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Stress Cardiomyopathy: A Case of Apical Ballooning Syndrome followed by Mid-ventricular Ballooning Syndrome as Recurrence

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Abstract Stress cardiomyopathy has an association with emotional or physical stressors and is found to be more common in postmenopausal women. It has a similar presentation as acute myocardial infarction. Involvement of apex at the initial presentation and recurrence at the mid ventricular segment is rare. We describe a case of a 46 year old female with apical involvement at the initial presentation but mid ventricular involvement at the recurrence. 46 year old woman developed sharp chest pain while giving awards at an honors assembly. She was brought to the ER and was found to have a troponin level elevation without any EKG changes. Cardiac cath did not show coronary artery disease. ECHO revealed a low EF of 30 % with apical ballooning. Patient's symptoms resolved and her EF normalized on repeat ECHO. 2 years later patient developed similar symptoms. She had a troponin elevation without EKG changes. ECHO revealed mid-ventricular ballooning with a low EF. Patient's symptoms again resolved and repeat ECHO showed a normalized EF. Our patient had two different episodes of stress cardiomyopathy. Recurrence rate has been reported up to 11.4 percent in previous studies (5). This case is unique as recurrence was at different anatomic locations i.e apex followed by the mid ventricular segments. Further research is needed to understand the underlying mechanisms associated with the stress cardiomyopathy as current hypothesis do not completely explain the disease.

Keywords: *apical, mid-ventricular, stress cardiomyopathy, recurrence*

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1. Introduction

Stress cardiomyopathy was first described in Japan and now is a well-recognized form of cardiomyopathy [1]. Generally it has an association with emotional or physical stressors and is found to be more common in postmenopausal women [2]. It has a similar presentation as acute myocardial infarction and about 1-2 percent of all patients with initial diagnosis of MI have stress cardiomyopathy [3].

Left ventricular apex is the most common site of ballooning however other variants have been described as well including involvement of mid ventricular segment. [4] Recurrence rate of stress cardiomyopathy is variable with one study showing a 4 year recurrence rate of up to 11.4 percent [5]. Involvement of apex at the initial presentation and recurrence at the mid ventricular segment is rare. We describe a case of a 46 year old female with apical involvement at the initial presentation but mid ventricular involvement at the recurrence.

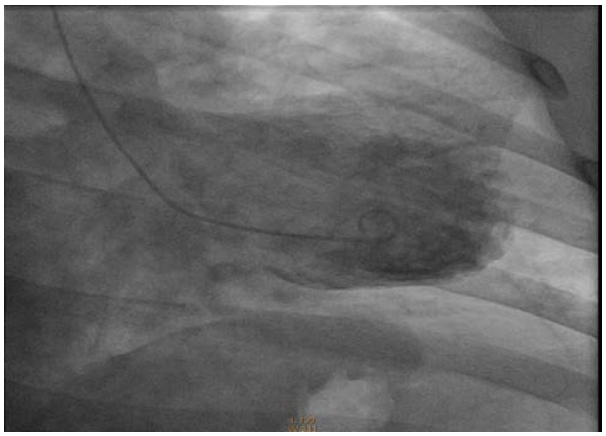
2. Case

A 46 year old woman developed chest pain when she was handing awards on stage at an honor assembly. She described the pain as sharp, knife like and radiating to the back and neck. It was associated with cold sweats and shortness of breath. She was brought to the ER. Her past medical history was positive only for mitral valve prolapse and she was not taking any medications. Her EKG and chest X-ray were unremarkable. Initial troponin I was 3.1 (Reference normal value: <0.50). Her pain persisted despite of nitroglycerine and heparin drip. Cardiac catheterization revealed normal coronary arteries. Left ventricular ejection fraction was found to be 30 percent with dyskinetic apex and hyperdynamic basal walls consistent with apical ballooning syndrome. Her symptoms improved and Echocardiogram done one month later showed normalized left ventricular wall motion and left ventricular ejection fraction of 57 percent.

