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Case Report: The Heart Breaking Tale of Takotsubo in the ED

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Case Report: The Heart Breaking Tale of Takotsubo in the ED

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Introduction:

Chest pain is one of the most common chief complaints that emergency room physicians deal with on a daily basis. Here we present a rare cause of chest pain on a patient diagnosed with takotsubo cardiomyopathy. This is a condition that has been on the rise since its initial discovery and therefore clinicians should consider this on their differential when assessing patients with chest pain, especially post menopausal women.¹

Case Presentation:

A 86-year-old Caucasian woman presented to the emergency department (ED) with a chief complaint of left-sided chest pain and headache that had been ongoing for about one week. She denied any shortness of breath, abdominal pain, fever and or chills. The pain began after the patient had a physical altercation with a neighbor who struck her in the chest and abdomen. Patient did note a past medical history of adrenal neoplasm. She denied alcohol, tobacco, and drug use. Her surgical history was non-contributory. Patient was seen in the emergency department after the initial altercation event, imaging at that time was negative for any bony abnormalities, echocardiogram (ECG) was non-ischemic in nature; however lab work did reveal a high sensitivity troponin to be >23,000.0 ng/L. Cardiology was consulted, heparin was recommended, and to transfer patient for cardiac catheterization to rule out infarct vs cardiac contusion. The patient left against medical advice (AMA) without any treatment; however, she did return a few days later with the same complaints.

At her second visit vitals were as follows: Blood pressure 184/88 mmHg, Heart Rate 88, Respiratory rate 15 breaths per minute, and temperature of 98.9 degrees F. Physical exam was positive for tenderness to palpation of left lower anterolateral ribs with an overlying contusion. Patient's troponin was 950.0 ng/L, creatinine 0.74, BNP and CK was not obtained. The remainder of her lab work was within normal limits. Patient's ECG did show ST elevations in the anterolateral leads with reciprocal changes in inferior leads. [Figure 1]. Patient was receptive to treatment this time, as the workup was more consistent with possible infarct vs cardiac contusion, aspirin, nitroglycerin and heparin were administered. She was then transferred to a secondary facility where she underwent cardiac catheterization which revealed nonobstructive coronary artery disease, an ejection fraction of 30%, moderate to severe left ventricular systolic dysfunction, and non-ischemic cardiomyopathy consistent with Takotsubo cardiomyopathy. In addition to this transthoracic echo was performed that showed left ventricular chamber size at the upper limits of normal with mild-to-moderately decreased left ventricular systolic function, with the distal 3rd of the left ventricle akinetic to dyskinetic which appeared ballooned and dilated, which is consistent with takotsubo cardiomyopathy versus a mid LAD occlusion. She was then admitted and was started on Lasix, Losartan, and Metoprolol. Patient was discharged home two days after admission in stable condition with follow-up with cardiology. Discharge medications included lasix, losartan, metoprolol, and aspirin.

Discussion:

Pathophysiology:

Takotsubo is a type of cardiomyopathy that is defined as dilation and impaired contraction of the left and or right ventricle that is caused by emotional or physical stress. This condition does have a characteristic finding of apical ballooning seen on left ventriculography or echocardiography.¹ The actual pathogenesis of takotsubo to this day is not very well understood. There are currently three major hypotheses. The first, most widely accepted theory, is catecholamine-induced cardiotoxicity. Due to an increase in emotional and physical stress the body releases increased levels of catecholamines via activation of the sympathetic nervous system. It has been noted that patients have had two to three times the normal amount of serum catecholamines which can lead to myocyte injury.⁵ This was supported by an initial endomyocardial biopsy that showed evidence of disorganized cytoskeletal and contractile structures, contraction bands, and increased extracellular matrix proteins that were no longer present after a subsequent biopsy was performed after functional recovery.^{3,6} The second theory is microvascular dysfunction in the form of impaired endothelium-dependent vasodilation, excessive vasoconstriction, and abnormal myocardial perfusion. These conditions have been noted to be seen in patients with estrogen deficiency, which supports why postmenopausal women are disproportionately affected.^{3,5,6} The last theory encompasses a patient's genetic predisposition. There is limited data, but there have been documented reports of familial cases.³ In addition to this, certain conditions have been thought to be linked to takotsubo such as: psychiatric/neurological disorders due to their effects on the cognitive centers of the brain and hypothalamic-pituitary-adrenal axis and autoimmune disorders such as Sjogren's and systemic lupus due to the coexisting nature of myocarditis and pericarditis with these types of conditions.^{5,6}

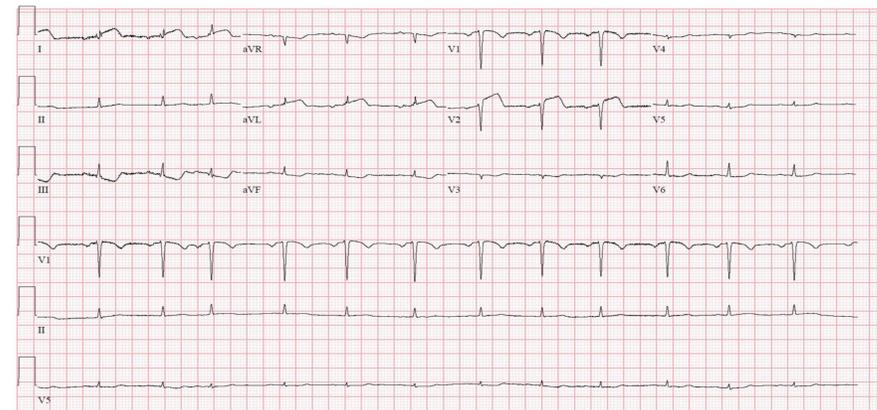


Figure 1. EKG showing ST elevations in a patient diagnosed with Takotsubo Cardiomyopathy

