The Medicine Forum

Volume 23 Article 7

2022

Another Case of Takotsubo Syndrome: Excluded by the Presence of Significant Coronary Artery Disease, or Caused by Significant Coronary Artery Disease?

Bharaniabirami Rajaram, MD
Thomas Jefferson University

Neveda Murugesan, MD
Thomas Jefferson University

Gregary D. Marhefka, MD
Thomas Jefferson University, Gregary.Marhefka@jefferson.edu

CARDIOLOGY

Another Case of Takotsubo Syndrome: Excluded by the Presence of Significant Coronary Artery Disease, or Caused by Significant Coronary Artery Disease?

Bharaniabirami Rajaram, MD[1], Neveda Murugesan, MD[1], Gregary D. Marhefka, MD[2]

1. Department of Internal Medicine, Thomas Jefferson University Hospital, Philadelphia, PA

2. Division of Cardiology, Department of Internal Medicine, Thomas Jefferson University Hospital, Philadelphia, PA

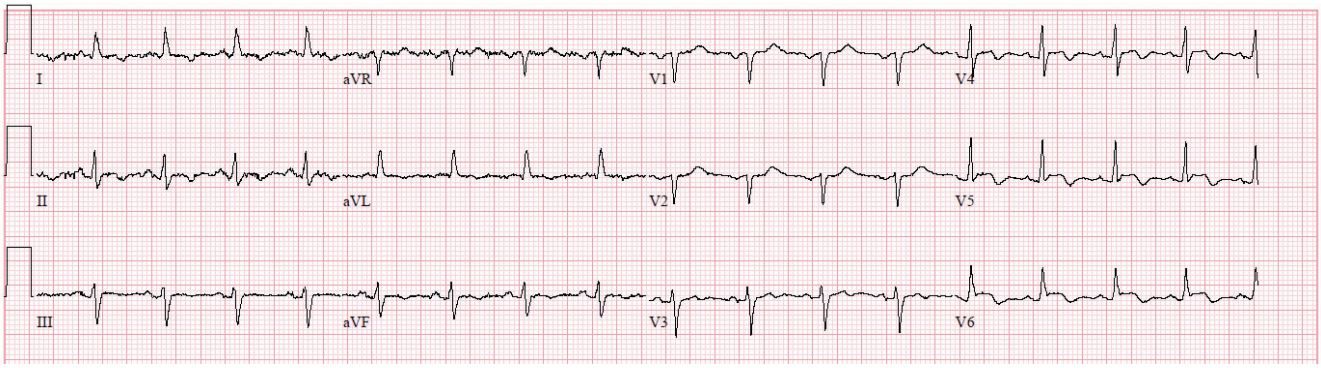

Figure 1: ECG on presentation. 1-2 mm ST-segment elevations and T wave inversions in V4-V6, consistent with a lateral STEMI.

INTRODUCTION

Takotsubo syndrome (TTS) is a reversible condition of abnormal myocardial contraction that was first given this name in Japan by Dr. Sato in 1991.[1] The name comes from the Japanese word for "octopus trap," which has a similar shape to that of the left ventricle on ventriculography during Takotsubo syndrome. It is also known as broken heart syndrome, stress-induced cardiomyopathy, or apical ballooning syndrome. The first descriptions of this phenomenon date as far back as the 1960s.[2-3]

TTS typically presents with symptoms and clinical signs suggestive of acute coronary syndrome (ACS). It may include ST segment elevations on electrocardiogram (ECG) characteristic of acute ischemia even though the syndrome is not caused by direct myocardial ischemia. On echocardiography, TTS is usually characterized by segmental wall motion abnormalities (SWMA) with hyperdynamic contraction of the left ventricular basal walls and akinesis of the apical walls. This results in the "apical ballooning" and is notably not in the distribution of typical coronary artery anatomy.[4] Traditionally, the diagnosis of TTS involves the aforementioned findings and coronary angiography showing no obstructive coronary artery disease (CAD).[5] We present here a case of an acute lateral ST-elevation myocardial infarction (STEMI) with subsequent cardiogenic shock due to TTS.

CASE PRESENTATION

History of Present Illness

A 68-year-old Caucasian female smoker without significant cardiac history presented to the emergency department of a suburban hospital with three days of progressive abdominal pain, initially thought to be a recurrence of her peptic ulcer disease. She described the pain as sharp and constant without radiation. She attempted to take over-the-counter antacids without any relief. She denied other symptoms such as dyspnea, chest pain, or palpitations. Physical exam at that time was unremarkable. Chest x-ray was did not show pulmonary edema or consolidation. Labs at the outside facility were notable for elevated troponin I at 3.18 ng/mL and lactate at 4.3 mmol/L. Her ECG showed ST elevations and terminal T wave inversions in laterals leads (I, aVL, and V4-V6) consistent with a lateral STEMI (**Figure 1**). The patient was transferred to our facility for emergent cardiac catheterization and cardiac intensive care.

Hospital Course

Upon arrival at our facility, the patient underwent coronary angiography, which revealed the culprit lesion was a 100% occlusion of the second obtuse marginal branch of the left circumflex artery . The left main, left anterior descending, and right coronary arteries were found to have only minimal luminal irregularities. A drug-eluting stent was successfully placed and resulted in 0% residual stenosis. Notably, LV end diastolic pressure was elevated at 34 mmHg. She was then transferred to the cardiac intensive care unit for further management. Physical exam was notable for bilateral crackles on lung auscultation. Repeated chest x-ray done five hours from initial image showed new bilateral hazy opacities, consistent with pulmonary edema. She was given intravenous (IV) tirofiban, oral dual anti-platelet therapy, high intensity statin, and IV furosemide. Beta-blocker and angiotensin converting enzyme inhibitor (ACEi) therapies were not initiated due to acute heart failure with mild hypotension (systemic blood pressure 100s/60s).

