

## Case Report

# Transient Ventricular Dysfunction (Takotsubo Cardiomyopathy)

José Marconi Almeida de Sousa, Marcos Knobel, Gustavo Buchelle,  
José Augusto M. de Sousa, Cláudio H. Fisher, Daniel Born, Nelson Akamine, Elias Knobel  
São Paulo, SP - Brazil

*The patient was a male with myasthenia gravis, hospitalized with acute respiratory failure due to decompensation of the underlying disease. He evolved with findings suggestive of acute myocardial infarction, with electrocardiographic and enzymatic alterations compatible with that diagnosis. The patient underwent emergency coronary angiography, which showed no severe coronary obstruction, although his left ventricle had significant systolic dysfunction with characteristic alterations, on ventriculography, of the syndrome described as transient ventricular dysfunction or Takotsubo syndrome. On evolution, complete recovery of the electrocardiographic alterations and systolic ventricular function assessed on echocardiography occurred, confirming the syndrome.*

Left ventricular systolic dysfunction is the final result of any pathological process affecting the heart in its many structures. It often results from primary muscular impairment with consequent heart failure. In this situation, myocardial damage is usually irreversible and prolonged. In some clinical situations, reversible ventricular systolic dysfunction has been well described, its prototype being the myocardial dysfunction of sepsis<sup>1</sup>. Recently, other situations have been reported evolving with myocardial dysfunction, also in a reversible form, but with peculiarities that distinguish them from septic dysfunction, mainly due to their characteristic clinical presentation, suggestive of acute myocardial infarction. Initially described in Japan<sup>2</sup>, the Takotsubo (a bottle used for trapping octopuses in Japan) syndrome or Takotsubo cardiomyopathy has been increasingly identified in daily clinical practice, including its important complications<sup>3</sup>. We report a case of that syndrome in a patient with respiratory failure of muscular etiology, which evolved with cardiogenic shock.

## Case Report

The patient was a 64-year-old male with myasthenia gravis for 1 year, an ex-smoker, who had quit smoking 20 years earlier and had a familial history of coronary artery disease. The patient, who

regularly used pyridostigmine, was hospitalized due to worsening of his neurological disease, with intense and generalized muscular weakness and clinical and gasometrical signs of hypoventilation.

Initially, the patient underwent plasmapheresis sessions, with no significant improvement. At the ward, he experienced chest discomfort and worsening of the dyspnea, being transferred to the intensive care unit, where he underwent electrocardiography that showed an ST segment elevation of approximately 2 mm from V1 to V6 with amputated R from V1 to V6 (fig. 1A).

Blood gas analysis showed significant hypoventilation (PCO<sub>2</sub>=105 mm Hg) with severe respiratory acidosis (PH=7.07; HCO<sub>3</sub>=29.9 mEq/L). The electrolytes and renal function were normal; troponin I was 5.0 ng/dL (NI=up to 1.0 ng/dL); and CKMb was 22 ng/dL (N=up to 3 ng/dL). The patient was intubated, put under mechanical ventilation, and underwent emergency cine coronary angiography. The examination showed that the left main coronary artery, the anterior descending artery, and the right coronary artery had no significant atherosclerotic lesions, and the left marginal branch of the circumflex artery had a 60% atherosclerotic lesion (fig. 2). The left ventricle had akinesia of the anteroapical wall and an ejection fraction of 33% (fig.3)

The patient evolved with arterial hypotension, which required 0.3 µg/kg/min of noradrenaline. A catheter was passed through the pulmonary artery, showing a cardiac index of 2.1 L/min and a pulmonary artery occlusion pressure of 12 mm Hg. On that occasion, his arterial lactate level was 31 mg/dL (normal, up to 22 mg/dL), the electrocardiogram showed an inactive zone from V1 to V6 (fig. 1B), and the echocardiogram showed apical dyskinesia, akinesia of the middle segment of all walls, and hypokinesia of the basal segment, mainly of the anterior and septal walls, with an ejection fraction of 20%. Dobutamine, at the dosage of 5 µg/kg/min, was initiated.

In the subsequent 24 hours, the patient evolved favorably. His blood pressure levels returned to normal with low doses of noradrenaline, and his lactate level normalized. He had an enzymatic curve typical of acute myocardial infarction with maximum CKMB and troponin levels of 22.3 ng/dL and 9.6 ng/dL, respectively. A new echocardiogram showed an improvement in the ejection fraction to 37%. The electrocardiogram showed recovery of the R wave and an alteration in the ventricular repolarization of the anterior wall. After 48 hours, noradrenaline could be suspended, and, after 72 hours, dobutamine.

