described in patients with traumatic brain injury (TBI).

Methods: Case report and review of literature.

Results: We report a 25-year-old lady with moderate traumatic brain injury (TBI) developing circulatory shock. Takotsubo cardiomyopathy (TC) was diagnosed by repeated echocardiography. Cardiovascular support by pressor agents led to hemodynamic stabilization after initiation of noradrenaline. Cardiac function fully recovered within 2 weeks. We performed an in-depth literature review and identified 17 reported patients with TBI and TC. Clinical course and characteristics are discussed in the context of our patient.

Conclusion: TC is rarely diagnosed after TBI and may lead to poor outcome if not addressed in time.

Introduction:

Takotsubo cardiomyopathy (TC) is a transient cardiac syndrome that involves left ventricular akinesis and mimics acute coronary syndrome (ACS). It was first described in Japan in 1990 by Sato et al [1, 2]. Although the exact etiology of TCM is still unknown, the syndrome appears to be triggered by a significant emotional or physical stress [3]. It has been widely described after severe brain insult like subarachnoid hemorrhage (SAH, 1.2–28 %) [4–6]; however, it rarely occurs in patients with intracerebral hemorrhage, ischemic stroke, and traumatic brain injury (TBI) [7]. Here, we report a case of moderate TBI presenting with severe TC and provide a comprehensive review of all reported TBI cases [8–20].

Case Report

A previously healthy 25 years old Bangladeshi young lady got admitted to the general intensive care unit (GICU) of a tertiary care hospital through ER with moderate traumatic brain injury (TBI) three hours following a RTA.

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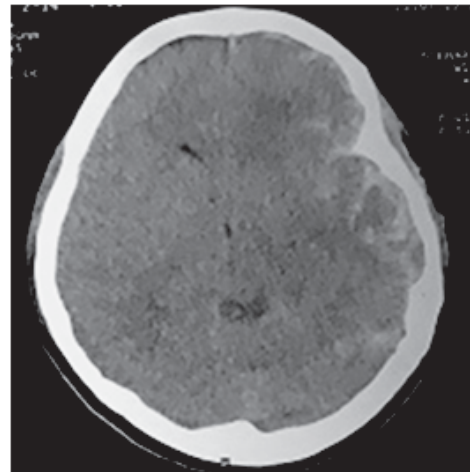


Fig 1: CT scan of head on admission

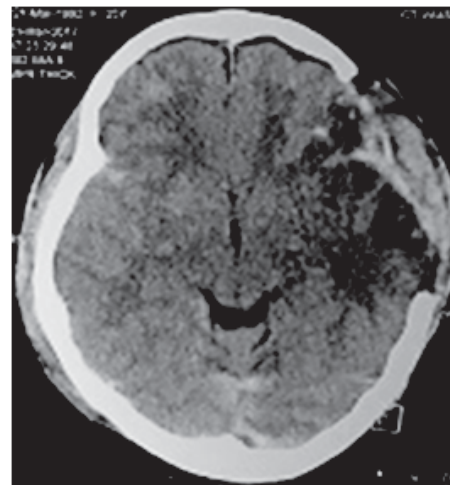


Fig 2: CT scan of head on 3rd POD

On presentation, she was disoriented and had a Glasgow Coma Scale (GCS) score of 11 (E₂V₃M₆). No focal neurological deficit was present and her vital signs were stable with BP 110/70 mm of Hg, HR 76bpm, regular. She vomited twice since the RTA and had no H/O any convulsion. Patient had multiple lacerations over her head and bleeding from right ear. There were no signs of trauma in any other parts of the body. CT scan of the head showed left fronto-temporal acute subdural haematoma with temporal contusion (Fig 1). Cervical spine screening was negative for any injury. All laboratory workups were normal except for some mild electrolyte imbalance. Initial ECG findings were also within normal limit. The following day patient developed hypotension associated with tachycardia (HR>130). All possible causes of hypotension including any concealed hemorrhage, acute MI or septic foci were ruled out. As patient remained hypotensive after adequate volume resuscitation, pressor support was required (Noradrenaline 0.4 mcg/kg/min). About 4 to 5 hours afterwards her GCS also dropped significantly from 11 to 9. At this time she was intubated and put on mechanical ventilator support for airway

