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Stress Induced (Takotsubo) Cardiomyopathy Triggered by Intubation

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INTRODUCTION/ABSTRACT

Stress induced cardiomyopathy, also known as Takotsubo cardiomyopathy, is a reversible cardiomyopathy that can impersonate a myocardial infarction or acute heart failure in those with severe emotional or physical stress.

Keywords: Stress induced cardiomyopathy; Takotsubo cardiomyopathy; Cardiomyopathy

CASE PRESENTATION

A 50-year-old Hispanic male with a past medical history of gastritis, sleep apnea, and recently diagnosed esophageal adenocarcinoma in July 2022 was scheduled for an esophagectomy, pyloroplasty, open J tube placement, and lymph node dissection on 12/21/2022. Postoperatively, the patient was transferred to the trauma intensive care unit (TICU) with continuous monitoring and mechanical ventilation. Once the patient maintained stable hemodynamics off pressors he was extubated to nasal cannula 12/23 (POD#2)

Early in the morning on December 26th (POD #5), the patient became febrile to 103 degrees Fahrenheit and tachycardic (120s-150s bpm). Sepsis pathway was initiated. Labs sent at that time were all within normal limits except for a mildly elevated troponin (20 ng/L, normal 1-19). A CT scan of the chest with IV and PO contrast confirmed a right sided esophageal leak with a large left pleural effusion, small right pleural effusion, and scattered atelectasis. Esophageal stent placement at the bedside in the ICU was planned for which the patient needed to be re-intubated.

The patient was induced and paralyzed for rapid sequence intubation with Etomidate 20 mg and Succinylcholine 100mg, respectively. A glidescope was used because of the patient's small mouth opening. Placement was confirmed via positive end tidal carbon dioxide, positive bilateral breath sounds, and absence of abdominal breath sounds. Immediately following intubation, the patient's blood pressure transitioned from hypertensive at 162/77 mmHg to hypotensive at 73/46 mmHg. ST segment changes were noted on telemetry, and a 12-lead EKG illustrated

sinus tachycardia with ST segment elevations in the inferior and lateral leads (Image 1). The patient was loaded with Aspirin 325 mg, Atorvastatin 40mg daily was started, and Levophed was resumed. Bedside point of care ultrasound showed mid to apical septal and mid inferoseptal- inferoapical hypokinesis of the heart; new findings compared to the transthoracic echocardiogram performed on 12/22/2022. Cardiology was consulted, and the catheterization lab was activated. Left heart catheterization was performed through the right common femoral artery which demonstrated minimal luminal irregularities of the left circumflex and right coronary arteries, normal angiography of the left main and left anterior descending arteries, normal coronary angiography, left ventricular end diastolic pressure (LVEDP) of 20mmHg, and left ventricular ejection fraction (LVEF) of 35% with apical hypokinesis and a hyperdynamic base consistent with Takotsubo cardiomyopathy (Image 2). Repeat labs were significant for elevated troponins (433 ng/L and 458 ng/L), no leukocytosis (5 x10^9/L), stable hemoglobin (8.7 g/dL), and elevated lactate (2.7 mmol/L).

Ultimately, the patient underwent esophageal stent placement in the operating room that evening. Intra-operatively he required both Levophed and Vasopressin for hemodynamic support.

Postoperatively he remained intubated and on Levophed, and he returned to the TICU.

The following day on December 27th, EKG showed sinus tachycardia with low voltage QRS, nonspecific T wave abnormality, and ST waves were no longer elevated. Additionally, formal transthoracic echocardiogram (TTE) showed hypokinetic motion of the apex and mid to apical cap of left ventricular segments with mild mitral regurgitation and LVEF estimated at 45-49% (Image 4). The patient was weaned off pressors on December 29th. TTE was repeated on January 5th, 2023, which demonstrated recovery to normal left ventricular function with LVEF estimated to be greater than 70%. He continues to take Aspirin 81 mg daily and metoprolol tartrate 25 mg twice daily maintaining normotension and heart rate 80-100 bpm.

