

## Case Report

# Case of Recurrent Takotsubo Cardiomyopathy

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**Received:** 12 June 2020; **Accepted:** 22 June 2020; **Published:** 24 June 2020

**Citation:** Abu B. Choudhary, Adnan S. Raza, Stephen J. Peterson, Rahul Yadav, Shahzad Saleem, Salman Haq. Case of Recurrent Takotsubo Cardiomyopathy. Cardiology and Cardiovascular Medicine 4 (2020): 239-243.

### Abstract

**Background:** Takotsubo cardiomyopathy or stress induced cardiomyopathy is a transient regional systolic dysfunction of the left ventricle. It often mimics acute coronary syndrome (ACS) that is reversible and in the absence of angiographically obstructive coronary artery disease (CAD). Cases of recurrent takotsubo cardiomyopathy are not common. One study analyzed 749 patients with takotsubo cardiomyopathy from the multicenter registry, found that recurrence was about 4% and most recurrences occurred in the first 5 years.

**Case:** 68 year old female with history of takotsubo cardiomyopathy presented to the hospital for abdominal pain, nausea, vomiting with symptoms starting after taking one of nitrofurantoin. On initial evaluation she was also endorsing worsening dyspnea and labs were concerning for elevated troponins. Echocardiogram revealed reduced ejection fraction and mid-apical walls akinesis. She underwent cardiac catheterization which showed non-obstructive cardiomyopathy making recurrent takotsubo the likely diagnosis.

**Conclusions:** Takotsubo cardiomyopathy is an acute transient reduction in systolic cardiac function that is

induced by emotional or physical stressors. It is a non-obstructive cardiomyopathy that resolves after the initial stressor is resolved. Actual pathogenesis is still unclear however there are hypotheses it involves excessive catecholamine release. Recurrence is rare however it can occur and usually occurs in postmenopausal women with certain risk factors.

**Keywords:** Takotsubo Cardiomyopathy; Coronary artery disease; Acute coronary syndrome

## Background

Takotsubo cardiomyopathy or stress induced cardiomyopathy is a transient regional systolic dysfunction of the left ventricle. It often mimics acute coronary syndrome (ACS) that is reversible and in the absence of angiographically obstructive coronary artery disease (CAD). The term regional refers to wall motion abnormality that extends beyond the territory of a single coronary artery. Cases have been reported with apical and midventricular patterns of wall motion abnormality.

Takotsubo cardiomyopathy was first described in 1990 in Japan. A registry of 3265 patients with troponin elevated ACS or ST elevation infarction, stress induced cardiomyopathy was diagnosed in about 1.2 percent of patients with troponin positive ACS. About 60% of those patients had a typical apical wall motion abnormality [1]. In another study 92 patients were evaluated in medical ICU and in 26 patients there was reported left ventricular apical ballooning with mean ejection fraction (EF) of 33%. Of these, 20 of 26 patients had normalization of EF within 7 days [2]. According to the international takotsubo registry, stress cardiomyopathy is more commonly seen in

females than males with 1750 patients with takotsubo 89% were women with a mean age of 66. It was reported that emotional triggers were less common than physical with 36% had a physical trigger (acute respiratory failure, post surgery, infections) while 28% had an emotional trigger (grief/loss, panic, anxiety) and 29% had no evident trigger [3].

The most common presenting symptoms are acute substernal chest pain 76%, dyspnea 47%, and syncope 7.7%, according to the international takotsubo registry [3]. Physical exam findings are typically significant for tachycardic, respiratory distress, crackles, hypotensive, and jugular venous distension. The most common EKG findings are ST segment elevations at the anterior precordial leads 46% [4]. Typically serum cardiac troponins, brain natriuretic peptide are elevated on admission and creatinine kinase may be normal or slightly elevated.

Non-invasive diagnostic testing is done with echocardiography. However patients usually present with features consistent with acute myocardial infarction with chest pain, electrocardiogram changes, and elevated biomarkers requiring urgent cardiac catheterization. Angiographic data reported patients either had no CAD or non-obstructive epicardial disease greater than 50% stenosis [4]. EF was reduced during the initial presentation that improved significantly over a period of days to weeks [4]. Management is typically conservative as it is a transient disorder and treatment is generally focused on treating underlying disease such as sepsis and emotional/physical triggers.