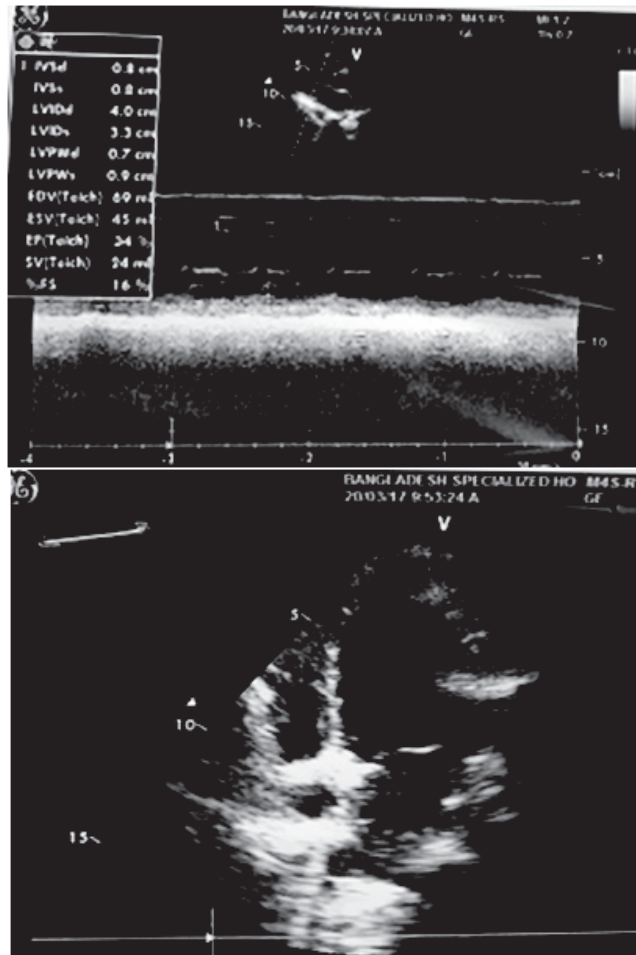


Fig 3: First ECHO on 2nd POD with EF 32%

protection. Patient underwent decompressive craniectomy (Fig 2) on that day and was kept sedated under mechanical ventilator support in the post-operative period. Patient was

otherwise stable but remained in shock requiring noradrenaline support. On the 2nd POD a bedside transthoracic echocardiography was done which showed global hypokinesia of septal, anterior, lateral and post-inferior LV with an EF of 31% (Fig 3). On the 3rd POD, patient's BP gradually increased requiring less noradrenaline support. Bedside echo was repeated which showed an increase in EF up to 42% (Fig 4). Patient was eventually extubated on the 4th POD when she was on minimum pressor support and was gradually weaned off noradrenaline on the 7th POD without any significant drop in BP. Patient was transferred out of GICU to cabin on the 9th POD with GCS 15/15, stable hemodynamics and in an afebrile condition. A bedside transthoracic echo done 2 weeks after the RTA showed no RWMA with an EF of 68% (Fig 5).

