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CLINICAL VIGNETTE

A Case of Takotsubo Cardiomyopathy

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Case Presentation

A 61-year-old male with history of hypertension and schizophrenia presented to his primary care clinic for follow-up of his ST segment elevation myocardial infarction (STEMI), which had occurred 4 days prior. A concerning EKG prompted referral to the Emergency Department at an outside hospital. He lives in a board and care facility and had very little recollection of the details of his treatment at the outside hospital.

Review of records revealed that patient had presented with shortness of breath, pre-syncope, and found to have ST elevation in multiple precordial leads along with elevated troponin of 26.0 ng/dL. Patient underwent cardiac catheterization and was found to have non-obstructive coronary artery disease. He was diagnosed as having Takotsubo Cardiomyopathy after echocardiography revealed a left ventricular ballooning with presence of hypokinesis and no evidence of thrombi. His ejection fraction (EF) was 40%. He was discharged on Amlodipine, Aspirin, and Metoprolol along with his maintenance psychiatric medications.

Upon arrival at the Emergency Room, he was free of chest pain, shortness of breath, or pre-syncope. His social history was notable for a 40-year pack history of smoking with current 1-2 cigarettes daily. He was afebrile with blood pressure of 130/92 mmHg, heart rate of 107, and respiratory rate of 18. His oxygen saturation was 95% on room air. Patient's exam was notable for a normal cardiovascular exam with no signs of murmurs or extra heart sounds.

Laboratory studies revealed a sodium of 128 mmol/L and a potassium of 5.6 mmol/L. His INR was 1.1 with a PT of 12.2 and PTT 33.9. His hemoglobin was 19.2 g/dL with a hematocrit 55.4%. Troponin was 5.5 ng/mL. EKG revealed ST elevation in leads V1-V4 and leads II, III, and AVF, which was unchanged from outside hospital EKG. Beside echocardiography indicated the presence of an LV thrombus, which was not indicated on echocardiography at the outside hospital. Formal transthoracic echo (TTE) was then performed, which found overall EF of 40-45%, impaired diastolic function, and akinesis of apex and the distal third of the left ventricle with an 1.3x 2.5cm apical thrombus, consistent with Takotsubo Cardiomyopathy. Patient was admitted for monitoring and initiation of anticoagulation with heparin with transition to

warfarin. Patient was bridged after two days and was discharged.

Discussion

Takotsubo Cardiomyopathy, also known as “broke heart disease,” is a confusing disease process that presents as an acute STEMI with no notable coronary vessel obstruction on cardiac catheterization but with abnormal shaping of the ventricles on echocardiography.¹ Aptly named because of the apical ballooning resembles a Japanese takotsubo pot used to catch octopus. These patients have a true decline in myocardial function. Troponins are usually mildly elevated with variable EKG changes, ranging from ST segment elevation (typically in precordial leads) to nonspecific abnormalities. Interestingly, it has also been associated with QT prolongation while ST depression is less common.² Patients typically present with an impressive EKG and elevated cardiac enzymes similar to the presentation of an acute coronary syndrome. A carefully obtained history often elicits a preceding episode of severe emotional or physical stress. It has been estimated that 70-85% of cases have a specific recognizable physical or emotional stressor within minutes to an hour prior to presentation. However, 15-28% of cases have no identifiable stressors.^{3,4}

Takotsubo Cardiomyopathy has recently been increasingly reported in the literature with increasing recognition in the last 15 years. What causes the broken heart? The precise cause is not known; several theories have been proposed including coronary artery spasm, microvascular dysfunction, neurogenic stunned myocardium, acute coronary syndrome with reperfusion injury, impaired fatty acid metabolism, and excess catecholamine burden to the myocardium causing damage.^{1,5} The last hypothesis is supported by patients with pheochromocytoma who present with a Takotsubo-like disease process. Of interest, demographically, Takotsubo predominately occurs in females with some studies reporting a female prevalence as high as 90%. The majority of the females are post-menopausal.⁴ Takotsubo Cardiomyopathy also has an association with previous chronic psychiatric or neurological disorders.⁴

Treatment of Takotsubo is supportive since there are no identifiable culprit lesions on cardiac catheterization and can include beta blockers, ACE inhibitors, diuretics, and

anticoagulants.³ Beta blockers have been used to impact the catecholamine surge but have not been associated with improvement of mortality.⁴ In severe cases, patients may require hemodynamic support such as intra-aortic balloon pump.⁴

The first incidence of Takotsubo Cardiomyopathy associated ventricular thrombus was reported around a decade ago.⁶ Subsequently, another reported that these ventricular thrombi could have serious cardio-embolic consequences leading to cerebral infarction.⁷ It is controversial whether these thrombi should be managed medically or surgically.⁸⁻¹⁰ Medical management with anticoagulation therapy is suggested for at least 3 weeks or until wall abnormalities improve.¹¹ However, further outcome studies are lacking to confirm whether anticoagulation should be continued past the wall abnormality period in the first 3 weeks.¹² Risks factors to anticoagulation include further aggravated bleeding during ventricular rupture, a rare but a potential severe consequence of Takotsubo pathology.

Templin et al.⁴ in the only long term study on Takotsubo Cardiomyopathy found a 1.2% thrombus rate and a 0.2% rate of ventricular rupture. There was an overall death rate of 5.6% per year and risk of cerebrovascular event of 9.9% per year.

The natural course of Takotsubo is variable. Traditionally, it was thought that Takotsubo cardiomyopathy was reversible once recognized and treated appropriately with return of ventricular function over several days to weeks and in-hospital mortality was as low as 1-2%.² However, a recent *New England Journal of Medicine* study noted that 21.8% of patients had serious in-hospital complications including death, arrhythmia, ventricular thrombus, and ventricular rupture. Additionally, 30 day rate of major events such as cardiac and cerebrovascular events were as high as 7.1%. In-hospital mortality occurred predominantly in men and was noted to be approximately 4%.⁴ Despite gradual return of function, chance of re-occurrence is approximately 5%.³

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