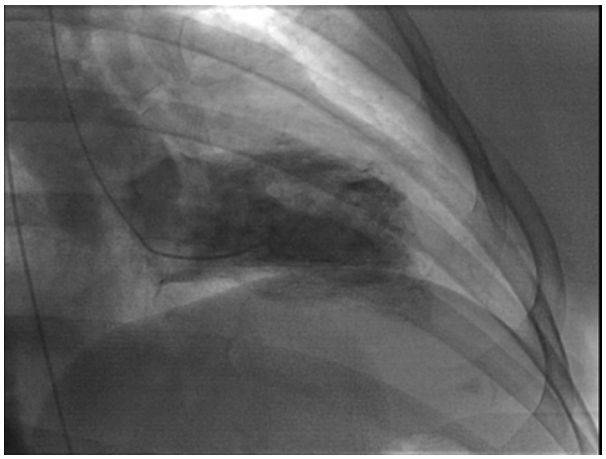
Two years later patient presented with retrosternal chest pain which was similar to the initial episode. This time patient was in a graduate class and did not report any stress. EKG was unremarkable but troponin I was found to be 1.43 which trended up to 2.16. Cardiac catheterization showed normal coronary arteries. LVEF was found to be 40 percent. There was circumferential akinesis of mid ventricular walls and hyper dynamic motion of base and apex consistent with mid ventricular ballooning syndrome (MVBS). Symptoms improved while patient was in the hospital and the follow up Echocardiogram done later

showed normalized wall motion and left ventricular ejection fraction of 60 percent.

THESE ARE THE AVAILABLE IMAGES. ECHO IMAGES NOT AVAILABLE. WE CAN TAKE OUT THE IMAGES IF THE QUALITY IS NOT GOOD ENOUGH



Apical Ballooning



Mid Ventricular Ballooning

3. Discussion

Our patient had two different episodes of stress cardiomyopathy. Recurrence rate has been reported up to 11.4 percent in previous studies [5]. This case is unique as recurrence was at different anatomic locations i.e apex followed by the mid ventricular segments. This has been described only in case reports [4].

The pathophysiology of stress cardiomyopathy is still unknown. It seems to be multifactorial in origin. However, increase in catecholamine levels during the stressful situations seem to have a strong association with this condition. [6] Increased catecholamine levels have been seen at the time of presentation and remain elevated up to 7-9 days after stress cardiomyopathy. [10] However they are not the sole factors responsible for stress cardiomyopathy. Female preponderance of the condition is paradoxical as it has been found that males have higher levels of catecholamines as compared to females in mentally stressful conditions. [8] If catecholamine surge had been the only factor causing the stress cardiomyopathy then incidence of this condition should be either more in males or has an equal incidence.

Stress cardiomyopathy can be a protective mechanism for the myocardium. As a result of severe stress, there is increased production of nor-epinephrine from sympathetic nervous system and epinephrine from the adrenal medulla. Basilar myocardium has a predominance of beta 1 adrenergic receptors which are activated more potently by the norepinephrine coming from the sympathetic nervous system. Their activation has positive inotropic effects in basilar myocardium as the concentration of these receptors is more. On the other hand adrenal medulla released epinephrine has more potent effect on apical and mid ventricular regions of the ventricle where there are more beta 2 adrenergic receptors. Activation of these receptors have negative inotropic effects and is likely responsible for the ballooning of the ventricular wall in the regions these receptors are more abundantly found. [9] This explains the fact that our patient had both episodes in regions where there is increased concentration of beta 2 adrenergic receptors. However, underlying mechanism for non-simultaneous ballooning during different episodes is still not well understood.

In our case although the patient denied any subjective feeling of stress during the first episode, the fact that she was on stage could have caused the surge of catecholamine levels. Second episode was however unrelated to any preceding stress. Emotional and physical stress is found in 2/3rd of patients [7].

Genetic predisposition has also been postulated as a major contributing factor in the pathophysiology of stress cardiomyopathy. The mutation which has likely association with this condition is substitution of arginine for glycine at amino acid 389 (Gly 389 Arg) of beta 1 adrenergic receptors. [9] This results in excessive stimulation of beta 1 adrenergic receptors and as a result the cardiac protective mechanism in the beta 2 receptor predominant regions causes ballooning of apical and mid ventricular regions.

Non-simultaneous ballooning of different cardiac regions rules out some of the previously proposed underlying mechanisms of this condition including wrap-around LAD and coronary vasospasms. It also demonstrates that the hypothesis of different concentrations of same adrenergic fibers in different regions might not be true.

Our patient had a recurrence at mid ventricular site 2 years after the initial episode and that does not follow the pattern of having non-apical form of stress cardiomyopathy at younger age as it is possible that the recurrence has different underlying mechanism and thus may present differently as initial episodes.

Recurrence of stress cardiomyopathy can occur without any specific stressor as in our case. If a patient has a history of previous episode of stress cardiomyopathy, it becomes the top differential in case of a repeat episode of chest pain. Work up with TTE/CMR in carefully selected patients in whom there is no ST segment elevation and are hemodynamically stable can save them another cardiac catheterization procedure in case diagnosis is established based on these tests. This can avoid patients undergoing an invasive procedure and its associated risk factors.

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