

A rare case of chronic obstructive pulmonary disease induced Takotsubo cardiomyopathy in a male

Ahsan Khan, Biren Patel, Firas Qaqa, Parthiv Patel, Fayeze Shamoon

ABSTRACT

Introduction: Takotsubo cardiomyopathy (TC) is a transient nonischemic cardiomyopathy causing wall motion abnormalities of a particular region of the heart, most commonly the apex. The classic presentation is of a postmenopausal female presenting with chest pain and congestive heart failure in response to an emotional stressor, but physical stressors may cause it as well. Uncommonly, men may present with this condition. We present a rare case of TC in a male, triggered by a COPD exacerbation. **Case Report:** A 62-year-old African American Male with a past medical history of COPD presented with shortness of breath, wheezing and chest tightness. The patient had increased his use of his albuterol inhaler to four times a day, which did not relieve his symptoms. His electrocardiogram showed right bundle branch block and nonspecific T wave changes and his troponins progressively increased to 2.47 ng/mL within 6 hours. Echocardiogram showed apical akinesis with a newly reduced ejection fraction of 30–35%. Cardiac catheterization revealed nonobstructive coronary artery disease and apical ballooning

consistent with Takotsubo cardiomyopathy. Since no other emotional or physical stressor such as sepsis could be identified, it was determined that the patient's COPD was the trigger. The patient was treated medically for his COPD exacerbation until his symptoms resolved. **Conclusion:** This case illustrates the need for a wide differential diagnosis in patient presenting with signs of COPD exacerbation and elevated troponins. COPD causing Takotsubo cardiomyopathy is rare, but not surprising. Evidence is showing that TC is likely caused by increased catecholamine and adrenergic stimulation. COPD exacerbations upregulate the sympathetic nervous system, but the use of beta-2 agonists and anticholinergics as treatment may potentiate TC further based on their mechanisms of action. TC in males is a rare occurrence and it is important to consider that males present with TC differently. They present with more physical rather than emotional precipitators, like our patient and with higher incidence of cardiogenic shock and out-of-hospital cardiac arrest. While the cause of TC is slowly being elucidated, the reason for higher prevalence in females is still unclear.

Keywords: Chronic obstructive pulmonary disease, Male, Stress cardiomyopathy, Takotsubo cardiomyopathy

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INTRODUCTION

Takotsubo cardiomyopathy (TC), first described in Japan in 1990, is known by a variety of names, such as apical ballooning syndrome, stress cardiomyopathy and broken heart syndrome. It is characterized by a transient systolic and diastolic dysfunction, classically in the apex of the heart, but may present in other regions as well. Previous studies suggested TC is predominantly preceded by emotional triggers. Subsequent studies revealed that the prevalence of physical triggers exceeds emotional ones and that an identifiable trigger may even be absent. TC most commonly affects postmenopausal women with a female to male ratio of 9:1. We present a case that is rare for reasons twofold: TC in a male patient, as well as chronic obstructive pulmonary disease (COPD) and possibly bronchodilators as its trigger [1, 2].

CASE REPORT

A 62-year-old African American Male with a past medical history of COPD presented to the emergency room complaining of worsening shortness of breath associated with cough and chest tightness. The patient had increased his use of his albuterol inhaler to four times a day, which did not relieve his symptoms. The patient's vital signs were within normal limits and his physical exam consisted of wheezing and decreased air entry bilaterally. The initial 12-lead ECG showed normal sinus rhythm with right bundle branch block, left anterior fascicular block, but no ST segment, or T-wave changes. Repeat ECG showed T-wave inversions in the inferolateral leads. Troponin I was elevated at 1.48 ng/mL and increased to 2.47 ng/mL 6 hours later. Patient was given dual antiplatelet therapy, along with a beta blocker, statin, ACE inhibitor and heparin. An echocardiogram (Figure 1) showed apical akinesis with an ejection fraction (EF) of 30-35%, a drop from 55-60%, measured years earlier. The patient underwent cardiac catheterization (Figure 2) which showed nonobstructive coronary artery disease and apical ballooning consistent with Takotsubo cardiomyopathy. Since no other stressors could be identified, it was determined that COPD and possibly increased beta-agonist use caused the TC. The patient denied any emotional triggers and had no signs or symptoms of any other conditions, such as infection. No percutaneous coronary interventions were required and the patient was treated medically for his pulmonary disease until his symptoms resolved.

DISCUSSION

Takotsubo cardiomyopathy is a transient hypokinesis, dyskinesis, or akinesis of a segment of the heart, most commonly apical, in response to an emotional or physical stressor. It may mimic acute coronary syndromes,

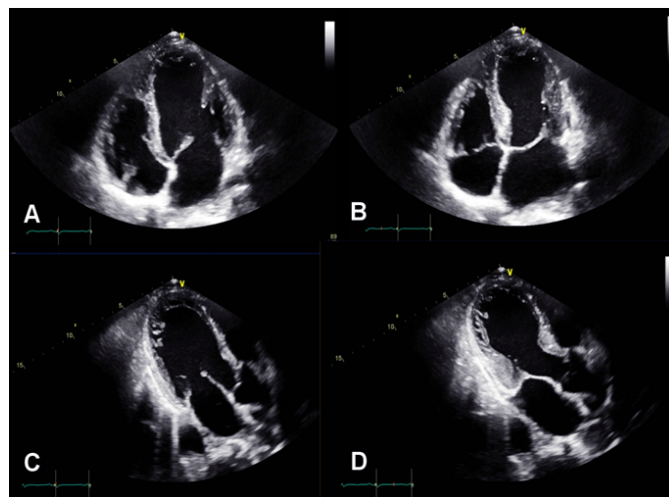


Figure 1: Echocardiogram showing apical akinesis in the apical four chamber view in diastole (A) and systole (B) as well as the three chamber view in diastole (C) and systole (D).

