CLINICAL VIGNETTE

Stress Cardiomyopathy

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A 72-year-old female with a history of HTN, hypothyroidism, and reactive airway disease presented to the emergency room with the acute onset of chest tightness with associated dyspnea, nausea, and diaphoresis. An EKG was performed and showed normal sinus rhythm of 90 with 2 mm ST elevations in leads V3-V6. Laboratories were significant for a troponin of 2.6 and CPK MB of 19.1. The patient underwent coronary angiography, which showed normal coronary arteries with apical hypokinesis with basal sparing and given these findings was diagnosed with stress cardiomyopathy.

Discussion

Takotsubo (stress) cardiomyopathy was first described in 1990 and is characterized by transient regional systolic dysfunction affecting the left ventricular apex. The name is derived from takotsubo, a Japanese fishing pot used for catching octopus whose shape mimics the apical ballooning that is characteristic of the syndrome. The diagnostic criteria for the syndrome includes the presence of a transient abnormality in left ventricular wall motion beyond a single coronary artery perfusion territory, the absence of obstructive coronary artery disease or evidence of acute plaque rupture, and the presence of new EKG abnormalities or elevated cardiac troponin levels.[1]

Presentation

Takotsubo cardiomyopathy presents with symptoms similar to an acute coronary syndrome with chest pain and often associated dyspnea or syncope. The EKG most often shows ST segment elevation and troponin levels are often elevated on initial presentation but do not show the amount of increase typically seen with the acute coronary syndrome. A study of 1750 patients admitted with takotsubo cardiomyopathy showed that 87% had an elevated troponin on admission. Patients with takotsubo cardiomyopathy typically had a rise of troponin by a factor of 1.6 during their hospital course, while patients with the acute coronary syndrome had a rise in troponin by a factor of 6.[2] BNP was elevated in 82.9% of patients in this study, which was higher than that seen in patients presenting with an acute coronary syndrome.

Epidemiology

Takotsubo cardiomyopathy typically presents in older women. A review of 7 case series showed a female preponderance of 82-100%,[3] and in a series of 1750 patients,[2] 89.8% were women. Patients with the takotsubo syndrome were usually older with a mean age of 66.8.

Takotsubo cardiomyopathy often occurs after an episode of physical (surgery, infection, or acute respiratory failure) or emotional stress, hence the name stress cardiomyopathy. The study of 1750 patients showed that 36% followed a physical trigger, 27.7% followed an emotional trigger, 7.8%, followed both, and 28.5% of cases had no obvious trigger.[2] Interestingly, following an earthquake in Japan in October 2004, the incidence of Takotsubo cardiomyopathy increased from 1 case in the 4 weeks prior to the earthquake to 25 cases in the 4 weeks following the earthquake.[4]

Patients with a history of psychiatric or neurologic disorders may be predisposed to the development of takotsubo cardiomyopathy. 55.8% of patients with takotsubo cardiomyopathy were found to have a history of either a neurologic or psychiatric disorder, suggesting a possible neurologically mediated mechanism of myocardial injury.

Etiology

At the present time, the cause of takotsubo cardiomyopathy is unknown. Due to its association with physical and emotional stress, one theory is that it is catecholamine mediated. A study examining plasma catecholamine levels on admission showed significantly elevated levels of epinephrine, norepinephrine, and dopamine, compared to other patients presenting with myocardial infarction.[5] Also, the wall motion abnormalities seen in takotsubo cardiomyopathy occur in the same distribution as those seen in cardiomyopathy due to catecholamine release from pheochromocytoma.[6] Possible mechanisms of myocardial stunning due to catecholamine release include epicardial coronary artery spasm, sympathetically mediated microcirculatory dysfunction, or catecholamine mediated direct myocyte injury. A possible explanation for the characteristic apical involvement seen in takotsubo cardiomyopathy is that the apical myocardium shows enhanced responsiveness to sympathetic stimulation, perhaps making the apex more susceptible to surges in circulating catecholamine levels.

Another theory about the etiology of takotsubo cardiomyopathy is that it is neurologically mediated. 55.8% of patients with takotsubo cardiomyopathy have a history of

psychiatric or neurologic disorders. One hypothesis is that the syndrome results from myocardial stunning due to vasoconstriction that is neurologically mediated and further study should be pursued to investigate this recently noted association.

Prognosis

The treatment of takotsubo cardiomyopathy is generally supportive care for congestive heart failure with diuretics and vasodilators. Short-term anticoagulation is often given to prevent thrombus formation due to left ventricular systolic dysfunction. Takotsubo cardiomyopathy typically shows resolution of left ventricular dysfunction over time. Despite this, long term follow-up shows an all cause death rate of 5.6% per patient year and a risk of cardiac and cerebrovascular events of 9.9% per patient year. Interestingly, men had higher rates of death and cardiac and cerebrovascular events compared to women. Recurrence is typically rare, 1.8% per patient year.[2]

Conclusions

The patient was discharged home on furosemide 40mg/day, ramipril 5mg/day, carvedilol 3.125 mg BID, and warfarin 4mg and did well with no lower extremity edema, paroxysmal nocturnal dyspnea, or orthopnea. She underwent a repeat echocardiogram two months later, which showed an ejection fraction of 55-60%, improved from an EF of 45-50%, and a ventriculogram, which showed apical ballooning and reduced ejection fraction. She has no adverse events or recurrence 6 years following her initial presentation.

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