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Case Report

Takotsubo Cardiomyopathy as a Complication/Sequelae of Prolonged Refractory Seizure: A Case Report from Nepal

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ABSTRACT

Takotsubo cardiomyopathy(TC) is a reversible, yet underdiagnosed cause of mortality and morbidity in the intensive care units. It occurs secondary to sudden catecholamine surge precipitated by any form of emotional or pathological stress. Association between central nervous system disorders and Takotsubo cardiomyopathy is being increasingly reported. Epilepsy is the second most common CNS disorder to trigger TC, SAH being the first. We report a case of TC in an elderly man with prolonged, recurrent seizure episodes refractory to the commonly used antiepileptic drugs (AEDs), who developed unexplained tachycardia, hypotension and elevated cardiac enzymes.

Takotsubo cardiomyopathy is an acute cardiac syndrome characterized by sudden onset chest pain or dyspnea, with ST-segment elevation and transient left ventricular dysfunction. The term "Takotsubo" was first described in 1990 in Japan, taken from the Japanese name for an Octopus' trap, which has a shape that is similar to the systolic apical ballooning appearance of the left ventricle [1]. TC is also known as ampulla cardiomyopathy, apical ballooning, neurogenic stress cardiomyopathy, broken heart syndrome, neurogenic stunned myocardium, transient regional left ventricular dysfunction, transient myocardial dysfunction, transient systolic dysfunction, neurogenic stressed myocardium, catecholamine cardiomyopathy, or reversible acute heart failure [2-3]. Central nervous system (CNS) disease is increasingly recognized as a trigger of TC. The association of seizure with TC is often underreported but if diagnosed and treated early, it is associated with low mortality and is considered reversible in most cases.

Case Report

69 years male with a history of Parkinsonism, old CVA with left-sided hemiparesis, and hypertension presented with uncontrolled abnormal body movement, facial deviation to the right side, and slurring of speech. Seizure was refractory, not controlled by intravenous medications, and the patient being unresponsive, he was intubated for airway protection and put on mechanical ventilation. Paralytics and fentanyl infusion were used to provide controlled ventilation. He was a non-smoker and also didn't consume alcohol. He was loaded with inj phenytoin and inj levericitam and placed on continuous electroencephalogram (EEG) which demonstrated abnormal nonspecific findings. CT head showed cerebral atrophy with deep white matter hypodensity in the periventricular region, corona radiata, and cerebral hemispheres. Since all were inconclusive, Lumbar puncture was done for CSF analysis which showed TC 15 cells/cumm, glucose 89 mg/dl, protein 29 mg/dl, ADA 1.68 with no growth on culture. Other laboratory investigation findings were hypokalemia with serum potassium 2.7 mmol/l, hypocalcemia with calcium 7.5, and hypomagnesemia with magnesium 1.3 (Figure 1).

Serial electrocardiogram (ECG) demonstrated sinus tachycardia with ST elevations in V2 and V3 with initial troponin was within normal range. Cardiology consult was obtained and ECHO demonstrated hypokinetic apical septal, lateral, anterior, and inferior segments, and

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mid anteroseptal segments and apical cap, LV systolic dysfunction with ejection fraction 40%, and moderate AR (Figure 2).

A few hours post-admission a rapid decrease in blood pressure (BP) (SBP 80–90s and DBP 40-45s) was noted, requiring the use of noradrenaline to maintain SBP > 100. Tachycardia and ST-elevation persisted in V1–3 leads. Later, phenylephrine infusion was added to maintain hemodynamics.

Secondary to hypoxia, excess secretions, CT scan of the chest was performed which revealed multifocal bilateral consolidations and bilateral pleural effusion, cardiomegaly, right and left failure, and negative for pulmonary emboli. ET aspirate culture had Acinetobacter spp growth for which antibiotics inj polymyxin b and inj meropenem were used.

Antiepileptics inj sodium valproate and tab phenobarbitone were subsequently added to control seizure outbursts along with midazolam continuous infusion. Midazolam was stopped gradually with the continuation of oral anti-epileptic medications.

