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"Nutritional or Hormonal", The Myth of Takotsubo Cardiomyopathy and Left Ventricular Thrombosis in Anorexia Nervosa Patients! A Case Report

Case Report

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Abstract

Takotsubo Cardiomyopathy is a condition characterized by transient LV hypokinesis without evidence of obstructive coronary disease that is typically triggered by a stressful event. We describe a 34-year-old woman with anorexia nervosa presenting with hypotension, hypoglycemia, and tachycardia and found to have Takotsubo Cardiomyopathy. On admission, EKG showed ST depression with T wave inversion in the inferolateral leads and Q waves in the anteroseptal leads (V1-V3). The initial troponin was elevated at 1.2 ng/ml and increased to 1.94 ng/ml 4 hours later. Initial TTE showed global hypokinesisexcept for a hyperkinetic basal segment suggestive of takostsubo cardiomyopathy and an EF of 15-20%. During the course of management, subsequent TTEs revealed improved EF with resolution of the cardiomyopathy, but also the presence of an LV thrombus. The pathophysiology of Takotsubo Cardiomyopathy is not fully understood, and the impact of the hypoestrogenic state and the nutritional deficiencies on the development Takotsubo Cardiomyopathy in anorexia nervosa patients remains to be a myth.

Keywords: Anorexia Nervosa; Takostsubo Cardiomyopathy; Left Ventricular Thrombosis.

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Introduction

Takotsubo Cardiomyopathy (TTC), first described in 1991 in Japan, is a condition that closely resembles ACS in symptoms, laboratory values, and EKG findings and results in acute LV dysfunction, but yet differs in that it is without evidence of coronary artery stenosis on angiography and presents after intense emotional or physical stress [1-4]. Mayo clinic has proposed several criteria for the diagnosis of TTC: (1) transient hypokinesis, akinesis, or dyskinesis in the left ventricular mid segments with or without apical involvement; regional wall motion abnormalities that extend beyond a single epicardial vascular distribution; and frequently, but not always, a stressful trigger; (2) the absence of obstructive coronary disease or angiographic evidence of acute plaque rupture; (3) new ECG abnormalities (ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin; and (4) the absence of pheochromocytoma and myocarditis. A few case reports have described the association between TTC and anorexia nervosa, but the exact pathophysiology has never fully been elucidated. We present a rare case of TTC and left ventricular mural thrombus presenting in a young female with anorexia nervosa.

Case Presntation

A 34–year-old woman with past medical history of anorexia, asthma, and mixed connective tissue disease presented to our facility non-responsive and hypoglycemic (blood sugar of 49). She was found to have decreased respiratory rate, hypotension, and tachycardia and accordingly she was admitted for possible narcotic overdose. Naloxone and dextrose were administered to the patient on the scene. In the Emergency Department, she continued to be hypotensive (BP=84/58) with a decreased respiratory rate (RR=10-12). Another dose of Naloxone was administered and her pupils became mydriatic. Due to her continued altered mental status and failure to respond to non-invasive measures, she was intubated for airway protection and transferred to the Medical ICU.

On arrival to the ICU, her GCS was 3, she was hypotensive, and had a RR of 12. Her initial EKG showed ST depression with T wave inversion in the inferolateral leads, in addition to Q waves

in the anteroseptal leads (V1-V3). The first set of troponins was elevated at 1.2 ng/ml and trended to 1.94 ng/ml 4 hours later. Cardiology was consulted and limited bedside Echo was obtained which showed poor Ejection Fraction with global hypokinesis and possible cardiomyopathy. No regional wall motion abnormalities were seen. In the ICU, the patient had repeated episodes of hypotension and was given multiple fluid boluses to maintain Central Venous Pressure of >7. Chest X-Ray was done and it was suggestive of right-sided aspiration pneumonia. As such, empiric antibiotics were started after cultures were drawn. The first blood cultures were positive for Staphylococcus Lugdunensis (Coagulase negative Staph).

Midodrine was started for maintenance of blood pressure. A working diagnosis of Stress Induced Cardiomyopathy was made, but other possibilities including vitamin B1 deficiency (beri-beri) were considered. Lab values were significant for pancytopenia, which was attributed to her severe nutritional deficiency. As the patient was symptomatically improved, she was placed on a CPAP trial and later extubated. A standard 2-D Echocardiogram was done and showed an EF of 15-20% with global hypokinesis except for the basal segment which was hyperkinetic (Figure 1), a finding consistent with Stress Induced Cardiomyopathy (SCM), also known as Takotsubo Cardiomyopathy (TTC). This finding was confirmed later by a strain analysis of the echocardiographic images (Figure 3).

