Takotsubo Cardiomyopathy Masquerading as Acute Coronary Syndrome

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Abstract

Takotsubo cardiomyopathy, also called transient left ventricular apical ballooning syndrome or Gebrochenes-Herz-Syndrome, is a rare entity which may masquerade as acute coronary syndrome (ACS) with sudden onset of chest pain and/or dyspnea, transient left ventricle apical akinesia, electrocardiographic changes typical of ACS and mild increase in cardiac enzymes in the background of normal coronary arteries.

Here, we present a case of a 45 year old female who presented with acute onset dyspnea and chest pain with severe ischemic changes on the ECG and raised troponin levels. She was treated with a provisional diagnosis of an ACS but on further workup was found to have Takotsubo cardiomyopathy. Her presentation to the hospital along with relevant clinical findings, electrocardiographic, echocardiographic and angiographic findings are presented in the article.

Introduction

Takotsubo cardiomyopathy, also called transient left ventricular apical ballooning syndrome, stress-induced cardiomyopathy, Gebrochenes-Herz syndrome consists of an acute onset of transient extensive akinesia of the apical and mid-portions of the left ventricle, producing ballooning of the apex in systole, in the absence of significant coronary artery disease. The syndrome is accompanied by angina-like chest pain, electrocardiographic changes and mild release of cardiac enzymes and biomarker levels, mimicking an acute myocardial infarction and is often preceded by an episode of emotional or physical stress, which may play a key role in the pathogenesis of the disorder.

It was first described by Satoh et al. and Dote et al. and was named “Takotsubo”-shaped cardiomyopathy due to its unique “short neck round-flask”-like LV apical ballooning resembling the Takotsubo (Japanese for octopus pot or trap) of Japan.¹

Case Report

A 45 year old female presented to the casualty with complaints of left sided chest pain with sudden onset dyspnea and one episode of vomiting around 2 hours ago. She had no history of hypertension, diabetes, ischemic heart disease, bronchial asthma or any other chronic comorbid conditions. She did not give any history of major stress or emotional breakdown in the past few days, but had some altercation with her brother a day prior to presentation.

On examination, the patient was conscious and cooperative. Pulse was 96 bpm, BP was 110/70mm of Hg, respiratory rate was 28/min and SpO2 was 94% on room air. Her jugular venous pressure was elevated. Bilateral basal crepitations up to mid-level were present on chest examination.

Cardiovascular examination revealed normal S₁ and S₂.

On investigations haemoglobin was 9.7 gm%. Serum electrolytes were also within normal levels. The creatinine level was 0.8 mg/dl.

ECG on presentation showed evidence of sinus rhythm with deep symmetrical T wave inversions suggestive of ischemia and a prolonged QTc (530 ms) (Figure 1).

2D-Echocardiography showed left ventricular ejection fraction of 25-30% with severe hypokinesia of the apical part of the left ventricle. There was mild dilatation of the apical part of the left ventricle (LV) (Figure 2).

Her cardiac enzymes values were raised with CPK – 162 mg/dl, CPK-MB – 24 mg/dl, and Troponin I – 0.75 ng/ml.

Based on the above findings, a provisional diagnosis of acute coronary syndrome (non-ST elevation myocardial infarction involving the anterior myocardium) was made. The patient was admitted to the intensive care unit and loaded with dual antiplatelets drugs and statins. She was also started on nitroglycerin infusion.

Fig. 1: ECG showing sinus rhythm with deep symmetrical T wave inversions with prolonged QTc (530 ms)

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low molecular weight heparin, diuretics and ivabradine.

Patient’s condition improved with the above line of treatment and she was also pain free. Subsequent ECGs on the following days showed deepening of the T wave inversions in anterior leads. The patient was planned for coronary angiography to evaluate coronary artery status and revascularization if needed on day 2 of hospitalization. However the patient developed hypotension on day 2 of hospitalization which responded well to intravenous colloids.

