

# True Case of Takotsubo Cardiomyopathy after Elective Cholecystectomy

Laura Desmarteau, D.O, Alexander Haber and Yatish Merchant, M.D

AtlantiCare Regional Medical Center, Pomona, N.J., U.S.A.

AtlantiCare

## Case Presentation

A 54-year-old female with no prior cardiac history was admitted to acute inpatient care for an elective laparoscopic cholecystectomy. Within the 24-hour post op period, she exhibited new onset tachypnea with a respiratory rate of 32 and hypoxia with an oxygen saturation of 87% on 4L of supplemental oxygen. Serum electrolytes were within normal range and CBC was significant for elevated WBC 29.8. Serial Troponins were obtained at the time of decompensation which showed an initial elevation of 8.88 and proceeded to trend up to 9.99. Additionally, Pro BNP was significantly elevated at 5,451. Subsequent electrocardiogram (ECG) was notable for new dynamic ST-T wave changes (Fig 1).

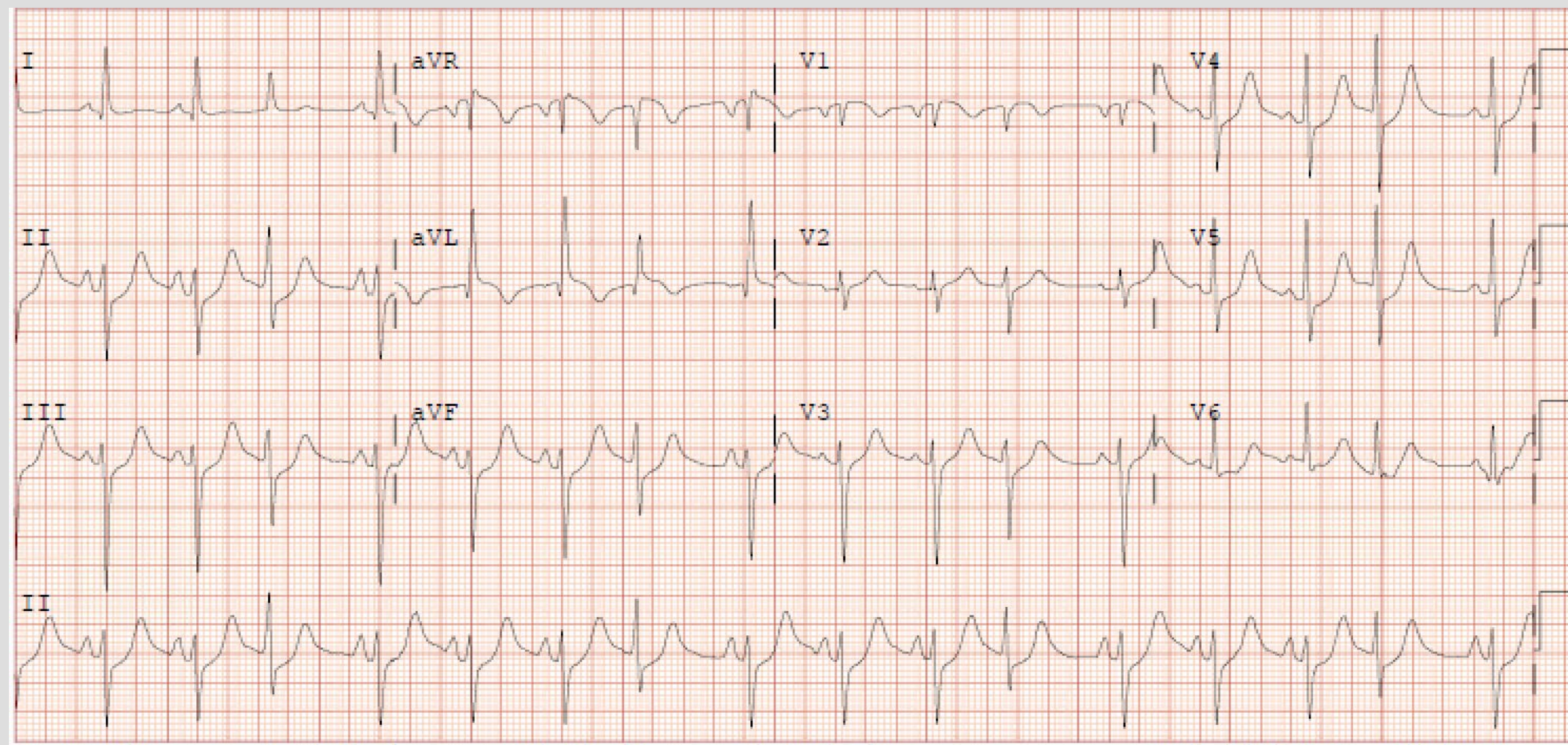


Figure 1. ECG obtained at time of decompensation.