The patient was under tab rosuvastatin, tab ecosprin, tab myoxin for cardiac stabilization and control of arrhythmia. Injection noradrenaline and inj phenylephrine infusion were started to maintain hemodynamics.

Tracheostomy was carried out as the patient was not able to wean off from a mechanical ventilator. Following tracheostomy, the patient's oxygenation was maintained on a tracheal mask.

Subsequent Echocardiography revealed improvement in the patient's cardiac status with ejection fraction\60%. Phenylephrine and noradrenaline infusion could be gradually tapered off.

Figure 1- ECG demonstrating low voltage, t wave inversion from V1-V6

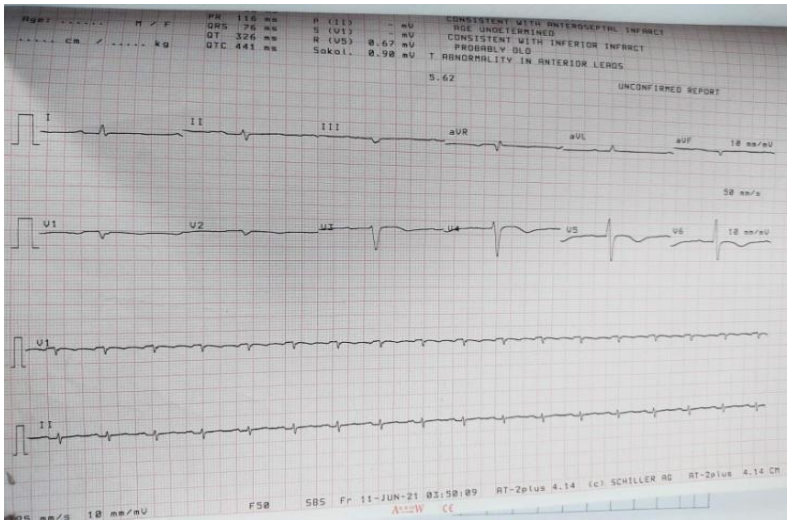
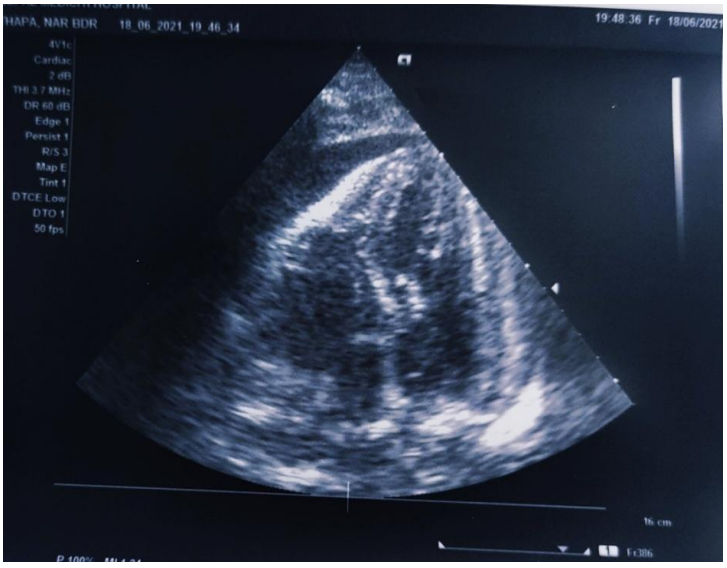


Figure 2- Echogardiocraphic view demonstrating Apical, septal, and inferolateral akinesis (white arrows)



Discussion

Takotsubo cardiomyopathy may present as a novel form of heart failure that is precipitated by sudden, unexpected emotional distress. It is common among elderly women. Abnormal catecholamine rise due to emotional distress seems to play a major role in the pathogenesis of this cardiomyopathy, rendering takotsubo cardiomyopathy a type of neurocardiological disorder that manifests as acute but reversible heart failure. TC is characterized by akinesis or hypokinesis of the mid and distal segments of the left ventricle coupled with a lack of vascular abnormalities on cardiac catheterization [2].

