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Case Report

Tako-Tsubo Syndrome: Atypical Nuclear Medicine Findings

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Abstract

The Tako-Tsubo syndrome is a reversible form of an acute stress-related cardiomyopathy that was reported during the last decade. It typically presents with a constellation of symptoms, electrocardiographic changes, and elevated cardiac enzyme levels consistent with an acute coronary syndrome. However, when the patient undergoes cardiac angiography, left ventricular apical ballooning finding is seen, but no significant coronary artery stenosis. This balloon-like morphology, being the hallmark of this entity, can be detected by imaging. We present a case report of a patient who was admitted to our hospital and met all the diagnostic criteria of the Tako-Tsubo syndrome. Myocardial perfusion imaging (MPI) showed an anteroapical perfusion defect at rest, moderated systolic dysfunction, and intraventricular asynchrony all assessed by gated-SPECT phase analysis. Two months later, all MPI findings returned to normal parameters.

Keywords: Broken heart syndrome, left ventricular apical ballooning, left ventricle dysfunction, Tako-Tsubo cardiomyopathy .

Introduction

In 1990, Sato *et al*.[1] described in Japan a new entity with symptoms similar to the acute coronary syndrome. There are similarities in clinical manifestation, electrocardiogram changes, and biomarkers levels. The Tako-Tsubo syndrome received its name from of the similarity between the shape of the left ventricle in systole and the form of the Japanese octopus trap with a narrow neck and wide base [Figure 1] Other names are "stress-induced cardiomyopathy," "heart break syndrome" and "transient ventricular ballooning syndrome". The typical feature is apical ballooning with compensatory hyperkinetic basal segments. This cardiomyopathy represents 1-2% of patients with acute coronary syndrome. It is frequently found in females most commonly postmenopausal; men are affected in less than 10% of cases.

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The pathophysiology has been uncertain up to now, although several theories have been discussed, for example: coronary vasospasm, myocarditis and coronary microcirculatory disturbances. Nevertheless the most accepted is an increase in the sympathetic activity because a close relationship has been noted between physical and/or emotional stress precipitating the acute event. To make the diagnosis a coronary angiography must show normal arteries without any stenosis or obstruction.[2-6] Complications such as cardiogenic shock and heart failure are unusual.[2] Recurrences and death during 2 years of follow-up are very low (less than 2%).[2] Left ventricular function improves in the majority of patients within 1-3 weeks.[4]

Case Report

We report a case of a 46-year-old black woman with the following risk factors: tobacco, high blood pressure (HBP) and family history of coronary artery disease. She was admitted in our Emergency Care Unit (ECU) suffering from sharp chest pain irradiating to her neck and left arm, associated with a brief loss of consciousness and vegetative manifestations. The physical examination was normal. Electrocardiogram (ECG) on admission at less than 1 hour of the onset showed sinusal rhythm, heart rate 100/min,

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ST segment elevation of 2 mm in V2-V3 and 1 mm in DII, DIII and aVF with inverted T-waves deep and symmetric in V4-V6 and QT interval within normal limits [Figure 2a]. The cardiac biomarkers of heart damage (creatine kinase CK and its isoenzyme CK-MB) were only very slightly elevated in serial measures. Echocardiography revealed a reduction of the left ventricular ejection fraction (LVEF), akinesis of the apical and distal anterior wall with hypercontractility of the basal wall producing the apical ballooning configuration of the LV in systole, typical of this disorder [Figure 3a-b]. The patient underwent emergency cardiac catheterization, which disclosed no significant epicardial coronary artery stenosis [Figure 4].

Two days after the onset, myocardial perfusion imaging (MPI) with Tc^{99} MIBI was performed. Perfusion abnormalities in the left ventricular apex, lateral and anterior walls were documented [Figure 5a-c]. Thickening and contractility were reduced in these segments [Figure 6a-c].

The analysis of ventricular synchronism was done with gated-SPECT phase analysis using the Emory Cardiac Toolbox. There was an increase in peak phase degree (225), phase SD degree (49.1), and in the phase histogram bandwidth degree (150) [Figure 7a]. Intraventricular asynchrony assessed by gated-SPECT phase analysis was an unexpected finding.

During the patient´s evolution no other complications appeared. All of the ECG abnormalities returned to the baseline in 7 days [Figure 2b] there was a total recovery in global and regional left ventricular function, evaluated by echocardiography [Figure 3c]. Perfusion defects and synchronism also disappeared in the same period, assessed by nuclear medicine. [Figure 5b, d and Figure 7b].

