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Focal Takotsubo Cardiomyopathy: A Case Report and Literature Review

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Abstract Takotsubo cardiomyopathy (TCC) or stress-induced cardiomyopathy, was first discovered in Japan in 1990 and is derived from the name "octopus pod" which describes the typical LV apical ballooning with a narrow base. The pathophysiology behind takotsubo remains incompletely understood. However, TCC association with conditions of catecholamine excess such as emotional or physical stress is well studied and is the most favored theory thus far. Atypical, rare forms of TCC such as mid-ventricular, basal, focal, and apical types have been identified with an incidence of 14.6%, 2.2%, 1.5%, and 81.7% respectively. [1] We present the case of a 48-year-old Caucasian female admitted status post seizure and drowning incident who was hemodynamically stable and with a physical examination grossly unremarkable on presentation. Her echocardiogram showed hypokinesis and regional wall motion abnormality involving the anterior septal and lateral walls. Her cardiac magnetic resonance imaging showed hypokinesia of the anterolateral mid to basal wall consistent with focal Takobsubo cardiomyopathy. The patient underwent cardiac catheterization, which showed non-obstructive coronary artery disease with anterolateral dyskinesis. She was discharged with close follow-up as an outpatient.

Keywords: *focal takotsubo cardiomyopathy, atypical takotsubo cardiomyopathy, cardiac resonance imaging*

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1. Introduction

Takotsubo Cardiomyopathy was first described in Japan in 1990 and is derived from the Japanese word "octopus pod" due to the resemblance in shape of the apical ballooning and the octopus traps. [1] TCC is characterized by transient wall motion abnormalities usually following catecholamine excess states such as pheochromocytoma, use of serotonin-norepinephrine reuptake inhibitor drugs, and emotional or physical stress. [2] According to the revised Mayo diagnostic criteria, TCC can be diagnosed by the presence of the following criteria: transient dyskinesis of the left ventricular wall, with evidence or absence of apical involvement often associated with a stressful trigger, however, may be absent. New ECG abnormalities or rising in cardiac troponin. No evidence of obstructive coronary disease. Absence of myocarditis or pheochromocytoma. [3]

Atypical forms of TCC have been identified and classified into mid-ventricular type, basal or reverse type, and focal type, with focal being the rarest form. [4] In the acute presentation, TCC can be indistinguishable from acute coronary syndrome with similar findings on electrocardiography, echocardiography, and elevated cardiac enzymes. [1] The use of cardiac magnetic resonance imaging in the diagnosis of focal takotsubo has been established to be an important tool to differentiate both etiologies, as this rare form is commonly misdiagnosed as acute coronary syndrome. [5,6,7]

2. Case Presentation

This is a 48-year-old lady with a past medical history of depression and previous seizures in the setting of Wellbutrin use, who was in her usual state of health at the swimming pool when she lost consciousness and fell forward. The patient was underwater for a few minutes before she was pulled to the side of the pool. She remained unresponsive and had five minutes of generalized body shakes. ALS was called. The patient regained consciousness and was found to be post-ictal. Upon arrival to the emergency room, her oxygen saturation was 85% on room air with a blood pressure of 128/78 and pulse of 67. The patient reported nausea and shortness of breath. Denied any chest pain, palpitations, dizziness, lightheadedness, or loss of urinary or bowel control. She was saturating 100% on a non-rebreather.

Her past medical history is significant for three previous seizures all in the setting of initiating or increasing the dose of Wellbutrin, the last seizure 10 years prior to presentation. Medications included

Wellbutrin and Prozac with the Wellbutrin dose increased to 75 mg PO daily two weeks prior to the presentation. On physical examination, the patient had a regular rate and rhythm, S1 and S2 sounds were present, and no murmurs, rubs, or gallops were appreciated. On lung examination, there were bilateral breath sounds without wheezing, rales, or rhonchi.