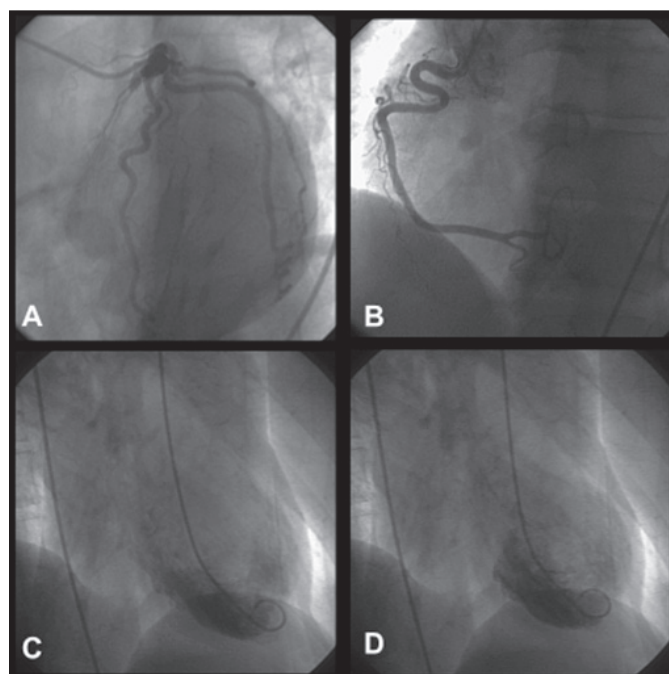


Figure 2: Coronary angiography showing patent coronary arteries (A, B). Ventriculogram via hand injection in diastole (C) and systole (D).

both in its clinical presentation, elevated cardiac enzymes and ECG findings, but resolves spontaneously with supportive care. Although TC is receiving more recognition, its pathophysiology is still not clear. Some proposed theories include impaired myocardial fatty acid metabolism, multivessel coronary artery vasospasm and catecholamine-induced myocardial stunning [3-5]. The catecholamine theory is especially gaining traction due to several supporting studies. Catecholamine levels were found to be elevated at presentation in patients with TC compared to those with myocardial infarction [3]. Endomyocardial biopsies of patients with TC showed

evidence of histopathologic catecholamine toxicity. Rat models demonstrated elevated markers of oxidative stress in TC, which were reduced with alpha and beta adrenergic blockers [1]. Density of adrenoceptors in left ventricular myocardium in animals are higher in the apex and lower in the base, which may explain propensity of apical ballooning caused by TC [6, 7].

COPD causing TC is rare, but it is consistent with our current understanding of its pathophysiology. COPD is an emotionally and physically taxing condition. High sensitivity troponins are often found to be elevated in patients presenting with COPD exacerbation and is associated with higher mortality. Another component of TC in a patient with COPD, is that the treatment itself may potentiate this condition [6–10]. The mainstay of treatment for COPD is bronchodilator therapy, including beta-2 adrenergic agonists and anticholinergics. High doses of Beta-2 agonists may cause adrenergic overstimulation of the heart, excessive catecholamines release and myocyte injury [8–11]. Anticholinergics may cause increased sympathetic activity. Both beta-2 agonists and anticholinergics may cause tachycardia and paradoxical bronchospasm [9]. The effects of these drugs, along with the emotional and physiologic stress of the COPD exacerbation creates a perfect storm for TC.

Takotsubo cardiomyopathy in males is a relatively rare occurrence. Less than 10% of patients who have TC in studies from western countries are male. There appears to be a higher percentage of males in Asian studies, from 13% to 35% [12]. Men and women have similar clinical presentations, but also have some important differences. A study by Schneider et al examined 324 patients with TC from 37 hospitals in Germany and Austria. Chest pain was reported more frequently in women, while dyspnea and syncope were reported equally. Men presented more with cardiogenic shock and out-of-hospital cardiac arrest. Interestingly, QTc prolongation was seen in both men and women, with women having a more significantly prolonged QTc only on the day of symptom onset. Since women normally have a longer QTc, it may have been disproportionately longer in men, leading to arrhythmias [12]. In fact, this was seen in a meta-analysis analysing QT prolongation in TC patients, where torsades de pointes was more prevalent in men [13]. Studies have also shown a difference in precipitating factors, with physical stressors more common in males and emotional stressors more common in females [12, 14]. These discrepancies may have something to do with the lack of potentially protective estrogen in postmenopausal women, in attenuating catecholamine response to mental stress [1, 12, 14].

CONCLUSION

Takotsubo cardiomyopathy caused by COPD exacerbations, especially in a male, is a rare occurrence. This case reminds us to keep TC as a differential in patients

with pulmonary disease, elevated cardiac enzymes and ECG changes. More research is required to delineate the reason for discrepancies of TC in males and females.

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Author Contributions

Ahsan Khan – Substantial contributions to conception and design, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Biren Patel – Substantial contributions to conception and design, Analysis and acquisition of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Firas Qaqa – Substantial contributions to conception and design, Analysis and interpretation of data, Revising the article critically for important intellectual content, Final approval of the version to be published

Parthiv Patel – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

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Guarantor of Submission

The corresponding author is the guarantor of submission.

Source of Support

None.

Consent Statement

Written informed consent was obtained from the patient for publication of this case report.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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