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TAKOTSUBO CARDIOMYOPATHY WITH HISTORY OF SEIZURES: A CASE MIMICKING ACUTE CORONARY SYNDROME

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ABSTRACT

First described in Japan, Takotsubo cardiomyopathy (TTC) is increasingly becoming recognized worldwide as a cause of sudden and reversible diminished left ventricular function characterized by left apical ballooning and hyperkinesis of the basal segments, often with symptoms mimicking acute coronary syndrome. Associated with physical or emotional stress, its exact pathogenesis has not been established, though evidence supports a neurohumoral etiology. Seizures have been reported as triggers of Takotsubo cardiomyopathy. It is unknown if seizure associated TTC differs from TTC associated with other triggers. In this report, we present a rare case of Takotsubo cardiomyopathy in a young female who presented with chest pain having a history of transient attacks of seizures.

Key Words: Broken Heart Syndrome, Stress Cardiomyopathy, Apical Ballooning Syndrome, Ischemic Heart Disease

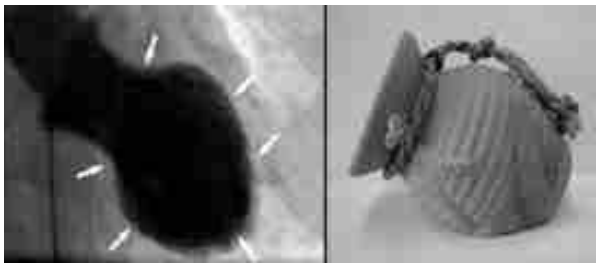
INTRODUCTION

Left apical ballooning syndrome, also known as Takotsubo cardiomyopathy (TTC), is a clinical syndrome of transient diminished left ventricular (LV) apical wall motion with relative preservation of the basal heart segment in the presence of normal coronary arteries. It was first described in Japan in the early 1990s and is named for ventricles which seem similar in appearance to a Japanese octopus trap on ventriculography scans(Figure 1).[1] The etiology of TTC is not completely understood. Several possible mechanisms, including microvascular dysfunction, coronary artery vasospasm, aborted myocardial infarction, and excess catecholamine stimulation, have all been proposed.[2,3] Typically, LV function returns to normal within six to eight weeks. Recurrence, which is increasingly being reported in the literature, can be related to neurological pathology mainly status epilepticus.[2-7] We present a case of a woman with history of generalized tonic clonic seizures that was having takotsubo cardiomyopathy / broken heart syndrome mimicking ACS.

CASE REPORT

A 22 years old young female presented to OPD with the three weeks history of severe, retrosternal chest pain at rest, gripping in nature, radiating to neck and

Figure 1: Left Ventriculogram of Takotsubo Cardiomyopathy Patient



Left: Ventriculogram (Contracting phase) of a patient with takasubto syndrome. Right: The Japanese takasubto (ceramic pot used to trap octopus) has a shape closely resembles to that of heart on left side. Image Courtesy by Dr. Stoshi Karisu-Japan

associated with sweating. She was having exertional dyspnea for two weeks (NYHA II-III), which was relieved by rest, and occasional episodes of Paroxysmal nocturnal dyspnea (PND). Patient had a history of generalized tonic clonic seizures almost one year ago that occurred in closed succession during which she had tongue bite and broke her tooth deeming it clinically status epilepticus. She took medical treatment for it for two months and improved. Having no more complaints of fits afterwards she discontinued medical treatment onward.

The patient have a depressive personality due to poor socioeconomic status and early death of her siblings including one brother and one sister who died due to unexplained cause rendering her severe emotional stress. On presentation, the patient was dyspnoeic, afebrile, normotensive (110/70 mm Hg) and had a heart rate of 80 beats/min. Physical examination revealed ill sustained, heaving, displaced apex beat to the left side 1 cm lateral to the mid clavicular line. No jugular venous distention or lower extremity edema was noted, and the lungs were clear on auscultation. The patient's complete blood count, renal function tests, basic metabolic panel and liver function tests were all within the normal range. ESR was reported above normal. Examination of CT brain showed no gross structural neurologic abnormalities. ECG revealed ischemic changes in the anterolateral leads (Figure 2).

Transthoracic echocardiography (TTE) demonstrated anteroseptal hypokinesia and basal hyperkinesia of LV with apical aneurysmal ballooning, ejection fraction approximately 40% with no definite clot at Lv apex. Rest of the parameters was normal (Figure 3). These kinetic changes and apical morphology were confirmed by LV gram. Coronary angiography was normal establishing the diagnosis of TTC. Patient was relieved by medication which included Aspirin (150 mg), Clopidogrel (75mg), Isosorbide dinnitrate (20mg), β-blockers, ACE inhibitors, statins along with LMWH and narcotics. She was discharged with continued treatment of anti-ischemic therapy along with low dose diuretic therapy.

Future risk may be prevented by avoiding stressful and depressive environment. Further studies will hopefully enable early diagnosis of TTC, clarify its etiology, refine therapy regimen and aid in its prevention.