The patient had a normal metabolic profile, mild leukocytosis of 11.7, negative procalcitonin, chest X-ray, urinalysis, and blood cultures. She underwent brain Magnetic Resonance Imaging and Electroencephalography, which were negative. Troponin peaked at 1.730 and downtrend. Electrocardiogram showed normal sinus rhythm at 88 beats per minute, normal axis, normal PR, QRS, and QTc, ST depressions in inferior lateral leads with Q waves in septal leads, and poor R-wave progression. The patient was loaded with Aspirin 325 mg and she started on Aspirin 81 mg daily and Lipitor 40 mg daily.

An echocardiogram was done and showed EF of 60-65% with hypokinesis and regional wall motion abnormality involving the anterior septal and lateral wall. The patient underwent cardiac catheterization, which showed non-obstructive coronary artery disease with anterolateral dyskinesis as shown in the figures. Cardiac Magnetic Resonance Imaging showed hypokinesia of the anterolateral mid to basal wall. (Figure 1) Plasma metanephrines to rule out pheochromocytoma were sent and returned negative.

The patient was diagnosed with focal takotsubo cardiomyopathy and discharged on Metoprolol 12.5 mg twice daily. She was asked to discontinue Wellbutrin therapy. Follow-up echocardiography outpatient after 6 weeks showed complete resolution of wall motion abnormalities and normal ejection fraction.

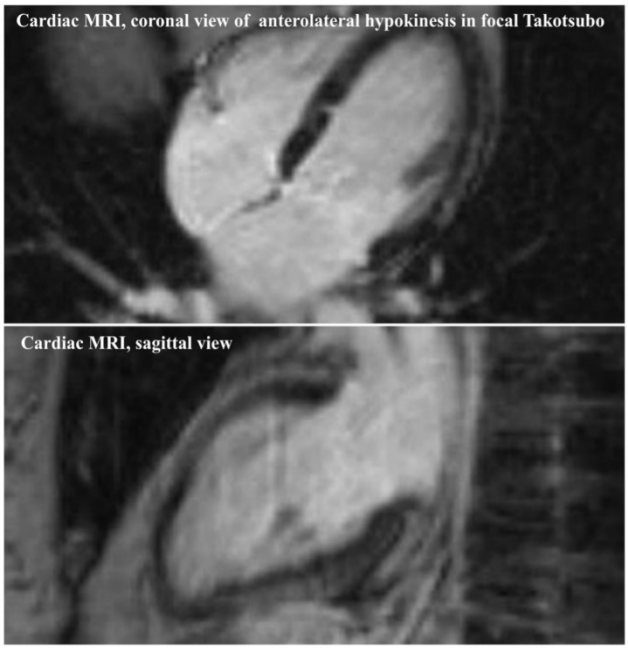

Figure 1.

3. Discussion

Diagnostic criteria for identifying patients with TCC include the following: transient dyskinesis of the left ventricular wall, no evidence of obstructive coronary disease or acute plaque rupture on angiography, presence of new ECG abnormalities, or raising of cardiac troponins, and absence of myocarditis or pheochromocytoma. [5] Furthermore, atypical forms of TCC were identified and are classified as mid-ventricular, basal (reverse takotsubo), or focal type based on wall abnormalities on LV angiography or transthoracic echocardiography. [5,8]

Cardiac magnetic resonance (CMR) represents a useful diagnostic tool for distinguishing between acute coronary syndrome (ACS) and focal takotsubo cardiomyopathy. One of the main features for differentiating between these two clinical entities is the evidence of regional wall motion abnormalities with extension beyond a single epicardial coronary artery present in takotsubo cardiomyopathy. Furthermore, biplane ventriculography should be performed on these patients, and if not available CMR or single-photon emission computed tomography should be performed for differentiating between ACS and focal TCC. [5]

A large retrospective study using data from the International Takotsubo Registry identified 1750 patients diagnosed with TCC between 1998 and 2014. Of the 1750 patients, 81.7% were diagnosed with typical TCC, 14.6% mid-ventricular, 2.2% basal, and 1.5% focal type. [5] Focal takotsubo is thus far identified as the rarest type of TCC with wall motion abnormalities typically in the anterolateral segment. [4] By comparing typical and atypical forms of TCC, the study outcome presented different clinical features including younger age, association with neurological disease, and less left ventricular ejection fraction (LVEF) impairment. Most importantly, the focal type had the least LVEF reduction. However, no significant differences were noted in the long-term outcome or hospital complications between the apical and non-apical types. [4]