## Case

68 year old female with a past medical history of hypertension, hyperlipidemia, diabetes mellitus and history of takotsubo cardiomyopathy presented to the emergency department with one day history of abdominal pain, nausea and vomiting. Her nausea and vomiting started after she took one dose of nitrofurantoin. Since that time she had multiple episodes that soon followed with abdominal pain and shortness of breath. The abdominal pain is localized to the left upper quadrant area associated with worsening shortness of breath. Patient had a similar presentation in 2012 at Mount Sinai Hospital, where cardiac catheterization, which showed non-obstructive cardiomyopathy with an EF of 10% that resolved on subsequent echocardiography. In 2017 outpatient echocardiogram reported EF of 66%, with no regional wall motion abnormality.

On admission she was normotensive, tachycardic to 102 and EKG showed RBBB (chronic) and new T wave inversions in V1. She was comfortable, breathing ambient air, speaking in full sentences. Normal cardiopulmonary exam with RRR, normal S1/S2 with no murmurs, rubs or gallops and lungs were clear to auscultation. Blood work showed hyperglycemia 311 mg/dL, corrected sodium 124 mmol/L for hyperglycemia, transaminitis (direct bilirubin 0.3mg/dL, total bilirubin 1.2mg/dL, ALT 113 unit/L, AST 46 unit/L). Troponin were elevated 0.405 ng/mL and pro-BNP 4,591 pg/mL. Urinalysis was significant for glucosuria and elevated WBC 11/HPF. She was admitted to the telemetry unit given elevated troponins and shortness of breath. She had a transthoracic echocardiogram later that day which was significant for EF of 35% with akinesis of the mid-

apical walls of left ventricle and elevated right ventricular systolic pressure of 48mmHg. At this time differentials included myocardial infarction or recurrent takotsubo cardiomyopathy. She underwent cardiac catheterization which was negative for ischemic cardiomyopathy and EF of 25%. Given these findings, she was diagnosed with non-obstructive cardiomyopathy likely from takotsubo cardiomyopathy. The likely stressor was nausea and vomiting when starting nitrofurantoin for urinary tract infection. Treatment plan was for medical management and repeat echocardiography for resolution of EF. Subsequent outpatient echocardiogram revealed resolution of EF.

## Discussion

Takotsubo cardiomyopathy or stress induced cardiomyopathy has been reported in many cases and diagnostic criteria has been established since being first reported. The Mayo Clinic diagnostic criteria include transient left ventricular systolic dysfunction, absence of obstructive coronary disease, electrocardiogram abnormalities, and absence of myocarditis [4]. Although diagnostic criteria has been established, the pathogenesis still remains unclear. One hypothesis is the release of excess catecholamines or excessive sympathetic stimulation can cause transient cardiomyopathy. One study evaluated 9 cases of stress cardiomyopathy precipitated after receiving dobutamine or epinephrine [5]. In all cases, the EF was reduced to an average of 35% with ballooning of the left ventricle and resolution within 7 days. Another study conducted at Johns Hopkins Medical Center evaluated the neurohormonal features of 19 previously healthy patients admitted to coronary care unit for chest pain precipitated by emotional stress [6]. The 19

patients were diagnosed with non-obstructive cardiomyopathy through cardiac catheterization but significantly reduced EFs. It compared the neurohormonal levels of the 19 patients with patients who were diagnosed with acute myocardial infarction and noted that the neurohormonal levels were much higher in the 19 patients without coronary artery disease. There is limited data about endomyocardial biopsy, however studies have shown effects of catecholamines on the myocardium leading to fibrosis and mononuclear cell infiltrates [7]. Another hypothesis is multivessel coronary vasospasms however, it is not usually seen on angiography [8].

Cases of recurrent takotsubo cardiomyopathy are not common. One study analyzed 749 patients with takotsubo cardiomyopathy from the multicenter registry, found that recurrence was about 4% and most recurrences occurred in the first 5 years [9]. In cases of recurrent takotsubo cardiomyopathy, patients typically had higher prevalence of arterial hypertension and chronic obstructive pulmonary disease [10]. In our patient's case, she had a recurrent episode 7 years after first. After the first episode she had complete resolution of cardiac function. The likely stressors were the adverse reaction she had to nitrofurantoin. She had multiple episodes of nausea and vomiting, which likely increased her catecholamine levels and led her into cardiomyopathy. She is doing well post discharge and again had complete resolution of cardiomyopathy on subsequent echocardiogram.

**Study Limitations:** There is a lack of ability to generalize with this report as it details one patient's disease process. Although the diagnosis was definitive, the cause was not able to be clearly established.

## Conclusion

Takotsubo cardiomyopathy is an acute transient reduction in systolic cardiac function that is induced by emotional or physical stressors. It is a non-obstructive cardiomyopathy that resolves after the initial stressor is resolved. Actual pathogenesis is still unclear, however there are hypotheses it involves excessive catecholamine release. Recurrence is rare however it can occur and usually occurs in postmenopausal women with certain risk factors.

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