

Case Report

Takotsubo Cardiomyopathy: A Recognizable Phenotype: Mid-Cavity Variant

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Abstract

Takotsubo cardiomyopathy is an important differential consideration when a patient presents with an acute coronary syndrome. Patients typically present with chest pain/pressure, shortness of breath, electrocardiographic changes, elevated biomarkers, normal or near normal coronary arteries, and wall motion abnormalities that are not consistent with a single coronary vascular bed. This constellation of findings is often associated with a precipitating emotional or physical stressor.

Variants of the classic left ventricular apical ballooning syndrome, including mid- and basal left ventricular wall motion abnormalities, are being more frequently recognized. The recognition and diagnosis of takotsubo cardiomyopathy has important implications for clinical management initially and in follow up. A small subset of patients may experience life-threatening complication during initial presentation, however, most patients recover and have a favorable long-term prognosis. The pathophysiology of takotsubo cardiomyopathy remains an enigma, but a surfeit of catecholamines seems pertinent to the underlying pathophysiology.

Keywords: Acute and long-term treatment; Acute coronary syndrome; Angiography; Electrocardiography; Mid-ventricular variant of takotsubo cardiomyopathy; Takotsubo cardiomyopathy phenotypes

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Introduction

Takotsubo Cardiomyopathy (TC) is a unique cardiomyopathy that is an important etiologic consideration in the differential diagnosis of acute coronary syndrome. The presenting symptoms and signs are similar to acute coronary syndrome after plaque rupture, but important distinctions include absence of coronary occlusion and wall motion abnormalities that do fit with a single vascular territory. TC usually has an emotional or physical stressor. The acute presentation of TC can include life-threatening hemodynamic compromise, but the long-term prognosis is better than the prognosis associated with acute coronary syndrome [1]. There are several morphological variants of TC that include apical segment, mid-ventricular, and basal involvement. Apical and mid segment involvement may occur together. Echocardiography is fundamental to identifying the various phenotypes of TC. The mid-ventricular variant of TC is characterized by transient hypokinesis, akinesis or dyskinesis of the left ventricular mid segments with normal to hyperkinesis of apical and basal segments. There is no acute plaque rupture. There are new electrocardiographic changes including: ST elevation and/or t wave inversion and modest elevation of cardiac biomarkers [2].

Case Report

59 year-old female presents with chest tightness and dyspnea. Symptoms occurred after a stressful conversation about the custody of her disabled 37-year-old son. On arrival to the emergency room she had an immediate ECG revealing t wave inversion V1-6 (Figure 1). This was compared to a normal ECG one year earlier (Figure 2).

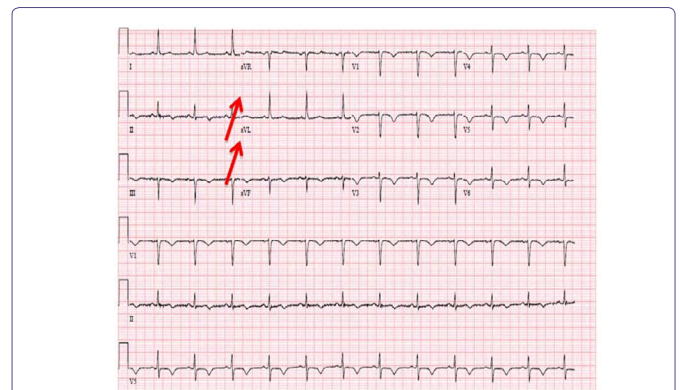


Figure 1: ECG revealing t wave inversion across the pericardium (Red arrows pointing to inverted t waves).

The initial troponin was elevated at 0.13ng/ml (< 0.05ng/ml). The patient was taken to the cardiac catheterization and found to have mild coronary artery disease manifest as a proximal 40% left anterior descending narrowing (Figure 3).

Contrast LV ventriculography did not reveal any segmental abnormalities (Figure 4). Post cardiac catheterization echocardiography revealed reduced Left Ventricular (LV) systolic function with mid LV

cavity segment hypokinesis with normal thickening of the apical and basal segments (Figure 5). The troponin level peaked at 0.38ng/ml (< 0.05ng/ml). The patient recovered in the hospital without hemodynamic or electrical disturbance.

A nuclear stress test revealed normal perfusion. The patient had all the classic features of mid-cavity variant of TS cardiomyopathy.

