

Takotsubo Cardiomyopathy in Anorexia Nervosa Patients: Why Is It Happening?

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Dear Editor,

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 and it includes the absence of obstructive coronary disease or angiographic evidence of acute plaque rupture as an essential component in the definition. 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 34-year-old woman with past medical history of anorexia nervosa 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. Due to her continued altered mental status she was intubated for airway protection and transferred to the Medical ICU.

On arrival to the ICU, 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

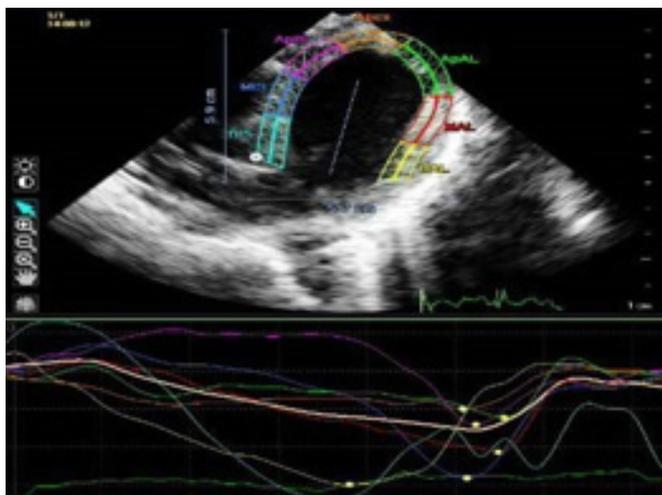
troponins was elevated at 1.2 ng/ml and trended to 1.94 ng/ml 4 hours later. Cardiology was consulted and limited bedside Echo was obtained which showed poor Ejection Fraction with global hypokinesis and possible cardiomyopathy. No regional wall motion abnormalities were seen. Midodrine was started for 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, standard 2-D Echocardiogram was done and showed an EF of 15-20% with global hypokinesis except for the basal segment which was hyperkinetic, 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 1).

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. 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. 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/mm³) 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.

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. One week later, another TTE was done and it showed normal EF with normal wall motion and absence of the LV thrombus. In addition, the study also demonstrated a moderate sized pericardial effusion. The patient continued to improve and prior to discharge, a TTE was done which revealed normal EF and complete resolution of the pericardial effusion. Another Strain Analysis of the Echocardiographic

Figure 1:





images was done to confirm resolution of Takotsubo Cardiomyopathy (figure 2). Upon discharge, she was admitted to an eating disorder program and is doing very well now.

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).

Very few cases of anorexia nervosa associated TTC have been described 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). 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.

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.

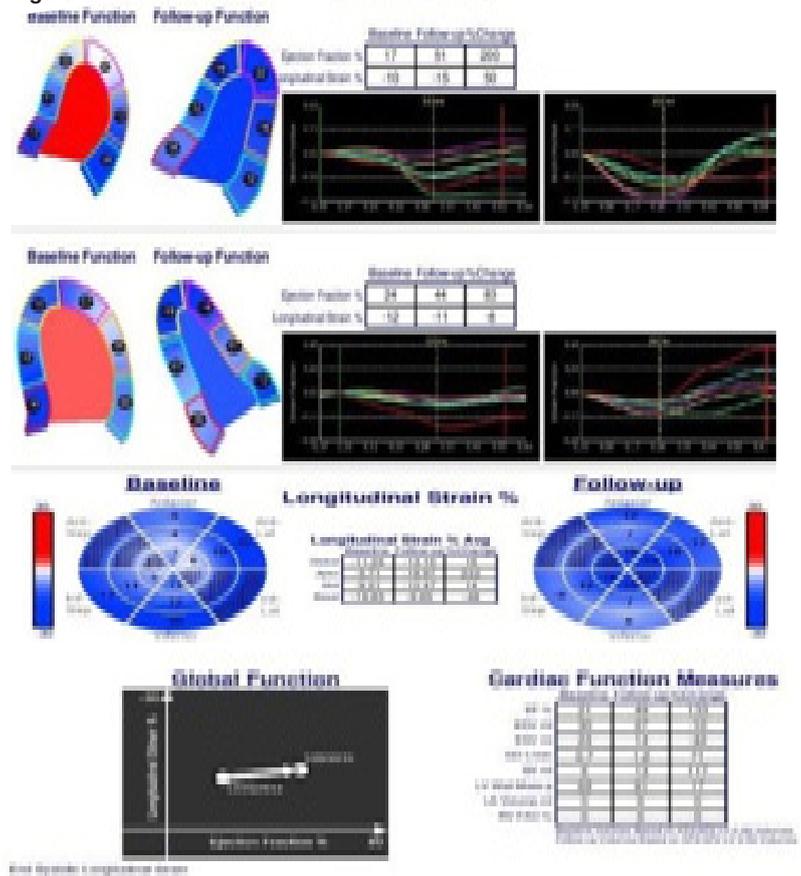
Statement of ethical publishing

The authors state that they abide by the statement of ethical publishing of the International Cardiovascular Forum Journal⁷.

Conflict of interest:

Neither I, nor any of the co-authors has any financial, non-financial, or commercial of interest to disclose. (We have no conflict of interest to disclose).

Figure 2:



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References

1. Kyung-Hee Kim, MD, Ho-Joong Youn, MD, Wook-Hyun Lee, MD, Jin-Suk Kim, MD, Jae-Gyung Kim, MD, Ha-Wook Park, MD, Jinsoo Min, MD, Gee-Hee Kim, MD, and Hae-Ok Jung, MD. A Case of Anorexia Nervosa Complicated With Strongly Suspected Stress-Induced Cardiomyopathy and Mural Thrombus. *Korean Circ J* 2011; 41: 615-617. / doi: 10.4070/kcj.2011.41.10.615
2. M.N.M. Volman, R.W. ten Kate, and R. Tukkie. Tako Tsubo cardiomyopathy, presenting with cardiogenic shock in a 24-year-old patient with anorexia nervosa. *The Journal of Medicine* 2011; 69: 129-131. Doi:10.1155/2015/292658
3. Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. *Am Heart J*. 2008;155:408 – 417. Doi: 10.1016/j.ahj.2007.11.008.
4. Yoshihiro J. Akashi, MD, PhD; David S. Goldstein, MD, PhD; Giuseppe Barbaro, MD; Takashi Ueyama, MD, PhD. Takotsubo Cardiomyopathy A New Form of Acute, Reversible Heart Failure. *Circulation*. 2008;118:2754-276. Doi: 10.1161/CIRCULATIONAHA.108.767012
5. Rina OHWADA, Mari HOTTA*, Hironari KIMURA, Sachiko TAKAGI, Naoki MATSUDA**, Kaoru NOMURA and Kazue TAKANO. Ampulla Cardiomyopathy after Hypoglycemia in Three Young Female Patients with Anorexia Nervosa. *Internal Medicine*. 2005; 44: 228-233. Doi: 10.2169/internalmedicine.44.228
6. Ana María Castillo Rivera, Manuel Ruiz-Bailén, Luis Rucabado Aguilar. Takotsubo cardiomyopathy – a clinical review. *Med Sci Monit*, 2011; 17(6): RA135-147. Doi: 10.12659/MSM.881800
7. Shewan LG, Coats AJS, Henein M. Requirements for ethical publishing in biomedical journals. *International Cardiovascular Forum Journal* 2015;2:2 DOI: 10.17987/icfj.v2i1.4.