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Apr 12th, 9:00 AM - 2:30 PM

### COPD exacerbation induced Takotsubo Cardiomyopathy

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## Introduction

Takotsubo cardiomyopathy or stress cardiomyopathy is a syndrome of transient left ventricular (LV) dysfunction mimicking myocardial infarction, but lacking obstruction of coronary artery disease (CAD) or acute plaque rupture. A characteristic differentiation from CAD is that regional motional abnormality extends beyond a territory perfused with a single epicardial coronary artery. Clinically, it is characterized by apical ballooning of the LV due to depression of mid and apical segments, with hyperkinesis of cardiac basal walls. Women are affected more than men, predominantly in the postmenopausal age.

## Case Report

A 54-year-old Caucasian female with a history of COPD, hypertension, uncontrolled diabetes mellitus, hyperlipidemia, depression and ongoing tobacco use presented with complaints of worsening shortness of breath two days prior to admission. She denied chest pain, worsened cough, palpitations, nausea or vomiting. On examination, she was in distress and anxious, with labored breathing. Upon examining the chest, decreased air entry was present in both lung fields with bibasilar wheezing. Initial lab tests showed mild respiratory acidosis, with pH of 7.24, pCO<sub>2</sub> of 47.4 and pO<sub>2</sub> of 65. Troponins on the day of admission was <0.30. She was initially managed as a COPD exacerbation and received intravenous methylprednisolone, but her breathing worsened and she was placed on BiPAP for acute hypoxic and hypercapnic respiratory failure. Soon after admission, she started complaining of severe right neck pain. Repeat EKG revealed localized lateral J point, anteroseptal q waves and 4mm ST-segment elevation in leads V3 and V4 reciprocal changes and without chest pain. Repeat troponins were slightly elevated to 0.42 ng/ml and CK-MB was elevated to 20.2 ng/ml. A transthoracic echocardiogram showed regional abnormalities in left ventricle with the apex, mid to distal septum and the anterior part of septum was akinetic.