Discussion Continued:

Incidence:

This condition was first seen in 1990 in Japan and now accounts for approximately 1-2% of patients presenting with suspected ST-Elevation Myocardial Infarctions (MI) or troponin elevated Acute Coronary Syndromes (ACS). This condition affects postmenopausal women disproportionately with multiple studies citing over 80% of confirmed cases being found in women with a mean age of about 66 years old.^{1,3,4}

Clinical presentation:

Patients will present similar to those that are suffering from an acute ACS primarily complaining of substernal chest pain, chest palpitations, dyspnea, and syncope; however further questioning should reveal symptoms were triggered by an event of extreme emotional life events and or physical stressors such as accidents and or major traumas. In addition to these symptoms, patients may also present with physical exam findings consistent with acute heart failure (HF), mitral regurgitation (late-peaking systolic murmur), sudden cardiac arrest, cardiogenic shock (hypotension, oliguria, cold extremities, or an abnormal mental status), and stroke secondary to embolization of an apical thrombus.^{3,5,6}

Diagnosis:

The most widely accepted way to diagnose takotsubo uses Mayo's Clinics Diagnostic criteria that requires all four to be present in the patient:^{3,6}

1. Transient hypokinesis, akinesis, or dyskinesis in the left ventricular mid segments with or without apical involvement; regional wall motion abnormalities that extend beyond.
2. A single epicardial vascular distribution; and frequently, but not always, a stressful trigger.
3. The absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.
4. New ECG abnormalities (ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin.
5. The absence of pheochromocytoma and myocarditis.^{3,6}



Figure 2. Dilated Left ventricle on ventriculography

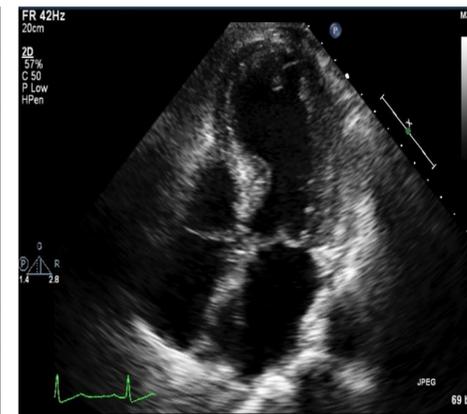


Figure 3. Apical Stress Cardiomyopathy on echo

Discussion:

Diagnosis Continued:

Additional imaging such as cardiac catheterization, transthoracic echocardiography (TEE), cardiovascular magnetic resonance imaging (MRI) can also be used to help narrow down the differential diagnosis which can lead you to the diagnosis of takotsubo. Cardiac catheterization will show normal or mild to moderate coronary atherosclerosis as well as left ventricular apical ballooning. TEE can also show apical ballooning and ventricular wall abnormalities. Cardiovascular MRI is more used to rule in/out other differentials by identifying presence or absence of myocardial edema, necrosis, and fibrosis.^{3,5,6}

Management:

Takotsubo resolves by itself in most cases without any need for immediate interventions. Symptom management is the mainstay for treatment. It is recommended that the inciting stressor be resolved for a more rapid resolution of symptoms. However, due to the fact that these patients typically present as ACS initial treatment should follow the ACS algorithm by providing aspirin (ASA), beta-blockers, ACE inhibitor, lipid lowering agents. It should be noted that these patients can develop acute heart failure, shock or thromboembolism so an appropriate workup and physical exam should be performed to rule these conditions in/out. Heart failure management should be followed according to standard guidelines. If thromboembolism is present, anticoagulation using warfarin is recommended.^{2,4,5,6}

Prognosis:

Most patients tend to recover their systolic ventricular function within one to four weeks after onset of symptoms. Those who have been diagnosed face about a 2% per year risk of recurrence with most occurring 3 weeks to 3.8 years after the first event. Due to the increased risk of severe in-hospital complications associated with takotsubo (acute heart failure, cardiogenic shock, stroke, tamponade, vascular access complications, and ventricular and papillary muscle rupture) in-hospital mortality is around 3-4% with men having a worse prognosis due to them having a higher prevalence of acute critical illness with elevated catecholamines. Long term treatment can include antithrombotics and HF therapy until the patient regains most if not all cardiac function.^{2,3,4,5,6}

Conclusion:

Takotsubo is a generally benign and self-limited diagnosis, however due to the fact that these patients present to the ED with ST elevations, initial interventions must be directed towards ACS treatment algorithms until cardiac catheterization has been performed ruling out coronary obstruction or rupture. Once the diagnosis of takotsubo is made patient's should be started on a form of antithrombotic and HF regimen until cleared by cardiology.

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