TC usually mimics acute STEMI with symptoms such as chest pain, dyspnea, syncope, and near-syncope. Although the exact cause is unknown coronary artery vasospasm, microcirculatory dysfunction, and transient obstruction of the left ventricular outflow tract have been proposed as possible causes of this disorder [4].

Diagnostic workup includes ECG and cardiac enzymes where ECG often reveals ST-segment elevations or depressions, deep and widespread T wave inversions, transient left bundle branch block, and arrhythmias [5]. Cardiac biomarkers such as troponin are elevated which can be as high as 24 times the upper limit of normal. Brain natriuretic peptide (BNP) can help to differentiate it from true ACS, as BNP levels are disproportionately high in takutsubo cardiomyopathy.

Mayo Clinic criteria for Takotsubo cardiomyopathy: a) Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always present. b) Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture c) New electrocardiographic abnormalities (either ST-segment elevation and/or T wave inversion) or modest elevation in cardiac troponin. d) Absence of pheochromocytoma or myocarditis [6].

TC has been documented with multiple medical conditions (hypoglycemia, pneumothorax, alcohol withdrawal, neuroleptic malignant syndrome, pheochromcytoma, sepsis, hemodialysis, and post anesthesia [2,7]. A less common etiology is acute neurologic dysfunction; this includes intra-axial bleeding, ischemic strokes, migraines, and epilepsy [5]. Epilepsy is the second most common CNS disorder to trigger TC, SAH being the first [8].

Prolonged seizure may increased the catecholamine levels, thus provoking takotsubo syndrome.Cardiogenic shock caused by takotsubo cardiomyopathy may be the acute presentation in some cases. The most common complication associated with TC is cardiogenic shock, though dysfunction can range from congestive heart failure, pulmonary edema, ventricular septal defect, ventricular arrhythmias, atrial fibrillation, conduction abnormalities, and stroke [2-3, 7].

CAD and acute STEMI are the primary differential diagnosis and should be ruled out based on presenting symptoms and the ECG findings.Patients with subarachnoid hemorrhage (SAH) also have myocardial dysfunction.

Takotsubo cardiomyopathy carries a mortality rate of up to 8% [9]. Investigation measures such as ECG, echocardiography, assessment of serum troponin levels, coronary angiography, and pharmacotherapy should be carried out without delay. Education of the patients and their close contacts are needed to ensure early recognition of seizures to prevent progression to status epilepticus and the development of takotsubo syndrome.

In general, the goal of treatment is to maintain an adequate cardiac output and hemodynamic stability. Intravenous fluids and short-term use of vasopressors may be sufficient as in most cases, it is reversible.

Conclusion

Catecholamines may be central to the mechanism of stress-related myocardial stunning and TC, further research awaits for a more complete understanding of the pathogenesis of this syndrome. TC as sequelae to seizure could be life threatening if not addressed.

References

[1] 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.

[2] Abdulla I, Kay S, Mussap C, Nelson GI, Rasmussen HH, Hansen PS, et al. Apical sparing in tako-tsubo cardiomyopathy. Intern Med J. 2006; 36(7):414-8.

[3] Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med. 2005; 352(6):539-48.

[4] Stöllberger C, Wegner C, Finsterer J. Seizure-associated Takotsubo cardiomyopathy. Epilepsia. 2011;52(11):e160-7.

[5] Dawson DK. Acute stress-induced (takotsubo) cardiomyopathy. Heart. 2018;104(2):96-102.

[6] Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. Am Heart J. 2008;155(3):408-17.

[7] Bennett JR, McCarty D, Wilson CM, O'Neill F. Takotsubo cardiomyopathy: an uncommon cause of ST segment elevation in intensive care. Anaesthesia. 2007;62(3):279-81.

[8] Finsterer J, Wahbi K. CNS disease triggering

Takotsubo stress cardiomyopathy. Int J Cardiol. 2014;177(2):322-9.

[9] 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 Intern Med. 2004;141(11):858-65.