Four days later, patient was transferred to the medical floor. Her nutrition was optimized. Patient electrolytes were monitored on daily basis for the concern of refeeding syndrome. Electrolytes were repleted as needed. Nutrition service was consulted in an attempt to improve her calorie intake and her appetite. Since blood cultures were positive with known aspiration pneumonia, she was started on moxifloxacin and metronidazole for pneumonia and-vancomycin for possible endocarditis given the positive blood culture. A repeat TTE was done (7 days after the first TTE) to rule out endocarditis. The study failed to demonstrate vegetations, but was significant for 0.8 cm loosely organized left ventricular apical thrombus (Figure 2). Interestingly, the EF was improved to 25-30%. Given the LV thrombus, the patient was started on Warfarin. Enoxaparin was held as the patient was surprisingly thrombocytopenic (platelet 24,000/mm^3) despite the left ventricle mural thrombus.

Despite her improvement, the patient became dyspneic and the Chest X-Ray was significant for newly developed bilateral pleural effusions, which were confirmed via CT scan of the chest. Interestingly, repeat Brain Natriuretic Peptide (BNP) was very high (>5000) despite normal BNP on admission. Consequently, the patient was started on furosemide and albumin. Several days later, the patient was started on spironolactone and her total fluid balance continued to be negative.

Figure 1. 2-D Echocardiogram showing hyperkinetic basal segment of left ventricle (arrow) with akinesia/hypokinesia of the apical and lateral wall.

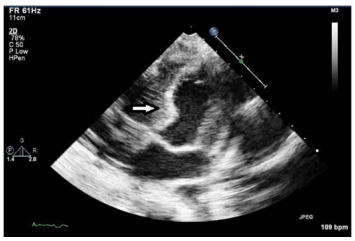


Figure 2. 2-D Echocardiogram showing LV apical thrombus (hyperechoic) occupying most of the left ventricle with mild dilation of left atrium.

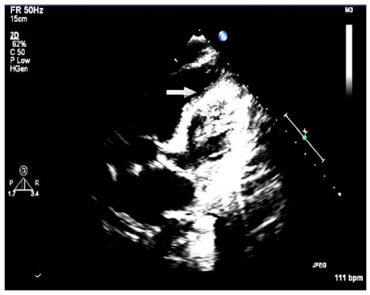
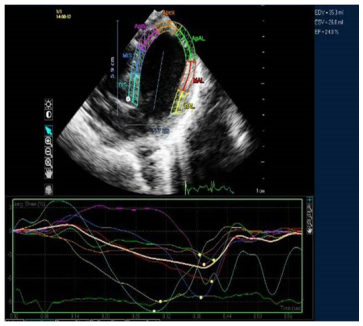


Figure 3. Echocardiographic Strain Analysis of the left ventricle illustrating the hyperkinetic basal segment (light blue color) and the akinetic /hypokinetic apical (green color) and lateral wall (yellow and red color).



A follow up TTE 8 days after the second TTE showed improved Ejection Fraction (EF) and wall motion with decreased size of LV apical thrombus (Figure 4a). One week later, another TTE was done and it showed normal EF with normal wall motion and absence of the LV thrombus (Figure 4b). In addition, the study also demonstrated a moderate sized pericardial effusion (Figure 5a). Repeat BNP was 1355 and management continued. The patient continued to improve and prior to discharge, a TTE was done which revealed normal EF and complete resolution of the pericardial effusion (Figure 5b). Another Strain Analysis of the Echocardiographic images was done to confirm resolution of Takotsubo Cardiomyopathy (Figure 6 a-b). Upon discharge, she was admitted to an eating disorder program and is doing very well now.

Discussion

TTC is a rare disorder that mimics the classic MI presentation and occurs in 1-2% of patients undergoing ACS work up [4]. Despite similar presentation, TTC has no angiographic evidence of vessel occlusion. However, echocardiographic findings reveal apical dyskinesia/akinesia and basal hyperkinesia and cardiac enzyme elevation [1-5]. TTC typically affects postmenopausal women and is triggered by intense emotional or physical stressors, including non-cardiac surgery, pheochromocytoma, thyrotoxicosis, trauma, serious illness, and catecholamine administration [2]. The definite pathophysiology of TTC has not been fully elucidated, but it is most commonly thought to be due to a large scale catecholamine release during times of stress, resulting in damage to the myocardium. Toxic levels of catecholamines are hypothesized to be responsible for the pathognomonic apical ballooning found on echocardiographic analysis [4].