Discussion

The first case of Tako-Tsubo syndrome was reported in 2001,[2] when its diagnostic criteria were established. It is important to point out that though there are prevailing incidences in elderly females, it is unusual before the menopause.[2-6] This was not our case. Several series in white populations of Europe, the United States and other countries have been reported, which demonstrate that this disease is not limited to specific ethnic groups or geographic zones.[5,6,8] The first case in Latin America was published in 2001;[9] three years later the first case in Cuba was reported, which was also a black woman.[10]

Thoracic pain and acute cardiac failure are frequent forms of presentation [7]; but syncope, as the first manifestation,

Figure 1: The Japanese octopus trap

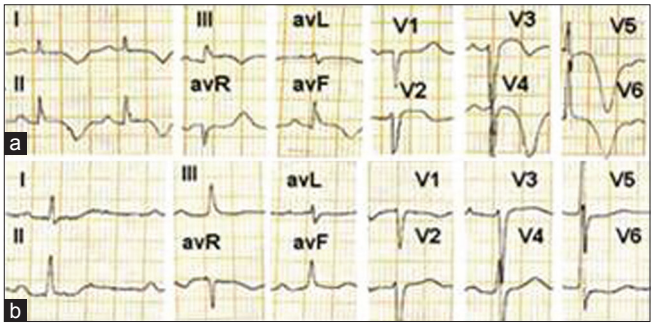

Figure 2: A 12-lead electrocardiogram (ECG) (a) shows ST-segment elevation and deep T-wave inversions in the V2-V3 and DII, DIII and aVF too in the acute onset. (b) Two months later

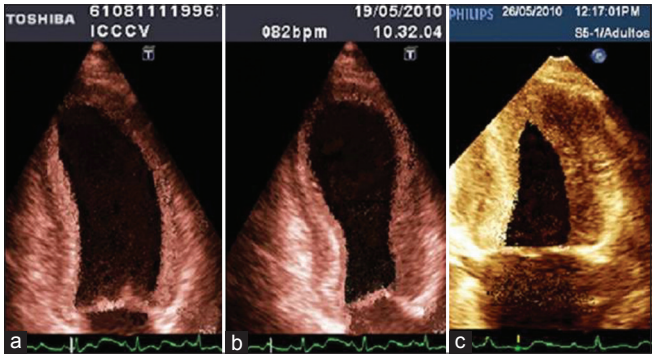

Figure 3: Echocardiography images (Two-chamber long axis) (a-b) Acute onset (a) End-diastole (b) End-systole (c) Two months later

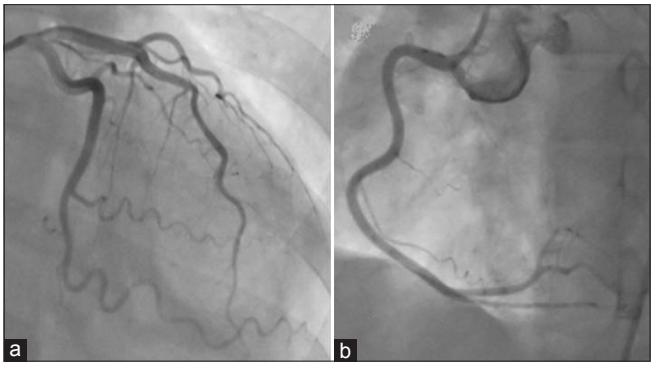

Figure 4: Coronary angiography (a) Left coronary (b) Right coronary. There is no epicardial coronary stenosis

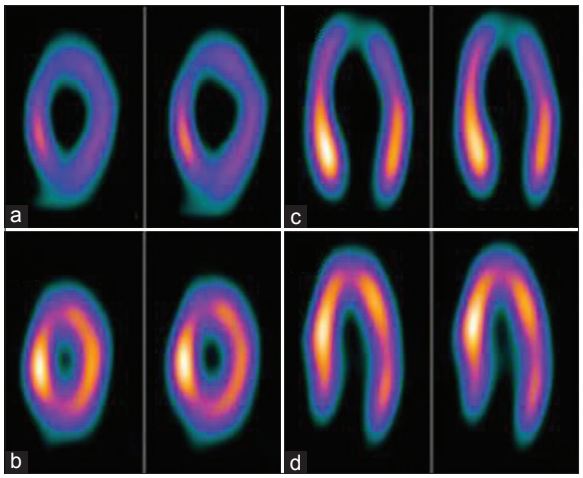

Figure 5: Tc99 MIBI-SPECT images. Early images (a, c) show decreased myocardial perfusion in distal anterior and lateral wall, and apex. (b, d) after complete recovery

