

# An Atypical Presentation of Takotsubo Cardiomyopathy with Mid-ventricular Wall Ballooning

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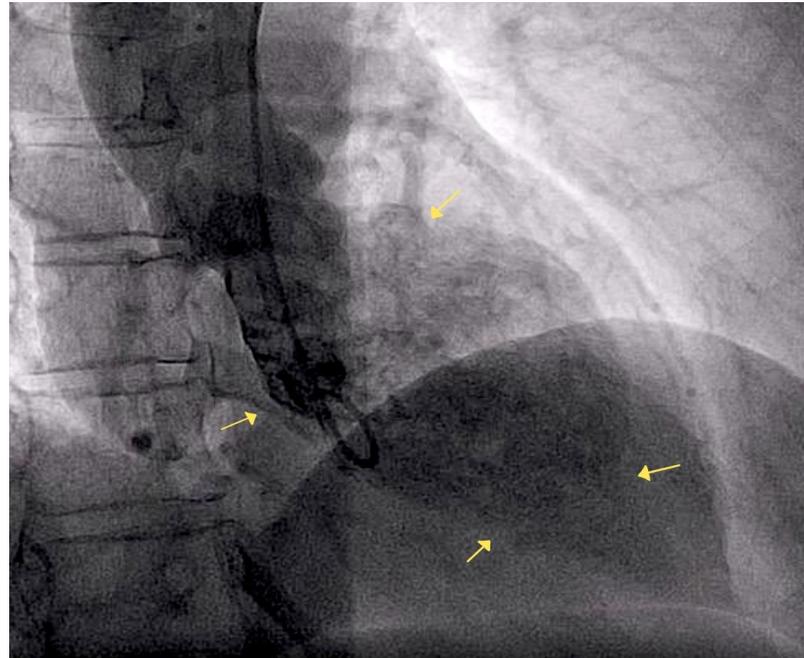
## INTRODUCTION

Takotsubo Cardiomyopathy was first described in Japan in the 1990's as a transient regional wall motion abnormality of the left ventricle (LV), with basal hyperkinesis, apical hypokinesis, and consequent apical ballooning, in the absence of coronary obstruction. Atypical presentations involving different LV segments have previously been reported (1). We describe an intriguing variant consisting of severe hypo-to-akinesis of the mid-ventricular segment and hyperkinesis of the apex and base; mimicking the presentation of acute coronary syndrome (ACS), likely triggered by an exacerbation of the underlying pulmonary disease.

## CASE DESCRIPTION

A 56-year-old African-American female with a past medical history of hypertension, diabetes, and chronic obstructive pulmonary disease (COPD), presented to the emergency room with sudden onset chest pain, dyspnea, and wheezing, requiring non-invasive positive pressure ventilation. She was tachypneic at 30 breaths per minute and tachycardic at 126 beats per minute.

Electrocardiogram showed nonspecific ST segment and T wave abnormalities. Chest X-ray demonstrated emphysematous changes with hyperinflated lungs. Labs revealed leukocytosis and a mildly elevated lactate. Steroids and empiric antibiotics were administered for COPD exacerbation. Troponin was elevated and trended up to a peak of 1225 ng/L. NSTEMI was suspected and she was administered aspirin, atorvastatin, and heparin. Cardiac catheterization was performed, revealing the absence of coronary obstruction, and



the ventriculogram demonstrated mid-segmental LV ballooning. Echocardiography then revealed the aforementioned wall motion abnormalities, with LV ejection fraction of 40%. Having ruled out ACS, the heparin drip was discontinued. She was started on sacubitril/valsartan and metoprolol, switched to oral steroids after clinical improvement, and was discharged with a follow-up appointment in 4 weeks to assess for reversal.

## DISCUSSION

Takotsubo cardiomyopathy has been estimated to be responsible for 1.7-2.2% of all suspected ACS presentations. It has traditionally been described in Asian (57.2% of all cases) and Caucasian (40.0%) populations, making our patient demographically unique (2). Though widely known to have a strong psychosomatic interaction resulting in catecholamine surge as the trigger, studies have

also recognized the role of physical stressors including hypoxia and hypercapnia, similar to our patient. Mid-ventricular Takotsubo is one of the atypical variants constituting 14.6% of all patients with the syndrome (3). It has been demonstrated that patients with atypical variants have fewer serious adverse effects including cardiogenic shock and heart failure when compared to those with the typical apical ballooning variant (4).

## CONCLUSION

Recognizing atypical variants of Takotsubo can have implications in the management of symptoms and complications. It is well known that in typical cases, basal hyperkinesis is worsened by exogenous catecholamines, i.e., vasopressors. However, hypotensive patients with atypical variants can be safely treated with vasopressors without concern for worsening the cardiac output (5).

## REFERENCES

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