CASE REPORT

Takotsubo Cardiomyopathy Presenting with Dyspnea

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Takotsubo cardiomyopathy (TC), or "broken heart syndrome", is an increasingly recognized condition that mimics acute myocardial infarction with morphologically characteristic left ventricular dysfunction in the absence of coronary artery disease. TC is seen almost exclusively in postmenopausal women following extreme emotional or physiologic stress. Although most patients present with chest pain, limited data suggest that African American patients with TC tend to present with atypical symptoms such as dyspnea or nausea. We present a 57 year-old African American female with TC who presented with severe dyspnea following the shooting death of her son. Added to existing data, our case alerts clinicians to consider Takotsubo syndrome in African American patients with atypical presentation. ***Journal of Hospital Medicine*** **2009;4:200–202.** *© 2009 Society of Hospital Medicine.*

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Takotsubo cardiomyopathy, also known as transient left ventricular apical ballooning, stress cardiomyopathy, and broken heart syndrome, is a condition that mimics acute myocardial infarction. Patients typically present with chest pain, electrocardiographic changes consistent with acute ischemia or infarct, and elevated cardiac enzymes in the absence of significant coronary artery disease. Left ventriculography demonstrates a characteristic pattern of dysfunction: dyskinesis of the cardiac apex and hyperkinesis of the base. This resulting appearance of apical ballooning is reminiscent of the takotsubo, a Japanese octopus pot with a wide base and narrow top. The syndrome occurs almost exclusively in postmenopausal women and demonstrates a distinct temporal association with extreme emotional or physiological stress. The pathophysiology is poorly understood, but one theory suggests that the transient cardiomyopathy reflects myocardial stunning due to excessive sympathetic output.[1] Treatment is supportive, and most patients rapidly recover normal systolic function.

A 57-year-old African American female with a past medical history significant only for chronic obstructive pulmonary disease presented with severe dyspnea that was progressive over several hours following the unexpected death of her son. She denied chest pain, palpitations, cough, or fever. On examination, she was afebrile with a blood pressure of 145/82 mm Hg, a pulse of 90 beats per minute, and a respiratory rate of 24 breaths per

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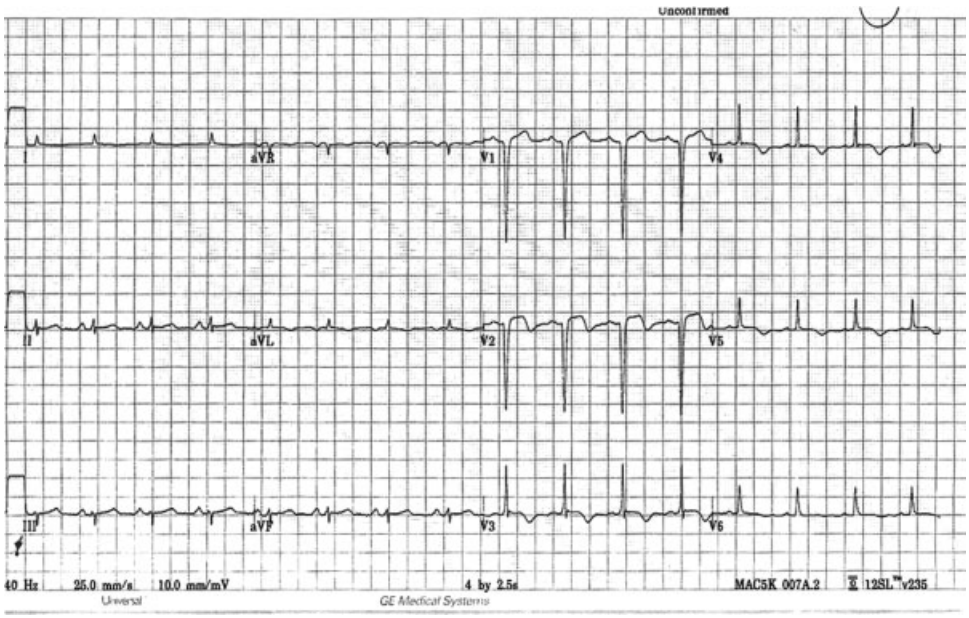

FIGURE 1. Electrocardiogram showing ST-segment elevations in leads V1 and V2 as well as T-wave inversions in all precordial leads.

minute with oxygen saturations of 88% on room air. Lung examination revealed coarse breath sounds with a slightly prolonged expiration phase, but it was otherwise clear. Cardiac examination was unremarkable. Chest radiograph showed only emphysematous changes. Initial electrocardiogram and serial cardiac enzymes were negative. A computed tomography pulmonary angiogram showed no evidence of pulmonary embolism. The patient was admitted with the diagnosis of chronic obstructive pulmonary disease exacerbation and treated with supplemental oxygen, bronchodilators, and corticosteroids.

On the following day, the patient developed worsening dyspnea, hypoxia, and diffuse crackles on examination. Electrocardiogram at that time demonstrated ST-segment elevations in leads V1 and V2 as well as T-wave inversions in all precordial leads (Figure 1). The troponin-I concentration was 1.92 ng/mL (<0.05 nL), and the brain natriuretic peptide concentration was 1425 pg/mL. The patient underwent urgent cardiac catheterization with no evidence of coronary artery obstruction. Left ventriculogram revealed a hyperdynamic base and akinetic apex extending into the mid-heart (Figure 2). Left ventricular systolic function was severely reduced, with an estimated ejection fraction of 10% to 15%. The normal diastolic ventriculogram image is shown for comparison (Figure 3).