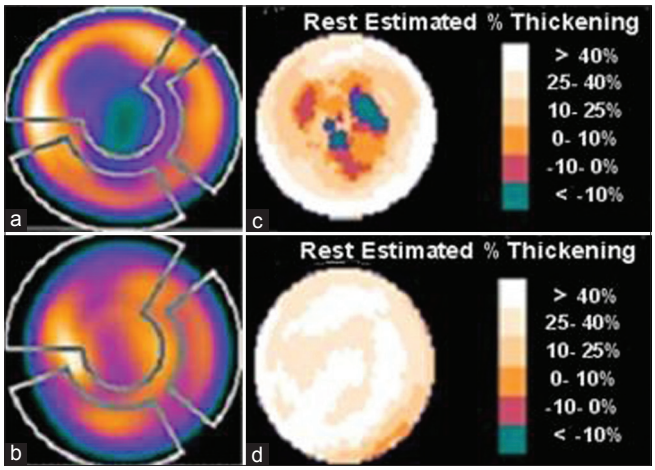

Figure 6: Bull-eye and thickening images. (a-c) Acute once. (b-d) Two months later

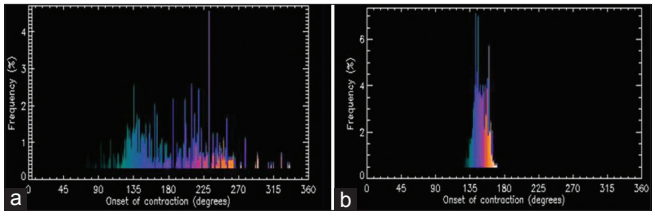

Figure 7: Gated-SPECT. Synchronism, (a) Acute onset. (b) Two months later

has been reported in a few cases.[2,5] The ST elevation in left precordial leads in ECG is a typical finding. Negative, symmetric and deep T waves, associated with the QTc interval's prolongation, are usually signs found after the second day of the onset of the symptoms.[4,5] In our case, the ST segment elevation in DII, DIII and aVF and T wave inversion in multiple derivations were found from the beginning of the symptoms [Figure 2]. This ECG pattern has been rarely described in the series of patients reported in the literature.

MPI was performed within 2 days of admission Tc^{99} MIBI uptake was decreased in more than one coronary vascular bed. The perfusion defects involved the apex and segments of the anterior and lateral walls. The regional wall motion and the systolic thickening in these segments were affected, showing an important area of akinesia. The ejection fraction at rest was moderately diminished (39%).

Tc uptake by myocytes is via a metabolism-dependent process located in the mitochondria and any mitochondria damage reduces the uptake, even when this damage could be reversible. Different authors consider that any metabolic and/or structural defects can cause scintigraphic abnormalities rather than perfusion changes due to coronary disease.[11]

It has been suggested that autonomic imbalance is related to the development of this condition. The catecholamine-associated myocardial toxicity is the most attractive theory due to elevated catecholamine levels that have been reported in patients with Tako-Tsubo cardiomyopathy.[12-16] The contraction band necrosis (which is characteristic of catecholamine-induced myocyte injury) found upon histological investigation[17] supports this "sympathoexcitation" hypothesis.

The analysis of ventricular synchronism by gated-SPECT phase analysis showed an intraventricular asynchrony, which might reflect the wall-motion abnormalities or perhaps left ventricular conduction system damage mainly in Purkinje fibers caused by catecholamine-related toxicity.[123] Iodine-metaiodobenzylguanidine (^{123}I-MIBG) uptake in cardiac sympathetic nerves is decreased in patients with stress cardiomyopathy, early images showed decreased myocardial uptake in the distal anterior wall, apex, and inferior wall of the left ventricle, suggesting cardiac sympathetic nerve affectation.[18]

A follow-up SPECT study was obtained 2 months after the onset of symptoms, and perfusion and function abnormalities returned to normal parameters.

The diagnostic criteria for this entity were found in spite of the atypical findings described before. But is very interesting this ventricular dyssynchronism, related to the contractility abnormalities, damage on Purkinje fibers very sensible to oxygen deprive or perhaps the effect of the decrease in sympathetic innervations could be the cause of this asynchronism detected.

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