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Case Report

A Case of Takotsubo Cardiomyopathy

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Case Report

A 72-year-old female with history of COPD presented to the ED for evaluation of shortness of breath. Her daughter was at the bedside during the interview and reported the patient became acutely short of breath, turning "blue in the face", while walking to her car. The daughter proceeded to call EMS. The patient denies any chest pain, hemoptysis, nausea, diaphoresis, and dizziness during the episode. The patient stated she had been under a significant amount of stress lately due to the recent death of her husband three months prior. In addition, she was anxious due to having undergone a bone marrow biopsy two days before for evaluation of a myeloproliferative disorder. Results of the bone marrow biopsy revealed polycythemia vera.

Physical examination

The patient was an overweight, Caucasian female in no acute distress. Her head was normocephalic, atraumatic. Pupils were equal, round, reactive to light and accommodation. Oropharynx was moist. The neck was soft and supple without JVD or bruits. The lungs revealed bibasilar crackles. No wheezing or rhonchi noted. The heart was tachycardia, regular rhythm without any murmur, rub, or gallop. The extremities were without cyanosis, clubbing, or edema. Distal pulses were 1 plus and symmetric throughout.

Diagnostic tests

Serial Troponin T's were elevated at 2.26 and 6 hours later at 2.48. CPK was 88. D-dimer was elevated at 2427. BNP 185. Platelets 579, Hemoglobin were elevated at 17.9. ECG revealed sinus rate and rhythm, left ventricular hypertrophy, and T-wave inversion in anterior and septal leads. Chest X-Ray was interpreted as congestive changes, basal scarring and mild emphysema. Telemetry revealed sinus tachycardia at 113-116 BPM. Echocardiogram revealed mildly reduced left ventricular systolic function with Ejection Fraction (EF) of 45 percent. Wall motion abnormalities in the interventricular septum. Mild concentric left ventricular hypertrophy. No gross valvular abnormalities.

At this point it was recommended the patient undergo left and right cardiac catheterization to rule out an ischemic source of wall motion abnormalities of the interventricular septum, reduced EF, and elevated Troponin T.

Left and right cardiac catheterization revealed mild coronary artery disease. Reduced left ventricular systolic function with evidence of apical hypokinesis consistent with apical ballooning syndrome or Takotsubo syndrome. Elevated left ventricular end-diastolic pressure. Mildly elevated pulmonary artery systolic pressures.

Discussion

Takotsubo Cardiomyopathy (TCM) or stress cardiomyopathy, first described in Japan in 1990, is a transient, acute condition characterized by apical ballooning of the left ventricle [1]. The clinical presentation of stress cardiomyopathy gives the appearance of an acute coronary syndrome, but lacks evidence of ischemic heart disease upon cardiac angiography [1]. In most cases of TCM, according to Reeder and Prasad, "the regional wall motion abnormality extends beyond the territory perfused by a single epicardial coronary artery" ^{2(p1)}. The exact etiology is unknown, but appears to be due to an emotionally or physically stressful event inducing a state of excess catecholamine leading to "diffuse catecholamine-induced microvascular spasm or dysfunction, resulting in myocardial stunning" [2] ^(p1). This state of transient left ventricular dysfunction predominately occurs in postmenopausal women at a mean age of 62-76 years [3].

Reeder and Prasad state, "The most common presenting symptom is acute substernal chest pain, but some patients present with dyspnea or syncope" [2] ^(p1). Electrocardiogram abnormalities, such as ST segment elevation, are commonly encountered in a patient with TCM. The amplitude of ST elevation in patients with stress cardiomyopathy is noted to be generally lower and will less frequently have reciprocal changes when compared to patients with a STEMI [3]. Less frequent ECG findings include ST depression, abnormal Q waves, T wave inversion, and non-specific [2]. In addition to ECG abnormalities, Vivo et al. states, "cardiac biomarkers, including creatine kinase-MB fraction and troponin I and T, are elevated in majority of patients although to a relatively lower degree than seen in MI" [3] ^(p1911-1912). Coronary angiography is required for diagnosis due to the similarity of findings on ECG and cardiac markers in TCM and Acute Coronary Syndrome (ACS) [1].

According to Merchant et al., "Patients should be treated as having ACS until proven otherwise. Aspirin and heparin should be initiated, and a cardiologist consulted" [1] ^(p108). After the exclusion of ACS through coronary angiogram, management of TCM consists of supportive treatment with beta-blockers, ACE- inhibitors, and diuretics [1].

Generally, the prognosis for TCM is considered favorable with full recovery of LV function occurring within 1 to 4 weeks [1]. Repeat echocardiogram is recommended in 4 to 8 weeks after discharge to document full recovery of LV dysfunction [3]. *Vivo* et al. states, "The most common complications are heart failure, pulmonary edema, dynamic outflow tract obstruction and cardiogenic shock" [3] ^(p1912).

Conclusion

The acute onset of dyspnea in a 72-year-old female with ECG abnormalities and elevated Troponin T's provided the misleading clinical presentation of an acute coronary syndrome while in the

emergency department. It is noted the patient presented with the classic description of a stressful event, inducing a state of catecholamine excess that "stunned" the apex of the heart, but lacked chest pain that is typically associated with this syndrome. The patient was treated as though symptoms were attributed to an ischemic source until cardiac angiogram was performed. A postmenopausal woman who presents with symptoms that suggest an ischemic cardiac event with a history of recent stressful episode should prompt a clinician to have a high index of suspicion for TCM.

The patient was managed symptomatically with IV diuretics to reduce symptoms of systolic dysfunction. Unfortunately, due to multiple comorbidities, the patient became septic during this hospital stay and expired 10 days after admission.

References

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