Coronary angiography on day 3 showed normal coronary arteries with right dominant coronary system (Figure 3). LV angiography showed LV apical hypokinesis with mild ballooning of the apex.

An impression of Tsakotsubo cardiomyopathy was made on the basis of clinical picture, relevant physical, investigational data and angiographic findings.

The patient was later treated with enalapril and a β-blocker. Subsequent stay in the hospital was uneventful. Patient was discharged on day 5. On discharge, patient was stable and asymptomatic.

Discussion

Tsakotsubo cardiomyopathy accounts for approximately 1% of admissions for suspected acute myocardial infarction. Most cases are reported in postmenopausal women aged 60–75 years. Acute emotional or physiological stressors generally precede symptoms, and the death of a close relative is the stressor reported most frequently. Given that Takotsubo syndrome is characteristically precipitated by emotional stress, elevated catecholamine levels might be useful in its diagnosis.

Several anatomic and physiologic factors might contribute to LV apical wall motion abnormalities: 1) the fact that the LV apex does not have a three-layered myocardial structure; 2) the easy loss of elasticity of LV apex after excessive expansion; 3) the fact that the LV apex is the border zone (locus minoris) of the perfusion area of major coronary arteries; and 4) the delay of functional recovery from global dysfunction. 1

Symptoms are in the form of precordial pain and dyspnea similar to the findings in the acute coronary syndrome.

It is precipitated by emotional or physical stress. There is a recognized tendency to a higher frequency in elderly individuals, principally women.

ECG typically shows ST elevation immediately after the event. T waves progressively become negative in various leads and the QT interval progressively lengthens. These changes gradually improve, but the T waves may remain negative for months.

Apical ballooning with rapid recovery may be noted on ventriculography and echocardiography.

There is a slight rise in the cardiac enzymes and troponin. Characteristically the clinical manifestations and ECG abnormalities are out of proportion to the degree of elevation of cardiac enzymes, but this difference cannot be used to exclude an acute coronary syndrome.

Coronary angiography shows normal or essentially normal coronary arteries.

Abnormalities may be detected on myocardial gamma scan in some cases. 3

Since differentiation between this cardiomyopathy and an acute coronary syndrome is often difficult at initial presentation, patients receive aspirin, β-blockers, angiotensin-converting-enzyme inhibitors, cardiac catheterization and intravenous diuretics if needed.

When a patient presents with hypotension, intravenous vasopressors might be required, and sometimes treatment with intra-aortic balloon pumps and left ventricular assist devices is necessary. Those with hypotension should be evaluated by echocardiography or cardiac catheterization for an intra-cavitary gradient, which maybe exacerbated by dobutamine and may result in a dynamic left ventricular mid-cavity obstruction. If a dynamic intraventricular pressure gradient is detected, inotropic drug therapy should be discontinued and intravenous β-blockers administered to increase diastolic filling time and left ventricular end-diastolic volume. 4 Supportive treatment leads to spontaneous rapid recovery in nearly all patients. The prognosis is excellent, and a recurrence occurs in <10% of patients. 5

Takotsubo cardiomyopathy can be misdiagnosed and treated as an acute myocardial infarction (AMI). The accompanying risk of treating
such patients with antiplatelets, anticoagulants and thrombolytics must be appreciated. An early coronary angiography may rule out an AMI. In the absence of critical coronary artery disease, the diagnosis of stress cardiomyopathy should be considered when the history reveals the presence of trigger like intense emotional stress and identification of an unique pattern of left ventricular dysfunction characterized by apical and midventricular contractile abnormalities with sparing of the basal segments and also when there is minimal elevation of cardiac enzymes despite the presence of large regions of focal akinesis in the myocardium. Because massive catecholamine release results in stress-induced myocardial stunning, we avoid using pressors and beta-agonists whenever possible and instead rely on mechanical circulatory support in patients with severe hemodynamic compromise.

References