

Abstract Form

Hospital Affiliation:	Kern Medical Center
Presenter Name (Last, First):	Narang, Vishal
Co-Authors:	Vishal K. Narang MD, Pearl Chan, Fowrooz Joolhar MD, Aslan Ghandforoush MD, Theingi Tiffany Win MD
Project Title:	A Case of Takotsubo Syndrome Secondary to Hypoxia and Drug Overdose

Research Category (please check one):

<input type="checkbox"/>	Original Research	<input checked="" type="checkbox"/>	Clinical Vignette	<input type="checkbox"/>	Quality Improvement	<input type="checkbox"/>	Medical Education Innovation
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Abstract

A Case of Takotsubo Syndrome Secondary to Hypoxia and Drug Overdose
 Vishal K. Narang MD, Pearl Chan MS3, Fowrooz Joolhar MD, Aslan Ghandforoush MD, Theingi Tiffany Win MD
 Abstract
 Takotsubo syndrome is a condition used to describe patients who present with typical symptoms and findings consistent with acute myocardial infarction (AMI). However, these patients are found to have non-occlusive coronary arteries with distinct features of left ventricular apical dysfunction. The hallmark of this syndrome is the association of a preceding emotional and/or physical trigger. In patients who present with ECG findings and elevated cardiac enzymes, it is important to perform coronary angiography to rule out AMI. Here, we present a patient with history depression and substance abuse who presented to our institution after being found down. Her cardiac course demonstrated progression from NSTEMI to STEMI before ultimately being diagnosed with Takotsubo syndrome.

Case Presentation
 63-year-old female with a history of methadone dependence and COPD was brought into the emergency room by EMS due to unresponsiveness. On arrival to the emergency room, patient was found to have a GCS of 7 with a blood pressure of 117/72 mmHg, heart rate 109 bpm, respiratory rate of 28 and hypoxic to 80%. Physical exam was notable for bilateral wheezing. Patient was administered 2mg of Narcan 10mg IV dexamethasone, and with albuterol inhalation solution however remained unresponsive. She was subsequently intubated for airway protection. Laboratory studies were significant for a lactic acid of 4.5 mmol/L, troponin of 0.08 ng/dL, BNP 385 pg/mL. Urine toxicology was positive for methamphetamine and benzodiazepine. Chest X-ray showed hyperinflation but no consolidation. Patient was admitted to the intensive care unit for further management.

The serial troponin levels peaked at 3.18 ng/dL. Initial EKG showed minimum ST elevation in V3 and V4. Medical therapy for NSTEMI was initiated, however invasive strategy was initially deferred due to an episode of active GI bleed as well as the possibility of Type 2 NSTEMI in the setting of severe hypoxia with severe encephalopathy. Echocardiogram showed akinesis of apex, apical to mid anterior, lateral, inferolateral and inferior walls with hyperkinesis at the bases, and a LVEF of 20%. This is suggestive of CAD/MI vs Takotsubo cardiomyopathy.

On day 3 of hospitalization a repeat EKG showed ST elevations in V3-V5 and an emergency coronary angiogram showed no obstructive coronary artery disease. Invasive LV gram showed typical feature of Takotsubo cardiomyopathy with apical ballooning with hyperkinetic bases of LV.

Conclusion
 Takotsubo syndrome is a condition that is becoming increasingly more recognized. It was first described in 1990 in Japan then again in 2005 in the United States. The diagnostic criteria has since been revised multiple times. New criteria states obstructed coronaries is not a contradiction to diagnosis of TS. Despite this our patient did have unremarkable coronary arteries and experience the hallmark preceding emotional and physical stressor before being hospitalized. In the case of our patient, her course was initially thought to be NSTEMI progressing to a STEMI, as evidenced by abnormal ECG and elevated cardiac biomarkers. However further diagnostic studies would prove the ultimate diagnosis of Takotsubo syndrome.