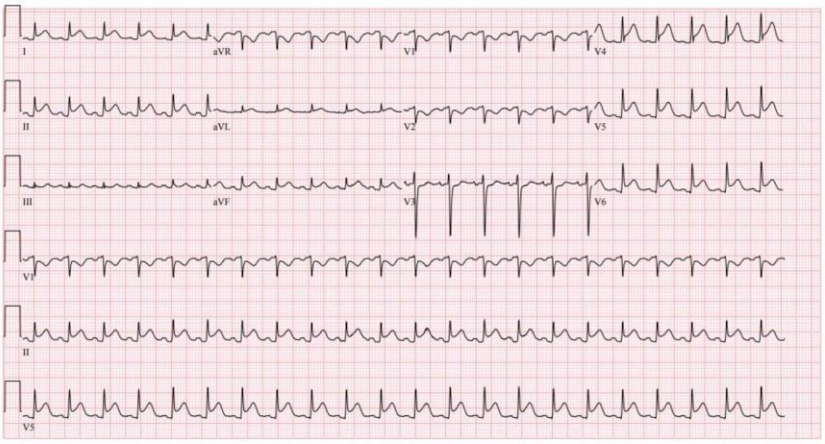

Image 1: EKG following re-intubation, recorded 12/26/2022 at 12:00:31

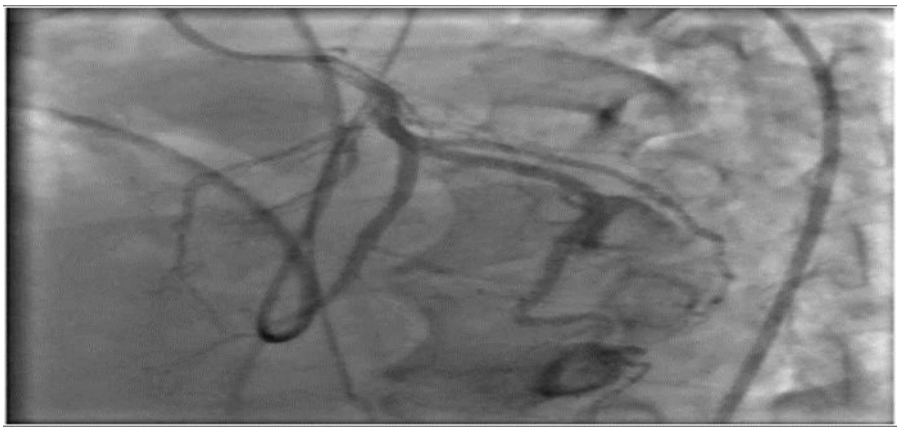

Image 2: Left heart catheterization showing normal coronary arteries.

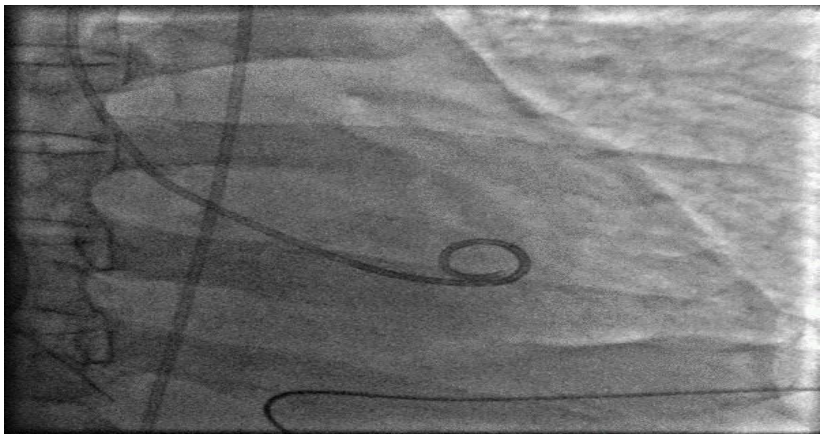

Image 3: Left heart catheterization showing apical hypokinesis.