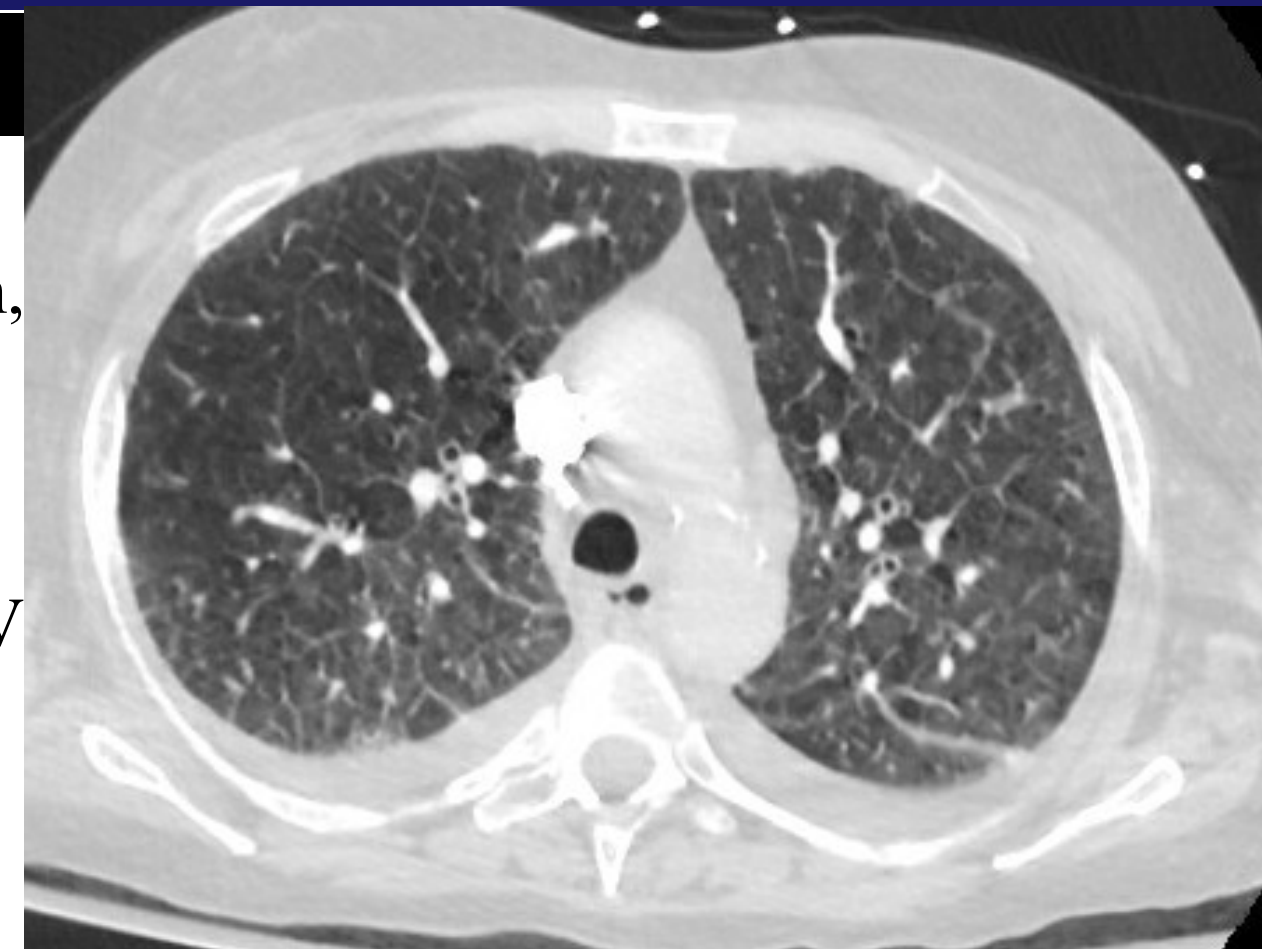


Figure 1: CT scan of the chest shows moderate bilateral pleural effusion with interstitial infiltration and features of COPD