DISCUSSIONS

Presenting with overlapping symptoms and initial findings, it is difficult to distinguish Takotsubo cardiomyopathy from an acute coronary syndrome. A history of recent emotional trauma can prompt a clinician to consider Takotsubo cardiomyopathy; however, emotional triggers have also been associated with acute coronary syndrome. Histological and nuclear imaging data in humans have shown regional differences in efferent sympathetic innervation where the basal ventricular wall possesses a greater mean density of nerve endings and local catecholamine concentrations compared to the apex, while the apex possesses higher concentrations of adrenoreceptors. This differential distribution is proposed to induce the LV dysfunction found in patients with TTC.[2,8,9] Neurogenic myocardial stunning has also been proposed as a cause of the findings in patients with TTC.[2,10] In the setting of intracranial injury or dysfunction, such as the catecholamine toxicity a surge in sympathetic release will cause a relative hyperdynamic contraction of the LV basal segments with a relative stunning, or ballooning, of the LV apical portion due to saturation of the adrenoreceptors in that distribution.[2] Therefore, there appears to be a correlation between intracranial dysfunction and LV dysfunction. Our present report, however, describes a patient TTC in the setting of previous history of epilepsy without structural evidence of neurologic dysfunction.

Figure 2: ECG Shows ST-elevation and T Wave Inversion in Anterolateral Leads

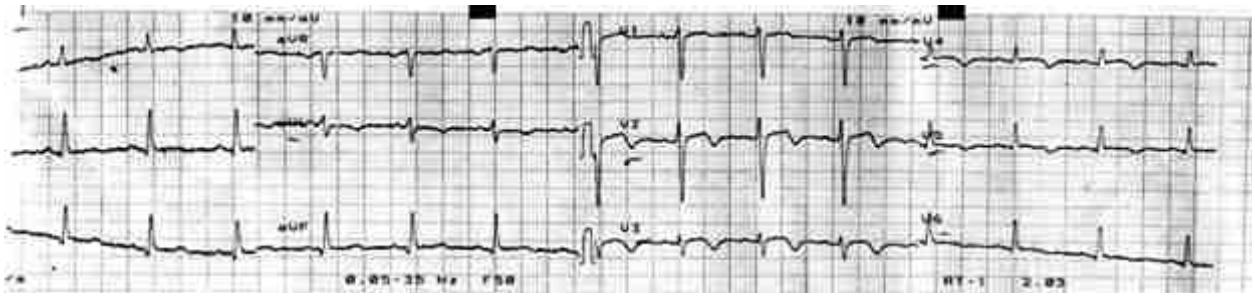
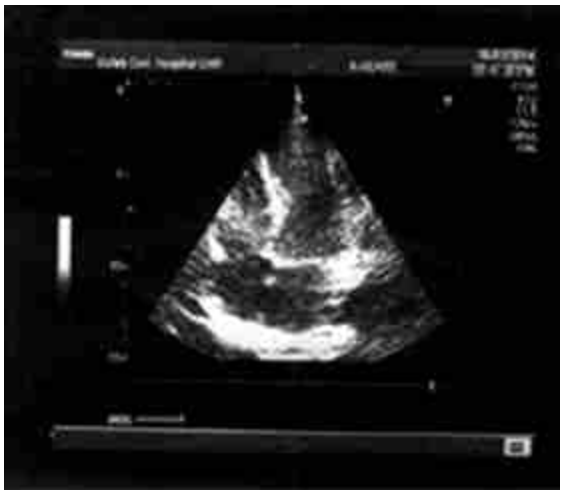


Figure 3: Echocardiography of Takotsubo Cardiomyopathy Patient



In the case described here, echocardiography revealed akinesis of the apex and the mid ventricle as well as basal hyperkinesis, wall motion abnormalities extending beyond the region supplied by one coronary artery. This feature is characteristic of Takotsubo cardiomyopathy, whereas wall motion abnormalities observed in acute coronary syndrome are often more localized. Myocardial viability is preserved in Takotsubo cardiomyopathy, which can be demonstrated using cardiac MRI. The most definite and standardized criteria to confirm TTC is as follows.

All four are required

1) Transient hypokinesis, dyskinesis of the LV mid segment with or without apical involvement. The regional wall motion abnormalities typically extend beyond a single epicardial coronary distribution.

2) Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.

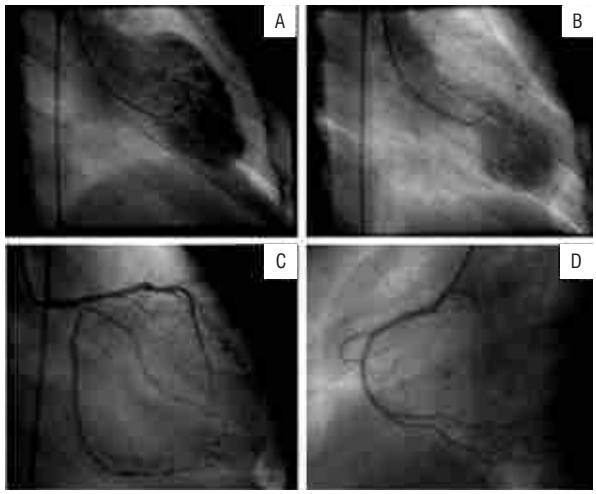
3) New ECG abnormalities (either ST-segment elevation and/or T wave inversion) or modest elevation of cardiac Troponin.

4) Absence of pheochromocytoma & myocarditis.

CONCLUSION

Although no clearly defined etiology for TTC exists, clinicians should be aware of the possibility of TTC in patients whose presentation mimics acute coronary syndrome, especially in the setting of emotional, physical, and specifically neurological stress. Additionally, it has been reported that patients with one episode of TTC are at increased risk for recurrence.[2] Though this condition rarely leads to death, it is imperative that the clinician be aware of this syndrome to ensure the prompt initiation of appropriate supportive care so that a return of normal LV function can be achieved.

Figure 4: LV gram Left and Right Coronary Angiography



A = Diastole, B = Systole
C = Left Coronory Angiography, D = Right Coronory Angiography

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