A STAT 2D echocardiogram (ECHO) was obtained, which showed an ejection fraction (EF) of 30% with a severely hypokinetic anterior wall and septum. Two days later a follow up ECHO showed improvement in the patient's EF to 45% and only mild global hypokinesis of the left ventricle. The patient was subsequently diagnosed with Secondary Takotsubo Cardiomyopathy. Prior to discharge patient underwent a cardiac stress test which showed no reversible ischemia or fixed infarct with a reported EF of 59%. The patient continued to follow with outpatient Cardiology and approximately 6 weeks after the initial diagnosis completely recovered.

## Discussion

Takotsubo (stress) cardiomyopathy (TC) is defined by the transient systolic dysfunction of the apical segments of the left ventricle. These abnormalities mimic the symptoms of a myocardial infarction in the absence of any evidence of obstructive coronary artery disease (1). Classically this type of cardiomyopathy has been described in the setting of emotional or psychological stress, but recent evidence has shown that physical stress, including surgical intervention, can also develop this condition (2-4) The presentation of TC is similar to the presentation of patients with acute coronary syndrome and accounts for approximately 2% of patients who present to the hospital with suspected acute coronary syndrome in the United States (4,5) These symptoms are also accompanied by similar changes in troponins, meaning that diagnosis is often muddled until patients undergo cardiac catheterization to rule out an acute coronary syndrome. At the time of catheterization patients show no evidence of obstructive disease, but rather left ventriculomegaly (3) At this time a standardized diagnostic criterion for TC has not been largely agreed upon. However, the proposed criteria established by the Mayo Clinic in 2014 (Table 1) has gained traction (6).

The pathophysiology of this condition has yet to be clearly elucidated, but current evidence from clinical studies point to the role of catecholamines inducing cardiac dysfunction. Wittenstein et al. demonstrated that patients with TC have circulating catecholamine levels 2 to 3 times the upper limit of normal (7). It is hypothesized that a stressor triggers the release of catecholamines, which then leads to TC by a combination of three mechanisms: acute coronary vasospasm due to endothelial

dysfunction, catecholamine induced myocardial stunning, and excessive transient ventricular afterload (5). This acute phase may then be followed by a chronic inflammatory state that could provide a potential explanation for the development in heart failure in a subpopulation of patients afflicted with T (8). Commonly an emotional etiology has been often associated with the inciting event of TC, leading to the colloquial name of "broken heart disease." However, physical stressors can also be the root cause of a TC exacerbation. Thus it is very important to maintain TC as part of the differential diagnosis of a patient who presents with new onset cardiac symptomatology in a post-operative setting.

