CASE REPORT

Stress-induced cardiomyopathy (Tako-Tsubo) in a premenopausal woman: A case report from Saudi Arabia

Abdulrahman Al-Moghairi *, Ziad Al-Harfi, Sadiq Al-Shouli

PSCC, Adult Cardiology, P.O. Box 27656, Riyadh 11427, Saudi Arabia

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Abstract
Tako-Tsubo cardiomyopathy (TTC) is a nonischemic cardiomyopathy characterized by reversible left ventricular dysfunction that is seen predominantly in postmenopausal women (> 80%). The syndrome has symptoms that are similar to acute myocardial infarction, such as electrocardiogram changes (ST-segment elevation and subsequent giant T wave inversion) and abnormal cardiac enzymes. The clinical prognosis is usually benign. This article reports a first case of a TTC in premenopausal Saudi woman. Early diagnosis of TTC excludes the use of stents, thrombolytics, and long-term coronary heart disease medications.

1. Introduction
Tako-Tsubo cardiomyopathy (TTC) is a potentially life-threatening cardiac syndrome characterized by transient left ventricular dysfunction, without angiographically significant coronary artery stenosis (Dote et al., 1991). TTC classically represents signs and symptoms similar to those of an acute coronary syndrome (ACS), with the onset of symptoms typically provoked by an intense emotional or physical event. It accounts for about 1–2% of all patients with apparent manifestation of acute myocardial infarction (Desmet et al., 2003; Gianni et al., 2006).

We believe that it still remains underreported and goes unrecognized in countries like Saudi Arabia, especially in premenopausal women. We report a case of TTC in a young Saudi female presented to our emergency department with acute chest pain mimicking ST elevation myocardial infarction preceded by history of psychological stress.

2. Case report
A 38 year old Saudi woman, housewife and mother of six children came to emergency department after a sudden onset of severe retro-sternal crushing chest pain at rest radiating up through the neck and left arm which lasted for one hour. This was accompanied by dizziness, restlessness, distress, breathlessness and profuse sweating. Nausea and vomiting were
The patient’s history had no attributable cardiovascular risk factors (smoking, diabetes, or hypertension) and no family history of coronary artery disease or premature cardiac events. However, family history of depression was positive. She was always distressed worrying about her children’s future.

At admission, blood pressure was 85/50 mm Hg, pulse rate was 100 beats per minute. Jugular veins were not distended. First and second heart sounds were normal, while third heart sound was audible and there were no audible lung rales. Electrocardiogram (ECG) showed sinus rhythm with ST elevation in leads V1–V4 (Fig. 1), and myocardial enzyme assays revealed elevated cardiac troponin I (7.26 ng/ml) with normal total creatine kinase and CKMB levels. The patient was immediately taken for coronary angiography, which showed no epicardial coronary artery disease and LV angiography was not performed. The echocardiogram (Fig. 2a) showed hypokinetic anterior basal septum and akinetic distal anterior wall and apex with apical dilation, moderate LV systolic dysfunction with preserved wall thickness, normal right ventricular size and function, normal valves, and no pericardial effusion. The patient was treated medically with aspirin, heparin, beta-blockade, and angiotensin-converting enzyme inhibitors. During her ten day stay in the hospital, she had a short run of nonsustained monomorphic ventricular tachycardia which was not hemodynamically destabilizing. Hence, psychiatric consultation was requested where the diagnosis of depression with panic attack was established and treatment with citalopram (20 mg) commenced. She showed a substantial symptomatic improvement since then.

She was seen at one and three months follow-up, with marked clinical improvement, her ECG revealed sinus rhythm with T-wave inversions in leads V1–V4. Repeat echocardiogram (Fig. 2b) showed normal regional wall motion abnormality and normalized apical shape. Other laboratory tests were normal including autoimmune screens, and thrombophilia profile.

Follow-up at one year and two years showed that she was asymptomatic with normal ECG and echocardiography. She continued taking aspirin 81 mg, bisoprolol 5 mg, and citalopram 20 mg daily.

3. Discussion

TTC is characterized by acute-onset, transient, and abnormal left ventricular wall motion with apical akinesis and basal normokinesis without any detectable coronary stenosis. Although, originally thought to mainly affect the Japanese and Caucasian population, cases have been reported from diverse ethnic background from all over the world (Donohue and Movahed, 2005; Sharkey et al., 2005; Hertting et al., 2006). The true incidence of TTC is probably higher than currently estimated, as it remains unrecognized and underreported in many centers. Universal diagnostic criteria for transient TTC syndrome have not been established. The clinical presentation varies from mild symptoms to severe cardiogenic shock, with the predominance of chest pain in most reports (Bybee et al., 2004). In the present case, initial diagnosis with ST elevation, hypokinetic anterior basal septum and akinetic distal anterior wall and apex with apical dilation, moderate LV systolic dysfunction in the absence of head trauma, intracranial hemorrhages, pheochromocytoma, or myocarditis.

However, specific ECG changes of TTC described by Ogura and colleagues include ST elevation in precordial leads, as in our case, with the absence of reciprocal changes and abnormal Q waves and they postulated that these changes might be explained by the fact that myocardial dysfunction is confined to the apical wall (Ogura et al., 2003). The moderate rise in
cardiac troponin, as in the present case, along with the absence of atherosclerotic coronary artery disease suggests little myocardial injury and rapid normalization of LV function for favorable prognosis (Akashi et al., 2005).

The treatment of TTC is generally supportive in nature and based on the patient’s overall condition. No controlled studies have provided data that define optimal medical management, but reasonable treatment, which we also followed includes standard therapy for left ventricular dysfunction including antiplatelet agents, angiotensin-converting enzyme inhibitors, and beta blockers to prevent excessive sympathetic activity (Buchholz and Rudan, 2007). In addition, the patient also received citalopram for the treatment of depression with panic attack which was diagnosed during her stay in the hospital. Anti-coagulant medications may be considered to prevent apical thrombus formation (Buchholz and Rudan, 2007).

Although complete recovery in most patients occurs during the first 3–4 weeks post event, this syndrome is not exclusive of complications like left heart failure with or without pulmonary edema, cardiogenic shock, tachy- and bradyarrhythmias, LV thrombus formation or free wall rupture, and death (Donohue and Movahed, 2005; Gianni et al., 2006). Sharkey et al. (2005) reported the recurrence of the syndrome in 2 out of 22 patients after re-exposure to the psychogenic stress within 10 months (Sharkey et al., 2005).

Although predominantly described in postmenopausal women, reports of this entity in younger adults and children exist in the literature (Gianni et al., 2006; Maruyama et al., 2006) as highlighted in the present case.

In summary, this sort of cardiomyopathy has yet to achieve recognition within the greater physician community in most parts of the world; the present case of TTC described herein, to our knowledge, was not reported in Saudi Arabia before and will be of general medical interest.

References