The patient continued to have intermittent chest discomfort and nausea, without evidence of worsening myocardial ischemia. Serial ECGs showed resolution of ST segment elevations and persistent T-wave inversions. However, repeated lactate was higher than previous and peaked at 6.5 mmol/L. Liver enzymes also increased triggering suspicion for shock liver. Post-intervention transthoracic echocardiogram (TTE) demonstrated a severely reduced left ventricular ejection fraction (LVEF) of 34%, with SWMAs, apical akinesis, and ballooning of the LV apex. Notably, as seen in some cases of TTS, there was no left ventricular outflow tract (LVOT) obstruction or increased intracavitary gradient due to hyperkinesis of the basal segments in this patient. These TTE findings were significantly disproportionate to the relatively small territory of SWMAs expected from her lateral STEMI and more consistent visually and clinically with TTS.

The patient required inotropic support with IV dobutamine in the setting of worsening end organ perfusion from cardiogenic shock. She was also continued on regular IV diuretics for volume status optimization. Status of her cardiogenic shock was not monitored with invasive hemodynamic monitoring, but end organ function was trended closely with serial lactate levels, liver enzymes, and creatinine. On the fifth day of hospitalization, she was successfully weaned off of dobutamine and was transferred from the intensive care unit to the general telemetry floor. TTE on this day showed recovery of LVEF to 60%, with focal inferolateral and anterolateral wall hypokinesis, and there was resolution of the periapical wall motion abnormalities.

DISCUSSION

The diagnosis of TTS is typically made with signs and symptoms of ACS, regional wall motion abnormalities on echocardiography, and the absence of an obstructive culprit lesion on coronary angiography. Bybee et al. proposed the Mayo criteria, which mandate that there is no angiographic evidence of obstructive coronary disease or plaque rupture.5 Other similar criteria have been proposed, and all include the exclusion of obstructive coronary disease and acute coronary thrombus.[6,7]

The pathophysiology of TTS remains poorly understood, though there have been several proposed mechanisms and theories. The most widely accepted explanation is that increased systemic catecholamine levels lead to decreased LV function through microvascular spasm causing transient ischemia, as well as direct cardiotoxicity.[4,6] Emerging data also suggests that there is a role for endothelial dysfunction and estrogen deficiency, resulting in epicardial and/or microvascular spasm.[8] This could partially explain the higher prevalence of TTS in post-menopausal women.

More recently, however, cases of concurrent TTS and MI have been described, suggesting that the co-existence of the two are not exclusionary, as once thought.[9,10] Adding to this small collection, we describe a case in which apical LV dysfunction was significantly disproportionate to the area of lateral infarction. Ultimately, resolution of the SWMAs supports the diagnosis of TTS. Some recent reports also suggest that transient apical LV wall thickening due to myocardial edema is a characteristic feature in the subacute recovery phase of TTS. This was not appreciated in the recovery echocardiogram obtained in this patient, but T2 weighted cardiac magnetic resonance imaging (MRI) may be a more sensitive modality to assess this.[11]

Our case demonstrates that an acute MI can be associated with and in this case, cause a stress response that leads to TTS. TTS and acute MI are not mutually exclusive and in certain cases should not be classified as such. If a patient with a seemingly small infarct develops cardiogenic shock, TTS should be considered in the differential diagnosis and can typically be discerned with TTE and managed expectantly.

REFERENCES

1. Dote K, Sato H, Tateishi H, Uchida T, Ishihara M. [Myocardial stunning due to simultaneous multivessel coronary spasms: a review of 5 cases]. *J Cardiol*. 1991;21(2):203-14. Japanese.
2. Parkes, C M et al. "Broken heart: a statistical study of increased mortality among widowers." *British medical journal* vol. 1,5646 (1969): 740-3.
3. Cebelin, M S, and C S Hirsch. "Human stress cardiomyopathy. Myocardial lesions in victims of homicidal assaults without internal injuries." *Human pathology* vol. 11,2 (1980): 123-32.
4. Kato, K et al. "Takotsubo syndrome: aetiology, presentation and treatment." Heart (*British Cardiac Society*) vol. 103,18 (2017): 1461-1469.
5. Bybee, K A et al. "Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction." *Annals of internal medicine* vol. 141,11 (2004): 858-65.
6. Wittstein, I S et al. "Neurohumoral features of myocardial stunning due to sudden emotional stress." *The New England journal of medicine* vol. 352,6 (2005): 539-48.
7. Redfors, B et al. "Stress-induced cardiomyopathy (Takotsubo)--broken heart and mind?" *Vascular health and risk management* vol. 9 (2013): 149-54.
8. Naegele, M et al. "Endothelial function and sympathetic nervous system activity in patients with Takotsubo syndrome." *International journal of cardiology* vol. 224 (2016): 226-230.
9. Hussain, M A et al. "Apical ballooning (takotsubo) syndrome with concurrent ST-segment elevation myocardial infarction." *BMJ case reports* vol. 2017 bcr2017220145. 24 Jul. 2017
10. Messas, N et al. "Takotsubo cardiomyopathy triggered by ischemic injury: When lateral myocardial infarction precipitate apical ballooning syndrome." *International journal of cardiology* vol. 202 (2016): 858-60.
11. Izgi, Cemil et al. "Myocardial edema in Takotsubo syndrome mimicking apical hypertrophic cardiomyopathy: An insight into diagnosis by cardiovascular magnetic resonance." *Heart & lung: the journal of critical care* vol. 44,6 (2015): 481-5.