

A case of takotsubo cardiomyopathy

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Transient left ventricular apical ballooning syndrome, also known as takotsubo cardiomyopathy, is a recently recognized entity originally reported in Japan. We are reporting a case of takotsubo cardiomyopathy who presented to our institution with acute chest pain after voting for the 2004 United States presidential election.

Key words: Takotsubo syndrome – Chest pain – Myocardial infarction

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Prechodný syndróm apikálneho vydutia ľavej komory, tiež nazývaný takotsubo kardiomyopatia, je nedávno zistená entita, a bola opísaná v Japonsku. Referujeme o prípade pacienta s takotsubo kardiomyopatiou, ktorý prišiel na našu kliniku s akútnou bolesťou na hrudi, po prezidentských voľbách v roku 2004 v USA.

Kľúčové slová: takotsubo syndrome – bolesť na hrudi – infarkt myokardu

A 72-year-old white woman presented to the emergency room eight hours after the acute onset of chest pain. Symptoms started after voting for the 2004 United States presidential election and then waxed and waned in intensity over the course. Chest pain was associated with nausea, diaphoresis, and shortness of breath. She denied tobacco, alcohol, or drug use. Her past medical history was significant for hypertension and hyperlipidemia for which she had been taking enalapril and atorvastatin. On arrival, blood pressure was 124/64 mmHg and heart rate was 80 beats/min. There was no jugular venous distension at a 30° angle, and carotid upstrokes were normal. On cardiac auscultation no murmurs, rubs, or gallops were appreciated. Respiratory crackles were heard in bilateral lung fields. The electrocardiogram revealed mild ST elevation and Q-waves in leads III, V1 to V3 with QT prolongation (**Figure 1-A**). A chest radiograph was consistent with mild bilateral pulmonary parenchymal congestion. Emergent coronary angiography revealed normal coronary arteries. Left ventriculography showed distal anterior, apical and distal inferior akinesis with hyperkinesis of the remaining walls (**Figure 2**). Peak serum troponin I was mildly elevated at 1.34ng/ml, although the serum creatine kinase remained within normal limits with

normal subfractions. Over the next two days, she showed a marked clinical improvement with resolution of heart failure. Repeat electrocardiogram on hospital day 3 showed deep T-wave inversion with persistent ST elevation, Q-waves, and QT prolongation in leads V1 to V5 (**Figure 1-B**). She was discharged on hospital day 5 on medical therapy including an ACE inhibitor, a beta blocker, and warfarin. Electrocardiogram three months later showed disappearance of Q-waves and R-wave reappearance in the anterior precordial leads (**Figure 1-C**). Follow-up echocardiography revealed normalization of left ventricular wall motion.

Discussion

Takotsubo cardiomyopathy was first recognized and described in the Japanese literature in 1990 by Satoh et al., who proposed the term “takotsubo” (Japanese for octopus trap) cardiomyopathy for its unique appearance of a narrow neck and rounded bottom on the end-systolic left ventriculography (1). Since Tsuchihashi et al. reported their largest case series with 88 patients, awareness and clinical interest in this unique syndrome have been raised globally (2). More recently, this syndrome has also been called left ventricular apical ballooning syndrome and several case series have been reported in the United States and across Europe over the last three years (3 – 6). Clinical manifestation of takotsubo cardiomyopathy is characterized by: 1. reversible balloon-like left ventricular apical wall motion abnormality with hy-