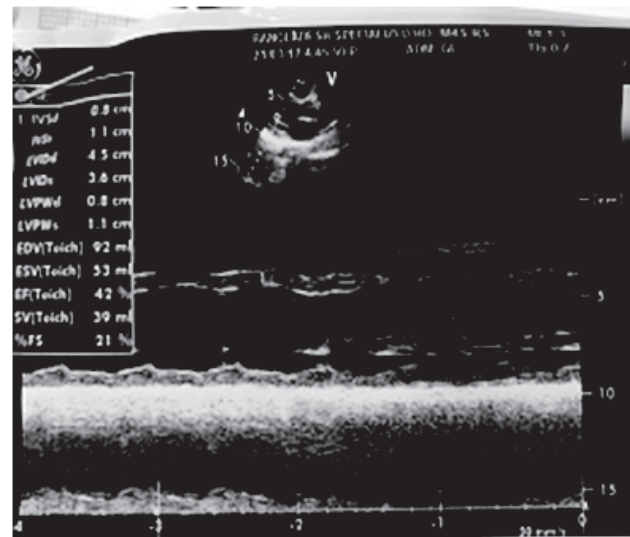


Fig 4: 2nd ECHO on 3rd POD with EF 42%

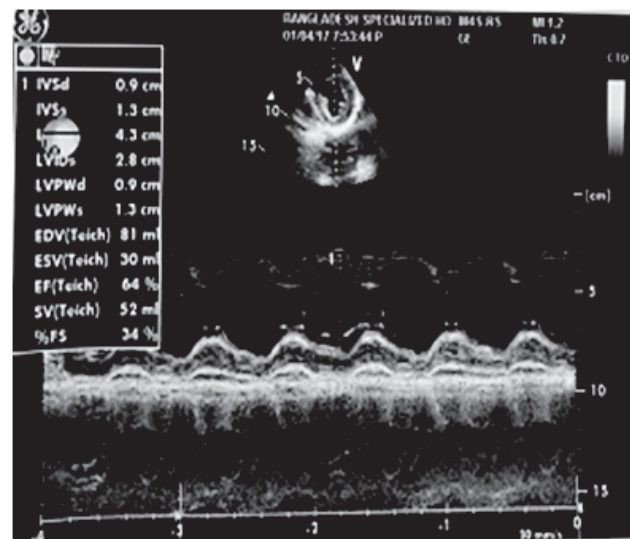


Fig 5: Normal ECHO 2 weeks after RTA

Review of Literature

Method

We performed a comprehensive literature search using the search terms ‘Takotsubo cardiomyopathy,’ ‘Tako-tsubo cardiomyopathy,’ ‘stress cardiomyopathy,’ ‘stunned myocardium,’ ‘Broken heart syndrome,’ ‘transient-left-ventricular ballooning syndrome,’ ‘apical ballooning syndrome,’ ‘myocardial dysfunction’ or ‘heart failure’ together with ‘traumatic brain injury,’ ‘head injury,’ and ‘polytrauma.’ Only articles in English language were included.

Results

Overall we identified 14 published articles involving 17 TBI patients with TC [8–21]. Among them, 14 were adults, all except two patients presented with loss of consciousness requiring endotracheal intubation and mechanical ventilation for airway protection. The brain injury pattern was variable including contusional hematoma, epidural hemorrhage (EDH), subdural hemorrhage (SDH), and traumatic SAH, 6 patients underwent neurosurgical intervention. TC was diagnosed within first day after admission in most patients. In 4 patients, coronary angiography was performed and confirmed TC. Electrocardiography abnormalities were found in 9/17 patients including ST segment and T wave changes, and 11/17 had elevated serum troponin level. Treatment differed; however, most received inotropic support using dobutamine. In addition, various drugs were used to sustain adequate blood pressure including adrenaline, noradrenaline, and vasopressin. In two patients, levosimendan was used. Echocardiography revealed abnormal results in all patients (100 %) and was reversible in the majority of patients within 7 days except in 3 patients where complete cardiac recovery took place after 12days, 17 days and 21 days respectively.

Discussion

Cardiovascular complications are common after brain injury and associated with increased morbidity and mortality. TC represents a serious manifestation of myocardial dysfunction and is defined as an acute, transient, and reversible heart failure syndrome due to regional wall abnormalities of the ventricular myocardium with associated new electrocardiography changes and elevation of myocardial biomarkers in the absence of culprit atherosclerotic coronary artery disease or cardiac condition causing the temporary ventricular dysfunction [22,23]. Underlying pathophysiologic mechanisms are still incompletely understood. Most investigations suggest an interconnected cascade of neuronal injury causing sympathetic overstimulation and direct catecholamine toxicity to the heart [24]. Damage to the insula and hypothalamus also initiates a complex cascade of events, including activation followed by dysfunction of the autonomic nervous systems and an intense inflammatory response, which have major adverse effects on the heart (Fig 6) [25].

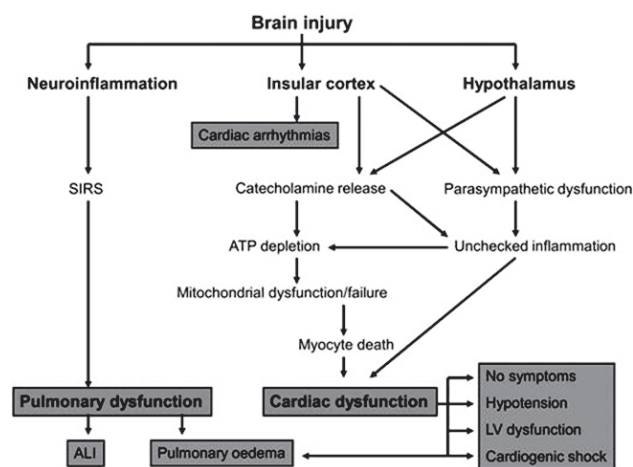


Fig 6: The pathophysiology of cardiovascular complications after brain injury.

SIRS, systemic inflammatory syndrome; ATP, adenosine triphosphate; ALI, acute lung injury

Our patient had full recovery of cardiac function 2 weeks after trauma. Even though transient and reversible in nature, some reports suggest recovery even up to 12-week post injury. Improving cardiac function in patients with TC may be achieved by using dobutamine, noradrenaline and other pharmacological or nonpharmacological treatment including extracorporeal life support [26]. Our patient improved after starting noradrenaline. Recently, the use of levosimendan has been reported in patients with aneurysmal SAH where other inotropes were found ineffective [27].

Conclusion

Takotsubo cardiomyopathy is an underdiagnosed disorder especially in patients suffering from traumatic brain injury. It is a reversible condition associated with good overall prognosis. Supportive management is the mainstay of treatment for this condition.

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