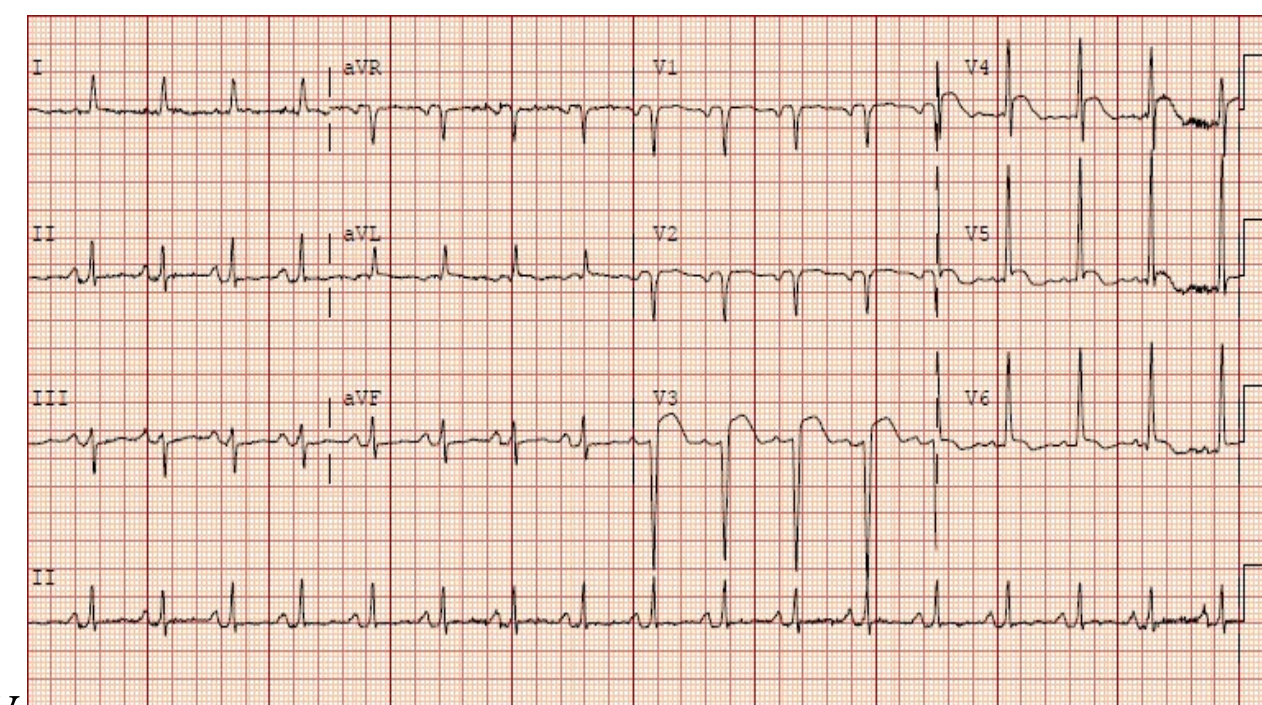


Figure 2: EKG shows anterolateral infarction

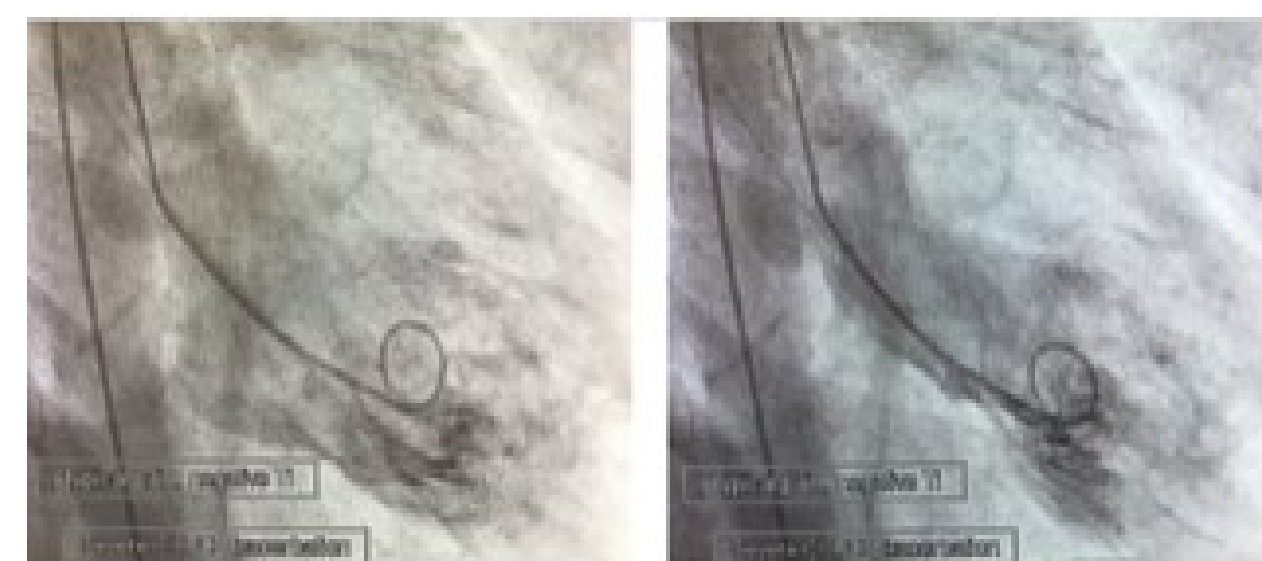


Figure 3. Ventriculogram during Left Heart Catheterization. Left: Normal left ventricle during diastole. Right: Left Ventricular Apical Ballooning during systole.

## Discussion

Many theories for the underlying pathogenesis of Takotsubo cardiomyopathy include catecholamine excess, coronary artery spasm and microvascular dysfunction without conclusive evidence. The trigger for this syndrome varies between emotional or physical stress such as domestic abuse, arguments, natural disasters or acute medical illnesses, but it can be idiopathic as in our case. The most common clinical manifestations include substernal chest pain, dyspnea or syncope. Other manifestations include heart failure, tachyarrhythmia, bradyarrhythmia, cardiogenic shock or even sudden cardiac death. Testing usually shows ST segment elevation, especially in the anterior precordial leads. Serum cardiac troponin and brain natriuretic peptide (BNP) are elevated in most of the cases, with modest elevation of creatine kinase enzyme. The Mayo Clinic suggested a diagnostic criteria for Takotsubo cardiomyopathy to include four elements, including transient LV dysfunction, new EKG abnormalities, absence of coronary artery obstruction, and absence of pheochromocytoma or myocarditis. Management is mostly supportive, with treatment of complications such as heart failure and hypotension amongst others. Most patients recover without complications, but death has been reported in up to 20% of the cases in one study.

## Conclusions

Takotsubo cardiomyopathy presents in 1 to 2 percent of troponin-positive acute coronary syndrome (ACS) with various clinical manifestations and various outcomes. Some patients have favorable outcomes based on their clinical performance and extent of cardiac muscle involvement. As in the case we presented, this syndrome can be entirely idiopathic, without a definitive underlying cause. Supportive management while hospitalized and early identification of complications improve the prognosis.

## References

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