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Takotsubo Syndrome: A Case Report and Review

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Background*: Takotsubo syndrome is a reversible condition characterized by transient left ventricular dysfunction, elevation of cardiac enzymes and no coronary obstructions. It usually occurs after physical or emotional stress, predominantly in postmenopausal women.*
Case summary*: a 50-year-old-woman was accepted in the emergency room with chest pain at rest (about 3 hours) with irradiation to the back. Admission ECG showed ST elevation in anteroseptal wall**.** Troponin I was high. She was submitted to cardiac catheterization, which revealed no arterial obstruction, apical hypokinesis suggesting Takotsubo syndrome.*
Conclusion: *Takotsubo syndrome (TTS)is an acquired cardiomyopathy, and the clinical presentation is similar to acute myocardial infarction (chest pain), electrocardiographic changes, elevated cardiac biomarkers. Many pathophysiological mechanisms have been proposed for this syndrome but is still unknown. Further randomized studies are needed to elucidate this condition.*
Key words*: acute coronary syndrome; left ventricular dysfunction; Takotsubo syndrome.*

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I. Case Presentation

A 50-year-old lady was admitted at the Emergency Room with intense chest pain at rest (about 3 hours) with irradiation to the back. She denied nausea, sweating, headache, dizziness, and dyspnoea. No smoker. No prior cardiovascular events. Hypertensive using only Losartan 100mg/d. Family history was positive for hypertension (mother), but further details were not available.

Physical exam was normal. BP: 114/76 mmHg.SO2: 98%. Admission ECG showed sinus rhythm and ST elevation in anteroseptal wall (**Figure 1**). Lab: Troponin I 5.3 ng/mL. Patient was submitted to thrombolysis with alteplase 100mg (15mg+50mg+35mg) + DAPT + beta-blocker + nitrate + statin.

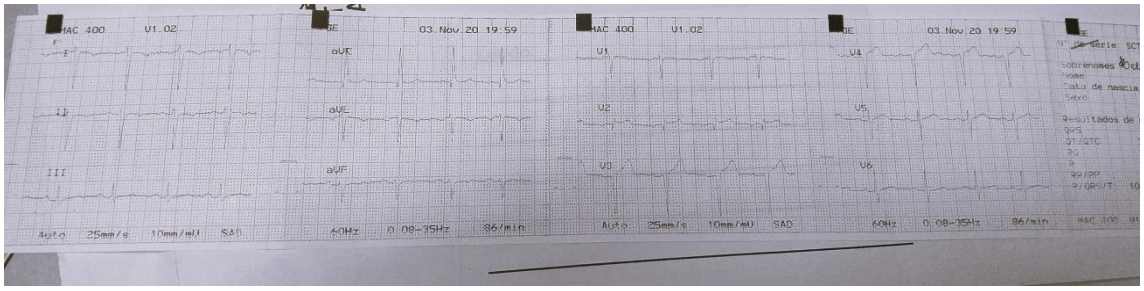

Figure 1. ECG showing ST elevation – anteroseptal MI

Twenty-four hours later, she was submitted to heart catheterization, which revealed no arterial obstruction (non-CAD), ventricular function preserved, apical hypokinesis suggesting Takotsubo syndrome (F**igure 2)**.

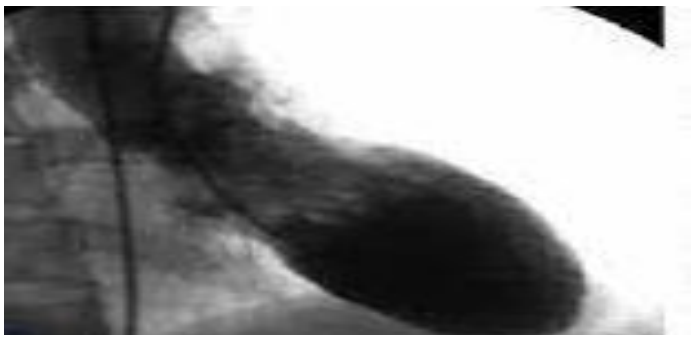

Figure 2. Heart Catheterization showing "apical ballooning – Takotsubo syndrome.