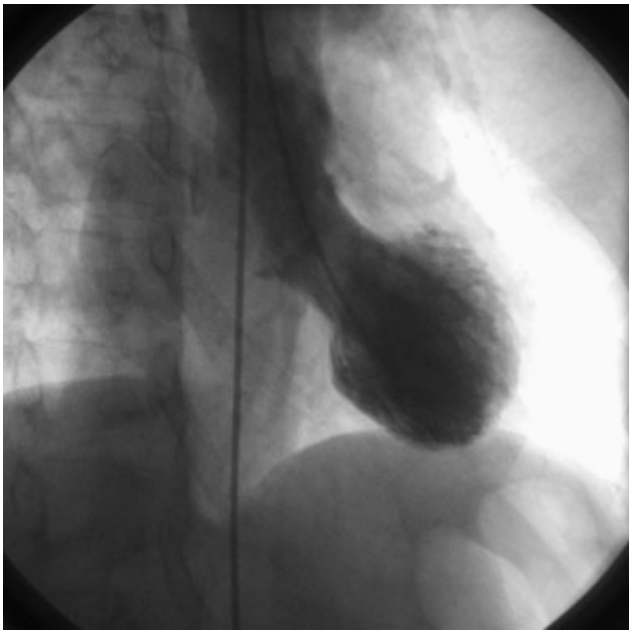

FIGURE 2. Left ventricular angiogram in systole demonstrating characteristic apical ballooning due to focal hypokinesis in takotsubo cardiomyopathy.

These findings were felt to be consistent with takotsubo syndrome. The patient required inotropic support briefly but experienced full clinical recovery by the sixth hospital day.

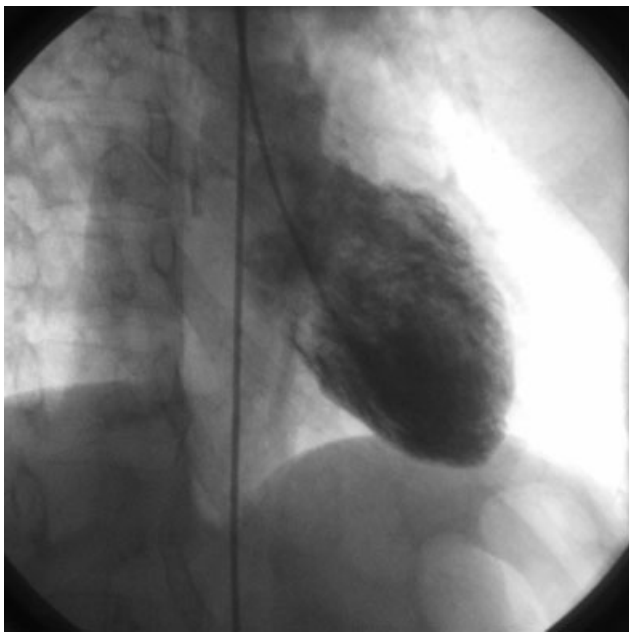

FIGURE 3. The patient's angiogram in diastole (shown for comparison).

DISCUSSION

Takotsubo cardiomyopathy was first described in Japan in 1991.[2] Although the condition made a relatively recent debut in the United States, with the first case series published in 2003, subsequent reports have suggested that the condition is not rare.[3] Recent analyses of Western populations estimate the prevalence to be approximately 2% among patients with acute coronary syndrome.[1,4,5] Because women compose the majority of patients with takotsubo cardiomyopathy, the prevalence among women as a subset of patients with acute coronary syndrome is likely much higher. The syndrome has been described as a clinical entity in Japanese, European, Caucasian, and African American patients.[2,3] Interesting differences appear to exist among different ethnic groups. For example, evidence suggests that the condition is more likely to be precipitated by emotional stress in Caucasians, whereas physiological stress is a more frequent trigger in Asians.[6]

Although chest pain is described as a cardinal feature in takotsubo cardiomyopathy, existing data suggest that African American patients may lack this typical symptom. The first African American female reported with takotsubo syndrome presented with heart failure and hypotension in the absence of chest pain.[1] Subsequently, Patel et al.[7] reported 5 African American women with takotsubo syndrome. Three patients presented with dyspnea, and 2 presented with nausea: none of the patients experienced chest pain. Our case adds to this evidence by describing an African American woman with takotsubo syndrome whose presenting symptom was severe dyspnea without chest pain. Unlike the majority of reported cases, electrocardiographic and biomarker abnormalities were not present in our patient at admission. As with our patient, the diagnosis of takotsubo cardiomyopathy may initially be overlooked in African Americans because of the atypical presentation. As takotsubo syndrome becomes increasingly recognized in the United States, clinicians are encouraged to consider the diagnosis in African American women who present with severe dyspnea in the setting of extreme emotional or physiological stress. Further research on the pathophysiology of takotsubo cardiomyopathy is needed to explain why such differences in presenting symptoms may exist.

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