Life threatening cardiovascular complications are seen in up to one-third of all deaths in patients with anorexia nervosa, including bradycardia, hypotension, arrhythmias, repolarization abnormalities, Takotsubo cardiomyopathy, and sudden death [1]. Very few cases of anorexia nervosa associated TTC have been described

Figure 4. (a) (left) 2-D Echocardiogram (4 chamber view/apical view) showing hyperkinetic basal segment of left ventricle (arrow) with some improvement in left ventricular thrombus (arrowhead) two weeks after admission. (b) (right) 2-D Echocardiogram (4 chamber view/apical view) demonstrating improvement in the basal segment (arrow) with absence of LV thrombus (3 weeks after admission).

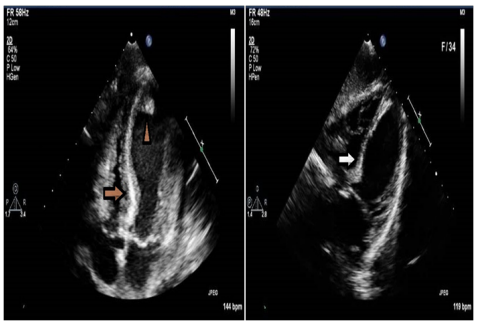


Figure 5. (a) (left) 2-D Echocardiogram (long axis view) demonstrating pericardial and left pleural effusions with pericardial membrane in-between (red arrow). (b) (right) 2-D Echocardiogram (long axis view) demonstrating very minimal pericardial effusion (arrow) and complete resolution of the pleural effusion 4 weeks after admission.

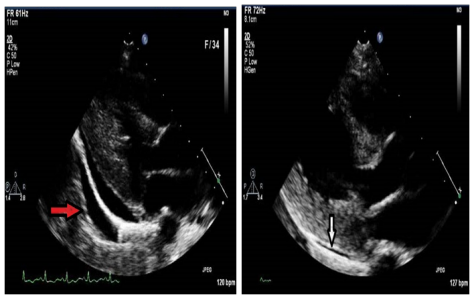
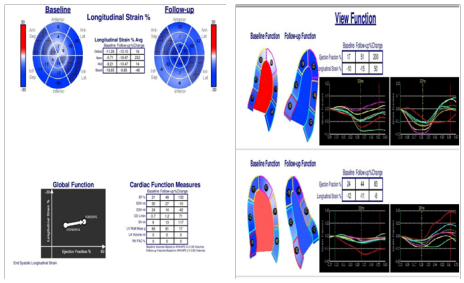


Figure 6. (a&b)Echocardiographic Longitudinal Strain Analysis of the left ventricle comparing the baseline (initial) echocardiogram and the follow up echocardiogram.The difference in wall motion and measurements clearly illustrates normal homogenous wall motion of all parts of the left ventricular wall on the follow up, and the complete resolution of Takotsubo Cardiomyopathy (4 weeks after admission).



to date. In the majority of TTC cases, the most frequent clinical symptoms were dyspnea and angina, resembling acute myocardial infarction [4]. Our case was unique and challenging in the very unusual presentation, the concurrent left ventricular mural thrombus, development of pericardial effusion, and the quick resolution of the abnormalities. The patient's severe malnourishment and hypoglycemia likely induced an elevation in circulating catecholamine levels, resulting in myocardial injury.

A more interesting theory is that reduced estrogen levels seen in both post-menopausal women and patients suffering from anorexia nervosa may predispose patients to TTC [2]. The change in endothelial function and vasomotor reactivity in response to catecholamines may possibly make these patients more vulnerable to catecholamine-induced myocardial stunning [2, 3, 7]. Consequently, the threshold for developing TTC is lower in patients with anorexia nervosa due to hypoestrogenemia (low levels of circulating estrogen). Moreover, we believe that the decrease in the muscle mass in anorexic patients, including the cardiac muscle mass, might increase the susceptibility of the left ventricular wall to shear stress forces produced by catecholamine surges, which will eventually result in apical ballooning and compensatory basal hypercontractility.

According to Haghi et al. [8], 8% of TTC patients had LV mural thrombi. However, anorexic patients who have TTC with mural thrombosis have not ever been reported in United States to the best of our knowledge.

Conclusion

In conclusion, the pathophysiology of TTC in patients with anorexia nervosa is multi-factorial, and physicians should be aware about the vulnerability of these patients to develop TTC and even LV thrombus in rare cases, such as our case. We believe that more research is needed to help understanding the hormonal and nutritional influences in TTC development.

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