Title

Broken Heart Syndrome or Stress Cardiomyopathy

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CLINICAL VIGNETTE

Broken Heart Syndrome or Stress Cardiomyopathy

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An 85-year-old female with a history of hypertension presented to the hospital with sudden onset of pressure like chest pain and dyspnea at rest. Her husband had died two days prior to admission and she was returning from his funeral when symptoms started.

In the ED she was hypertensive and tachycardic. She was in mild respiratory distress. Her neck veins were elevated. Cardiac auscultation confirmed a S3 gallop. Her lung exam revealed the presence of fine rales at both bases with no dullness on percussion. She had no lower extremity edema.

A previous ECG was normal (Figure1). ECG in the ED revealed minimal ST segment elevation with deep symmetric T – wave depression in the anterior leads (Figure 2). Troponin level was mildly elevated. Her chemistries were within normal limits. Chest radiograph showed prominent pulmonary vasculature with mild cardiomegaly.

She received oxygen and intravenous furosemide with significant improvement in her symptoms. She was treated for an acute coronary syndrome with aspirin, plavix, heparin, beta-blockers and statins. ACE-inhibitors were added on the second day. A bedside echocardiogram revealed wall motion abnormality in the anterior wall and apex with a hypercontractile base (apical ballooning).

Predischarge coronary angiography revealed non-obstructive coronary artery disease. Left ventriculography showed apical ballooning of the left ventricle in systole. The basal areas had preserved wall motion (Figures 3 and 4).

The patient was discharged on aspirin, beta-blockers, ace inhibitors and statins. A repeat echocardiogram performed 4 weeks after presentation revealed normal systolic function with no wall motion abnormalities.

Background:

Tako-Tsubo cardiomyopathy (also known as transient ventricular apical ballooning syndrome, apical ballooning cardiomyopathy, stress induced cardiomyopathy, ampulla cardiomyopathy , broken heart syndrome, Gebrochenes-Herz-syndrome and simply stress cardiomyopathy), is a rapidly reversible form of cardiomyopathy with weakness of the apical regions of the heart with relative sparing of the basal areas. This abnormality is noted in the absence of significant coronary artery disease. In 1991, Dote et al [1] described the dilated apical area and a contracted basal appearance of the left ventricle in systole and named the cardiomyopathy “tako-tsubo” after a Japanese fishing pot used for trapping octopus, which resembles the shape of the left ventricle at end systole. Since then, many variants of this condition have been described.

Pathophysiology:

The mechanisms of this condition are not clearly known, but a surge in catecholamines from underlying stress is the most common etiology. Other cases postulated are coronary artery vasospasm, endothelial dysfunction due to disturbances in microcirculation and a small left ventricular size promoting a left ventricular outflow tract obstruction with resulting

ballooning of the apex[2]. Most likely the etiology is multifactorial with an abnormal response to catecholamines (released in response to stress), failure of the microvasculature and some amount of vasospasm.

The putative mechanism is neurogenic stunned myocardium, similar to catecholamine-induced cardiomyopathy in patients with pheochromocytoma[3].

Another intriguing question surrounding tako-tsubo cardiomyopathy is why the apical wall is affected but the base is spared. The apex is structurally vulnerable because it does not have a 3-layered myocardial configuration, it has a limited elasticity reserve, it can easily become ischemic as a consequence of its relatively limited coronary circulation, and it is more responsive to adrenergic stimulation[4]. All of these factors make the apex more sensitive to a catecholamine-induced surge postulated as the mechanism behind Tako-tsubo cardiomyopathy.

Presentation:

This syndrome often affects postmenopausal women who present after a significant acute stressful situation of an emotional or physical nature. The cause of female preponderance is not known. The typical presentation with stress cardiomyopathy is a sudden onset of congestive heart failure or chest pain associated with ECG changes suggestive of an anterior wall myocardial infarction[5].

Diagnosis:

Patients are generally hospitalized. Echocardiography reveals wall motion abnormalities in the apical area with normal contraction of the base and creation of a left ventricular outflow tract gradient. Non-invasive studies for ischemia may reveal perfusion abnormalities. Cardiac catheterization is generally required to confirm the absence of coronary artery disease.

The majority of patients have a quick recovery with resolution of cardiac function before discharge. In rare cases, the clinical course is complicated by cardiogenic shock, decompensated heart failure, ventricular arrhythmias and death. Patients with T-wave abnormality and physical stress tend to have an increased risk of complications. Long-term cardiac abnormalities are rarely seen and should prompt evaluation for a different etiology. In some cases, the apical area of hypokinesis is associated with apical thrombus formation[6].