Patient was hospitalized in Coronary Unit (CU). She evolved into bradycardia, hypotension, undergoing temporary pacemaker implantation. About 24 hours later, there was haemodynamic stabilization, and the pacemaker was removed. She remained in CU for three days, and then transferred to private room. After 7 days, she was submitted to Transthoracic Echocardiography which showed normal wall thickness, normal global and segmental ventricular function (systolic and diastolic). EF: 70%. Chest X-Ray was normal. ECG: unspecific alterations in ventricular depolarization. Patient was discharged from Hospital asymptomatic after 15 days. Currently in use of Losartan 100mg/d and ASA 81mg/d.

II. Discussion:

Takotsubo syndrome (TTS)is an acquired cardiomyopathy and the clinical presentation is similar to acute myocardial infarction (chest pain), electrocardiographic changes, elevated cardiac biomarkers (Troponin and creatin kinase elevation) and left ventricular wall motion abnormalities ("apical ballooning"), being indistinguishable from acute myocardial infarction (AMI), except for the lack of atherosclerotic plaque rupture and coronary thrombosis, with complete recovery of ventricular systolic function. [1,2]

TTS affects predominantly postmenopausal women, but can also occur in men, children and in young women. It characterizes by an acute onset of left ventricular dysfunction, most often triggered by a physical or an emotional stress. Unfortunately, there is no established guidelines concerning its aetiology (including ESC guidelines for acute and chronic Heart Failure)[3], being listed as "unclassified" cardiomyopathy. According to International Takotsubo Registry (InterTAK), more than half of the patients had an acute, chronic or prior neurological or psychiatric disorder.[2,4] (Table 1)

Table 1. InterTAK diagnostic score

Clinical aspects	Points
Female	25
Emocional trigger	24
Physical trigger	13
Absence of ST depression	12
Psychiatric disorders	11
Neurological disorders	9
QTc prolongation on ECG	6
Probability of TTS:	
Less than 1% with score of $\geq$ 30.	
Approximately 90% with score $\geq$ 70.	

The incidence of TTS has been estimated about 3-5% of all patients with the diagnosis of acute coronary syndrome. Women are predominantly affected and account for 90% cases, approximately. The course of the disease is benign, with mortality rate of 2%. The presence of hidden critical illness is a contributor for its mortality.[4,5]

The pathophysiology is unclear, but some mechanisms have been proposed from its prior presentation. Most patients with TTS have normal coronary arteries at angiography, but abnormal coronary flow, due to acute microvascular dysfunction, suggesting a vascular dysfunction caused by catecholamines and/or a direct myocyte damage [4,6,7]. Catecholamine excess cause cardiac toxicity and can form reactive species with direct effects on membranes, in addition to an increased sympathetic activity, causing vasoconstriction and, subsequently microvascular dysfunction (microvascular spasm).[1,4] The reversibility of microvascular dysfunction added to the activation of the cell survival guarantees complete functional recovery in the majority of patients. Typically, myocardial dysfunction may involve the left ventricular apex or midventricular segment, and sometimes the bases [7]. Most patients have akinesis or hypokinesis of the apical and midventricular segments ("apical ballooning") on the left ventricle. Microcirculatory impairment tends to resolve over time during the natural course of the disease. Coronary flow reserve (CFR), evaluated by transthoracic echocardiography increases overtime, showing a progressive normalization at 3 or 4 weeks.[8,9]

Management of TTS includes pharmacological treatment, such as beta-blockers and ACEIs/ARBs are mostly administered to patients. Also, aspirin, statin and nitrates do not prevent TTS recurrence, but should be administered as indicated for other associated conditions. Although TTS is a disease increasingly under investigation, its pathophysiology is still largely unknown, as well as treatment and prognosis. Further randomized studies are needed to fully elucidate this disease.[10,11]

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