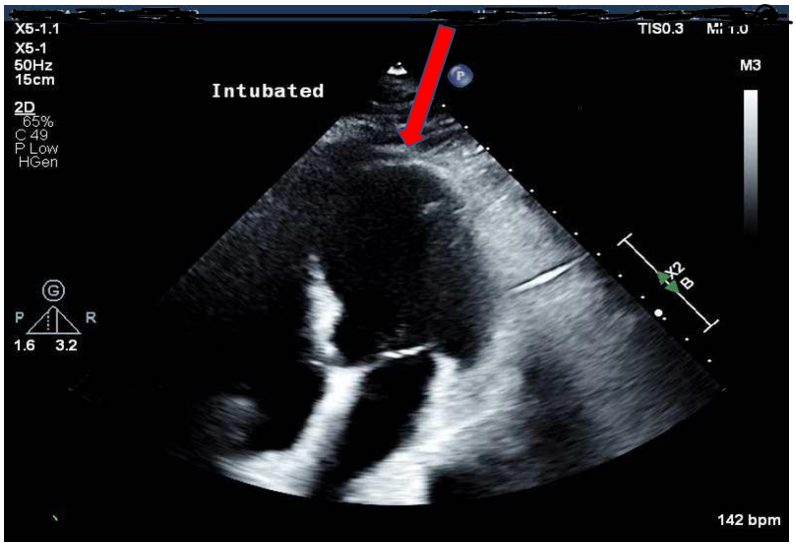

Image 4: Transthoracic echocardiogram showing ballooning of the Left Ventricle.

DISCUSSION

Stress induced cardiomyopathy, also known as Takotsubo cardiomyopathy, is a reversible cardiomyopathy that can impersonate a myocardial infarction or acute heart failure in those with severe emotional or physical stress. This syndrome is more prevalent in women (85-90% of cases) and those aged 65-70.[1] This cardiomyopathy has been seen in patients of various races with Hispanic being one of the least common.[2] Of the men who have been diagnosed with this condition, there is a high correlation with severe physical stress.[3] Additionally, those with cancer are at heightened risk for developing stress induced cardiomyopathy as a cancer diagnosis may trigger a great deal of both emotional and physical stress.[4]

The exact pathophysiology and pathogenesis of stress induced cardiomyopathy is unknown, however, there are multiple proposed mechanisms. The most esteemed theory is that of catecholamine induced myocardial injury. Excess catecholamines after a stressful event can cause increased contractility, tachycardia, and vasoconstriction which can lead to microvascular dysfunction and macrovascular spasms. This can result in the disruption in the normal function of the myocardium.[5] Signs of a heart attack such as decreased ejection fraction, increased cardiac enzymes, and ischemic changes on EKG can be seen however, there are no abnormalities on coronary angiography.[5]

Our patient fit the criteria for stress induced cardiomyopathy as he had a decreased ejection fraction, elevated troponins, EKG ST elevations, and echocardiography illustrating apical hypokinesis without angiographic abnormalities all in the setting of an acute stressful event, reintubation. Interestingly, he is not the sex, age, or race typically seen amongst patients with Takotsubo cardiomyopathy. Having recently been diagnosed with cancer may have increased this patient's risk for developing Takotsubo cardiomyopathy due to the emotional and physical stressors that come with this diagnosis. Additionally, the recent esophagectomy complicated by esophageal perforation led to further physical stress and cardiac irritation. Once the diagnosis has been made, treatment can be initiated to maintain hemodynamic stability in the acute setting and prevent cardiac remodeling long-term. Angiotensin-Converting Enzyme Inhibitors (ACEi) or Angiotensin Receptor Blocker (ARB) pharmacologic management is found to be associated with improved survival after the acute event if blood pressure allows.[1] Echocardiography can be done weeks to months later to make sure proper function of the heart is present.[1]

Proper management and interdisciplinary collaboration allowed this patient to undergo esophageal stent placement following his acute stress induced cardiomyopathy episode.

CONCLUSION

Takotsubo Cardiomyopathy can be seen in a wide variety of patients and is not confined to a certain sex, age, or race. Knowledge of patients' physical and emotional stressors, awareness of presentation consistent with Takotsubo Cardiomyopathy, comprehension of lab and imaging findings demonstrated by Takotsubo Cardiomyopathy, and interdisciplinary collaboration are essential for proper diagnosis and effective management of this condition.

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