Management:

The acute care of these patients is largely supportive. Patients with fluid overload should be treated with diuretics. Anticoagulation should be considered in patients with an intracavitary thrombus. In rare cases of cardiogenic shock, patients require placement of an intra-aortic balloon pump and infusion of inotropes. There are no randomized trials to evaluate the efficacy of any form of treatment in this population. However, treatment with beta blockers and angiotensin converting enzyme inhibitors are the major therapies.

Prognosis:

Despite the grave initial presentation, most individuals survive the initial acute event, with a very low rate of in-hospital mortality or complications. Although, infrequent, recurrence of the syndrome has been reported and seems to be associated with the nature of the trigger[7].

Conclusion:

In summary, Tako-tsubo cardiomyopathy is commonly confused with an acute coronary syndrome and requires hospitalization and cardiac catheterization for accurate diagnosis. The majority of patients have a favorable outcome post discharge.

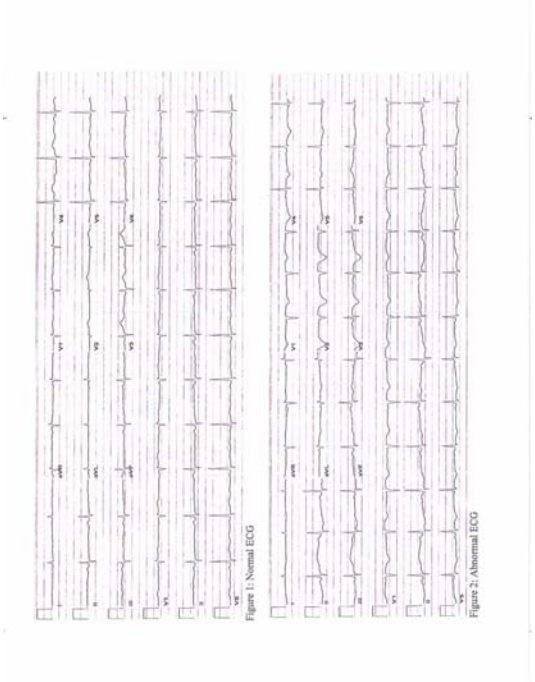

Figure 1 and 2

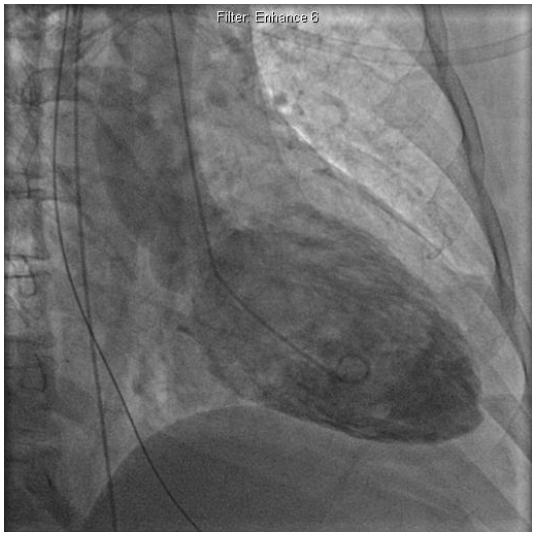

Figure 3

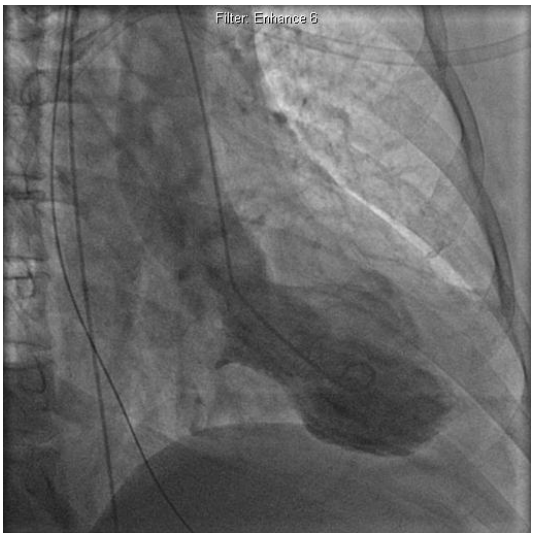

Figure 4

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