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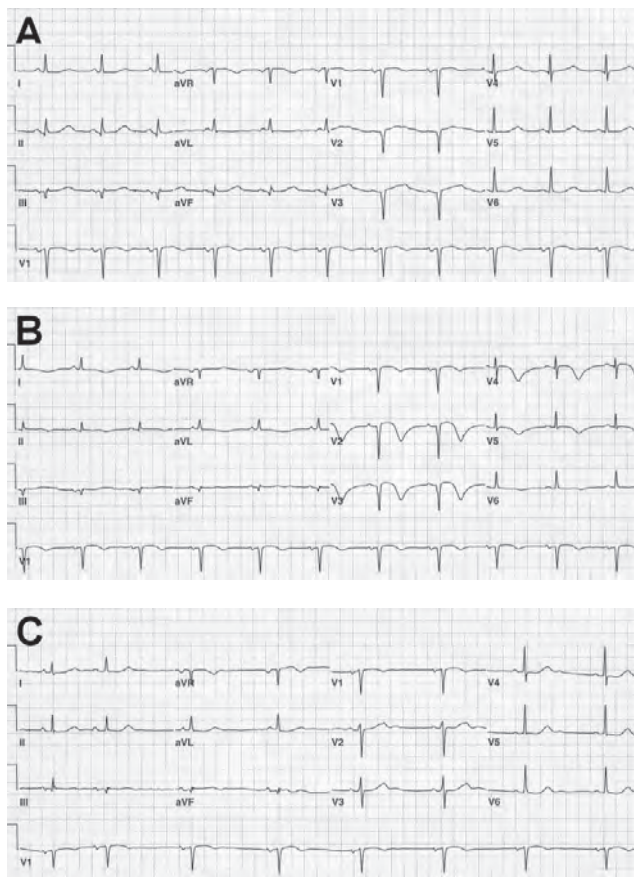


Figure 1 (A) Electrocardiography on presentation to emergency department demonstrating mild ST elevation and Q-waves in leads III, V1 to V3 with QT prolongation. (B) Repeat electrocardiogram on hospital day 3 revealing deep evolutionary T-wave inversion with persistent ST elevation, Q-waves, and QT prolongation in leads V1 to V5. (C) Follow-up electrocardiogram three months later demonstrating disappearance of Q-waves and R-wave reappearance in the anterior precordial leads.

percontraction of the basal segments; 2. chest pain and ST-T segment abnormalities on electrocardiogram mimicking acute myocardial infarction; 3. minimal evidence of epicardial coronary artery obstruction; 4. limited myocardial enzyme release relative to the extent of wall motion abnormalities; 5. an induction by physical or emotional stress in most cases; and 6. having a favorable prognosis. In this report we described a case of takotsubo cardiomyopathy, with typical clinical presentation and course, among thirteen cases we have experienced in our institution over the last two years. The electrocardiographic pattern observed in this case, such as ST elevation in precordial leads and QT prolongation followed by the evolutionary T-wave inversion, is the most common pattern previously reported in the several case series (2–6). Q-wave regression, which is known to be associated with small infarcts and stunned/hibernating viable myocar-

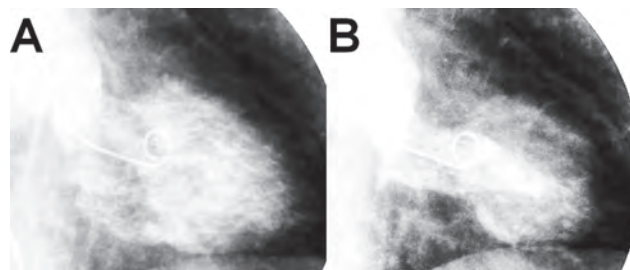


Figure 2 Left ventriculography during diastole (A) and systole (B) showing distal anterior, apical and distal inferior akinesis with hyperkinesis of the remaining walls

dium, is another finding frequently observed in this syndrome (2, 6). In one case series only nine out of twenty-four patients, who initially presented with pathologic Q-waves, had persistent Q-waves in the subacute period (2).

Although there is no consensus as to the mechanism responsible for transient ballooning, catecholamine toxicity has been proposed as the most likely cause of myocardial stunning in this syndrome (7). One of the possible mechanisms contributing to the focal wall motion abnormalities is the intense activation of the sympathetic nervous system, characterized by massive secretion of catecholamines from the terminals of sympathetic nerves in tissue. The high concentration of catecholamines in the myocardium would lead to intracellular calcium overload which, in turn, causes a reduction of myocardial contractility (8). Adrenoceptor density varies throughout the heart with greater density in the apex, possibly explaining the vulnerability of this territory to the sympathetic surge (9).

The prognosis of takotsubo cardiomyopathy tends to be benign. In their case series, Tsuchihashi et al. reported an in-hospital mortality rate of 1% (2). However, serious complications including cardiogenic shock due to left heart failure and/or intraventricular obstruction, ventricular arrhythmias, left ventricular mural thrombus formation, and left ventricular free-wall rupture have been reported. Prompt recognition of this syndrome and aggressive management with pharmacologic agents and, if indicated, mechanical hemodynamic support are crucial to achieving favorable prognosis of this syndrome.

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