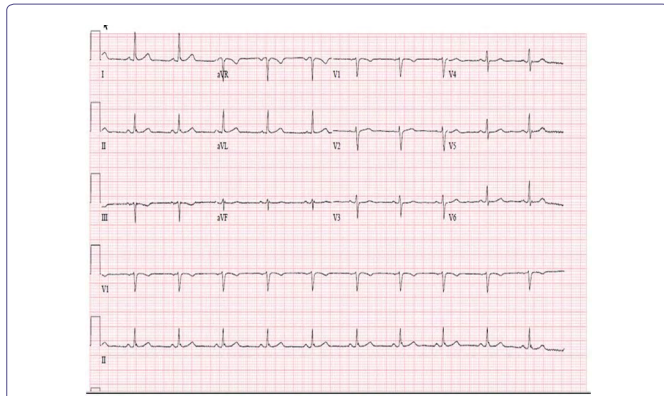


Figure 2: Normal ECG one year before when the patient was free of symptoms.

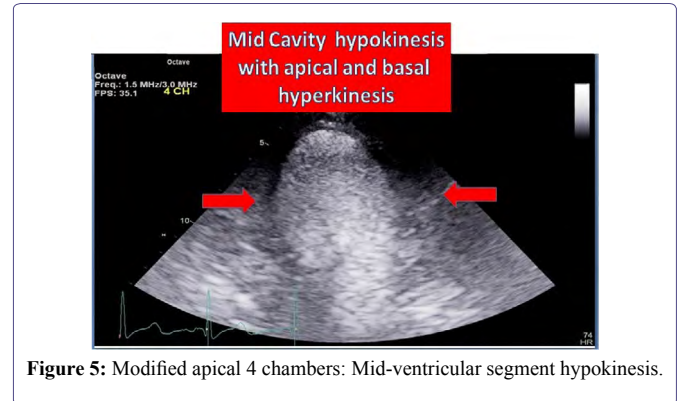


Figure 5: Modified apical 4 chambers: Mid-ventricular segment hypokinesis.

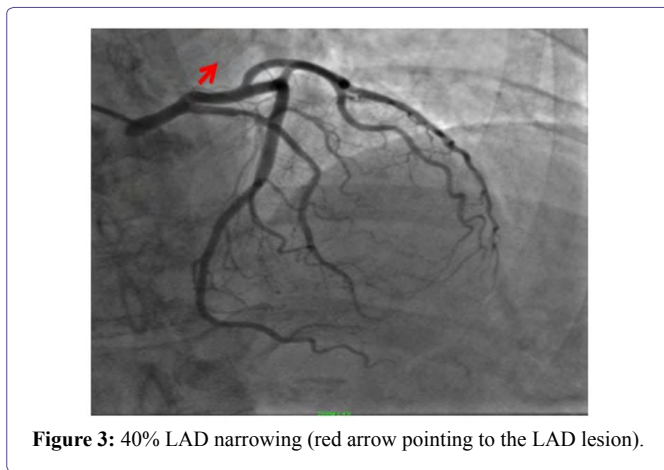


Figure 3: 40% LAD narrowing (red arrow pointing to the LAD lesion).

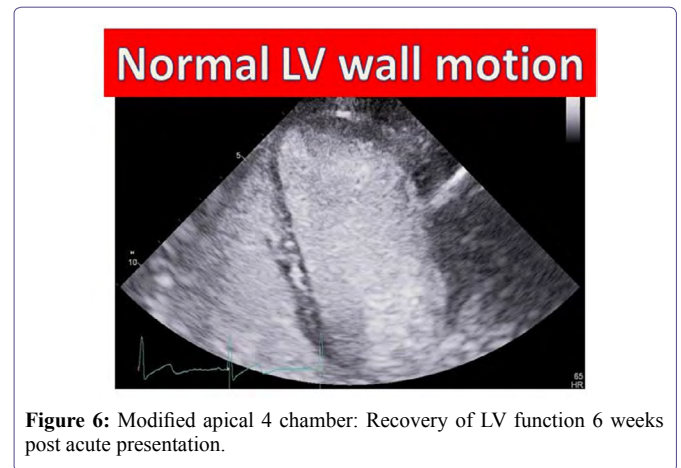


Figure 6: Modified apical 4 chamber: Recovery of LV function 6 weeks post acute presentation.

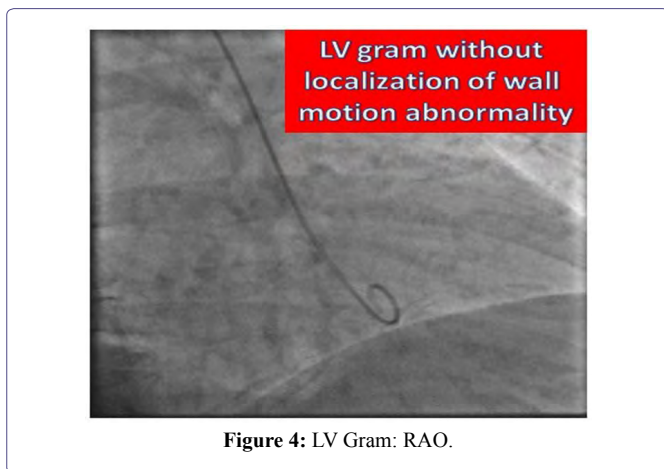


Figure 4: LV Gram: RAO.