A study published in Japan in 2016 proposed that atypical forms of TCC, specifically the focal type, are under-reported and misdiagnosed as acute coronary syndrome secondary to a transient thrombotic event or artery spasm. [5] The study design used the cardiac catheterization database of 3 hospitals in Japan and identified 144 patients diagnosed with TCC. Of the 144 patients, 59% of patients with the apical type, 34% were mid-ventricular, 0% basal, and 6.9% were focal type. The small sample size and the limitation of the patient population to Japanese descent limit the generalization of the study findings.

We conducted a literature search on PubMed using the words “focal takotsubo” and “atypical takotsubo”, we could identify 5 published case reports of focal takotsubo cardiomyopathy. Demographics, causes, clinical characteristics, and outcomes of all 6 cases are summarized in Table 1. 5 out of 6 patients were females, with a mean age of 58.3 (range 48 - 64). 3 out of 6 patients had an emotional trigger. All patients showed electrocardiography changes with 4 out of 6 presenting with ST-segment elevations. All patients had normal coronaries on angiography. 2 of the 6 cases had decreased LVEF on presentation (range 45 - 65%). Only 1 patient had negative cardiac enzymes on presentation. All patients had anterior wall motion abnormalities with 2 of 6 having anterolateral dyskinesis. All patients showed complete recovery on follow-up imaging. (Table 1)

Table 1.

Patient	A(presented case)	B [9]	C [10]	D [6]	E [7]	F [11]
Age in years	48	55	64	61	62	63
Sex	Female	Female	Male	Female	Female	Female
Stressor	Drowning and seizure	High dose IL-2	Alcohol withdrawal	Emotional stressor	Emotional stressor	Emotional stressor
Dyspnea	Yes	No	-	-	-	-
Chest pain	No	No	-	Yes	Yes	Yes
Wall motion abnormalities	Dyskinesis of anterolateral mid to basal wall	Hypokinesis of apical anterior and apical lateral wall segments	Focal anterior and septal akinesis of the left ventricle	Anterolateral ballooning	Focal dyskinesis of the mid anterior left ventricular (LV) wall	Mid- anterior segmental ballooning associated with basal, mid-inferior and apical hyperkinesis
Ejection Fraction on presentation	65%	45%	Decreased	55%	-	-
Troponin	Positive (1.730)	Negative	-	Positive	-	Positive (5.03)
Cardiac catheterization	Normal coronary arteries	Normal coronary arteries	Normal coronary arteries	Normal coronary arteries	Normal coronary arteries	Normal coronary arteries
Electrocardiogram changes	ST depressions in inferior lateral leads with Q waves and poor R wave progression in septal leads	T-wave inversions in the anterolateral leads	ST- segment elevation was observed in leads I, a VL , and V2	Minimal ST-elevation (0.5 mm) in I and aVL and poor R wave progression V1–V4	ST-elevation in V1-V2	ST-elevation in leads V2–4.
Outcome	Recovered	Recovered	Recovered	Recovered	Recovered	Recovered

The management of this condition is often with a conservative approach and focuses on relief of the emotional or physical burden that may be the trigger of this pathology. It has been reported in the literature that the use of angiotensin converter enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs) has been associated with improved survival at one-year follow-up in patients with TTC with or without heart failure. [1]

Up to 5-10% of the patients can develop cardiogenic shock which may or not be associated with the evidence of left ventricular outflow tract obstruction (LVOTO). In patients without LVOTO, inotropic agents such as dopamine or dobutamine may be beneficial, however, if LVOTO is present, inotropic agents should be avoided and fluid resuscitation should be started with closing monitoring of signs of pulmonary vascular congestion. [12,13]

During the course of this illness, the patient can also develop acute heart failure which can be managed with goal standard medical therapy including the use of ACE inhibitors or ARBs along with diuretics unless there is evidence of LVOTO in which case preload and afterload reduction should be avoided. [14]

The focal takotsubo type is the rarest form and is not well-reported in literature. It is associated with less left ventricular ejection fraction reduction compared to typical TCC.

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