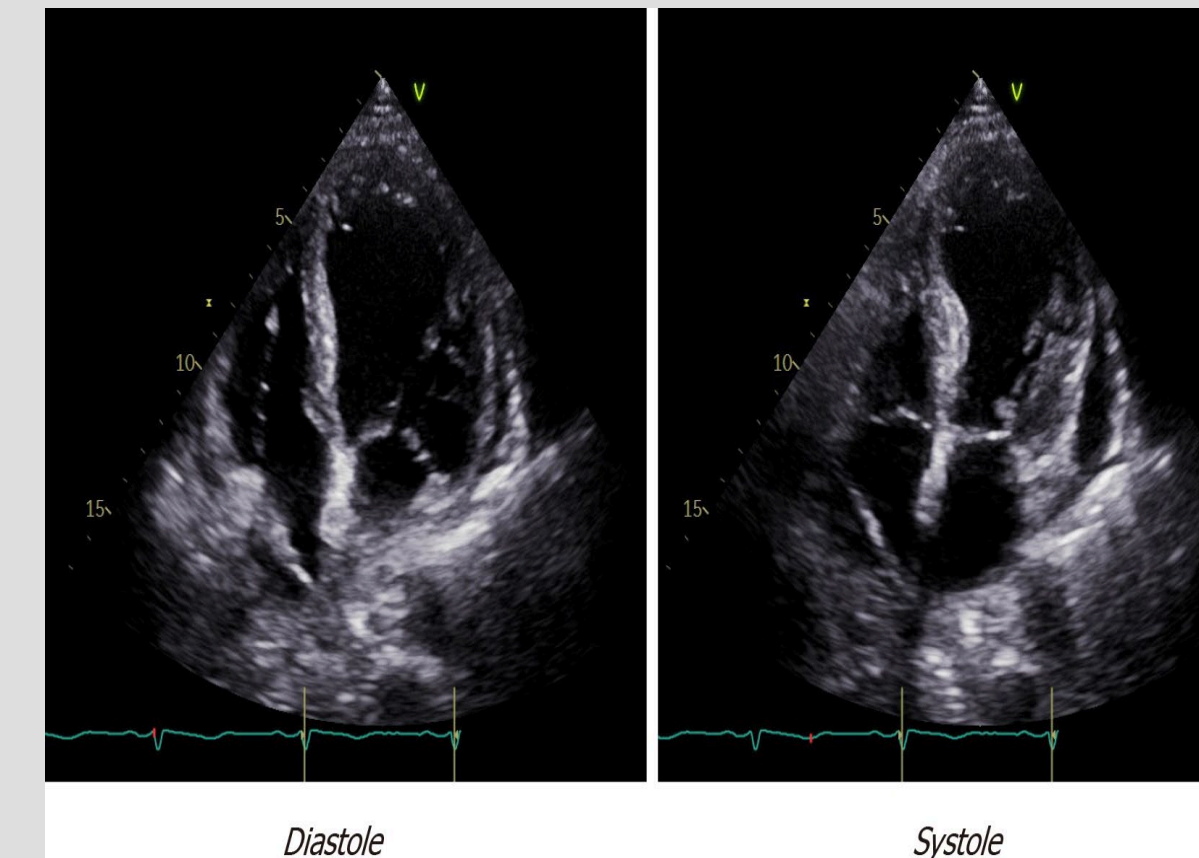


Figure 2. Echocardiogram demonstrating apical ballooning (9)

## Revised Mayo Clinic Criteria

- Transient hypokinesis, akinesis, or dyskinesis of the left ventricular segments with or without apical involvement; abnormalities must extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always present
- Absence of obstructive coronary disease
- New electrocardiographic (ECG) abnormalities usually ST-segment elevations, and/or T wave inversions or a slight increase in cardiac troponin
- Absence of pheochromocytoma or myocarditis

Table 1

## Conclusion

Takotsubo Cardiomyopathy is a rare but significant cause of decompensation in patients with otherwise benign cardiac histories. Both psychological and physical stressors can play a role in the initiation of the hypothesized cascade of catecholamine release that is thought to lead to the disorder. While diagnostic criteria are not yet fully agreed upon, it is important to maintain TC in one's differential for not only medical but surgical patients in the postoperative period.

## References

1. Patankar GR, Choi JW, Schussler JM. Reverse takotsubo cardiomyopathy: Two case reports and review of the literature. *J Med Case Rep.* 2013;7:2-4. doi:10.1186/1752-1947-7-84
2. Maron BJ, Towbin JA, Thiene G, et al. Contemporary definitions and classification of the cardiomyopathies: An American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. *Circulation.* 2006;113(14):1807-1816. doi:10.1161/CIRCULATIONAHA.106.174287
3. Eitel I, Von Knobelsdorff-Brenkenhoff F, Bernhardt P, et al. Clinical characteristics and cardiovascular magnetic resonance findings in stress (takotsubo) cardiomyopathy. *JAMA - J Am Med Assoc.* 2011;306(3):277-286. doi:10.1001/jama.2011.992
4. Templin C, Ghadri JR, Diekmann J, et al. Clinical Features and Outcomes of Takotsubo (Stress) Cardiomyopathy. *N Engl J Med.* 2015;373(10):929-938. doi:10.1056/nejmoa1406761
5. Akashi YJ, Nef HM, Lyon AR. Epidemiology and pathophysiology of Takotsubo syndrome. *Nat Rev Cardiol.* 2015;12(7):387-397. doi:10.1038/nrcardio.2015.39
6. Scantlebury DC, Prasad A. Diagnosis of takotsubo cardiomyopathy – Mayo Clinic criteria –. *Circ J.* 2014;78(9):2129-2139. doi:10.1253/circj.CJ-14-0859
7. Wittstein I, Thiemann D, Lima J, et al. Neurohumoral Features of Myocardial Stunning Due to Sudden Emotional Stress. *N Engl J Med.* 2014;352(6):2467-2477. doi:10.1056/NEJMoa1311376
8. Scally C, Abbas H, Ahearn T, et al. Myocardial and Systemic Inflammation in Acute Stress-Induced (Takotsubo) Cardiomyopathy. *Circulation.* 2019;139(13):1581-1592. doi:10.1161/CIRCULATIONAHA.118.037975
9. Wu, B. F., Shi, J. R., & Zheng, L. R. (2020). Takotsubo cardiomyopathy associated with bronchoscopic operation: A case report. *World Journal of Clinical Cases*, 8(24), 6517.