The patient returned 6 weeks later and she was free of symptoms with robust activities. Echocardiography revealed normal LV systolic function with normal wall motion of all segments (Figure 6).

Classic Features of Takotsubo Cardiomyopathy

Presentation

Chest pain is the most common presenting symptom (80%). Dyspnea and pulmonary edema occur roughly 20% of the time. Cardiac arrest, cardiogenic shock, hemodynamically significant arrhythmias are infrequent [1]. An emotional or physical stressor is common. Common triggers include death of a loved one and anxiety and pain related to medical procedures and operations. Less common triggers include opiate withdrawal, cocaine use, dobutamine stress testing, intravenous ergonovine, and thyroid storm. TC should be considered in the differential thought process for postmenopausal women who present with symptoms of myocardial ischemia who experience emotional or physical stress.

Prevalence

More than 80% of TC cases occur in postmenopausal age 65-72 years. There is available data suggesting that 1/30 patients having cardiac catheterization have a variant of TC. The best estimates suggest that 0.7% to 2.2% of patients presenting with suspected acute coronary syndrome have symptoms and ECG findings consistent with stress induced cardiomyopathy [3]. Increased awareness and recognition may elevate the estimated prevalence.

Typically apical or mid-ventricular segment akinesia of the left ventricle (or both) is present. Also, not common is isolated mid-ventricular segment akinesia with apical sparing (Our case). It has been reported that as many as 40% of the TC cases are the mid-ventricular variant [4]. Basal akinesia with mid cavity and apical sparing has been identified [5]. There is no evidence identifying different pathophysiologic mechanisms for the phenotypic variant of TC.

Electrocardiography and coronary angiography

ECG findings in TC are variable with 1/3 of patients having ST elevation with the anterior leads being the most common involvement. Another frequent presentation is ECGs with deep t wave inversions and non-specific ST-T changes. Prolongation of the QT interval occurs, but torsade de pointe is rare. The ECG changes may be dependent upon the morphological variant of the presenting TC. Typically there is absence of obstructive coronary artery disease or acute plaque rupture identified at catheterization. Stable non-obstructive coronary artery disease may accompany TC syndrome. Left ventriculography may be helpful in identifying apical and basal variant of TC but seem less helpful with the mid-cavity variant.

Cardiac biomarkers

The biomarker troponin is typically elevated upon presentation with TC and is evidence of myonecrosis. Troponin typically peaks after 24 hours, but blood levels are lower than would be expected on the basis of ECG changes and the extent of wall motion abnormality. There are cases where myonecrosis is insignificant and cardiac magnetic resonance imaging reveals an absence of gadolinium hyperenhancement [6].

Treatment: Acute, Sub-acute and Long-term

Acute treatment

Patients with TC should be treated analogous to patient with acute coronary syndrome. The initial analysis should be coronary angiography and cardiac imaging. Imaging is critical in situations when fibrinolysis therapy is being considered with a ST elevation presentation. The differential diagnosis of hemodynamically significant hypotension in a TC patient is pump failure or dynamic outflow tract obstruction. It is important to recognize this differential and imaging will identify the etiology of the hemodynamic instability. Pump failure may require intravenous pressor support and mechanical support with an intra-aortic balloon. Alternatively, with outflow tract obstruction inotropic agents are contraindicated and treatment should include intravenous fluids, short acting beta-blockers, and peripheral vasoconstrictors such as phenylephrine are indicated [7]. Mechanical complications in TC, such as rupture, are rare [8]. Right ventricular involvement is TC occurs and involvement at presentation may predict adverse outcomes. Electrical complications such as atrial fibrillation, ventricular tachycardia, and ventricular fibrillations occur with TC and appear to be related to elevated catecholamine levels.

Sub-acute and long-term treatment

Only a small number of cases of TC-related death have been reported. Death prior to hospitalization is described but the incidence is unknown [9]. Complete recovery of left ventricular systolic function is essential to confirm the diagnosis. Recovery time is variable ranging from days to several weeks [1]. Medical treatment is empiric with

beta-blockers and angiotensin-converting enzyme inhibitors until left ventricular function recovers. Left ventricular thrombus has been reported [3]. Anticoagulation therapy must be individualized based upon the degree and duration of akinesia. Recurrence occurs in 10% of patients [10]. The long-term use of beta-blockers has not been studied to determine efficacy.

Conclusion

Mid-ventricular variant of takotsubo cardiomyopathy is a recognizable phenotype that requires a high index of clinical suspicion and meticulous echocardiographic imaging. Echocardiography is the imaging modality of choice to clearly delineate the mid cavity hypokinesis.

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