On the tenth day after the event, the patient persisted on mechanical ventilation with no other organic dysfunctions. Mild apical hypokinesia existed; the ejection fraction was 60%, with

Hospital Israelita Albert Einstein  
Mailing address: José Marconi Almeida de Sousa - Rua Vicente Felix,  
60/121 - Cep 01410-020 - São Paulo, SP, Brazil  
E-mail: jmarconi@cardiol.br, jmarconi.dmed@unifesp.epm.br  
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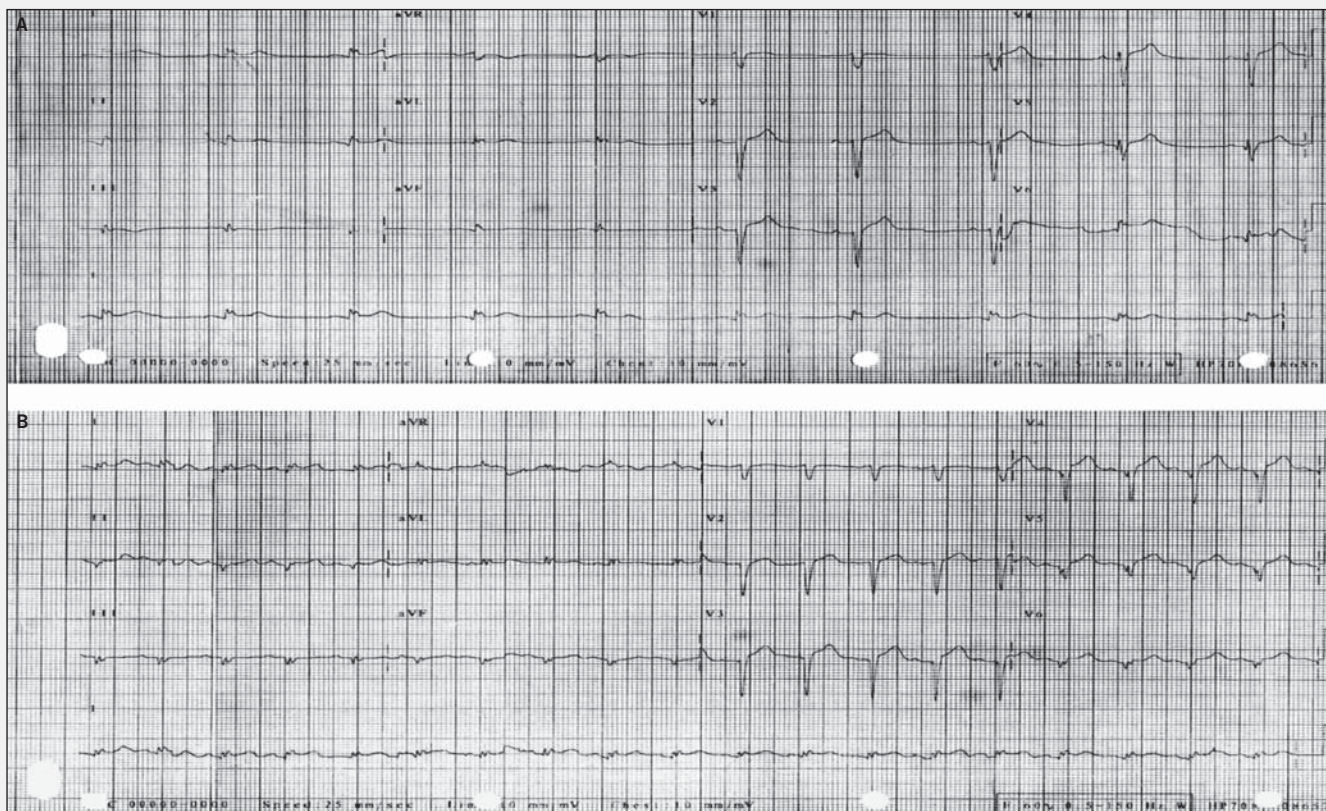


Fig. 1 - A) Electrocardiogram showing an ST-segment elevation from V1 to V6 with potential loss in that region and B) another 20 m after, with QS.



Fig. 2 - Left coronary artery in the cranial RAO and LAO views, showing coronary arteries with no severe obstructive lesions.

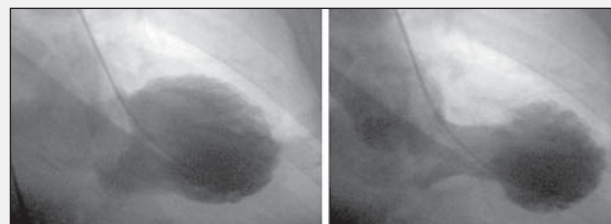


Fig. 3 - Left ventricle in the RAO view in final diastole and systole, resembling the device used for trapping octopuses in Japan (Takotsubo).

recovery of the electrocardiographic changes (fig. 4). After stabilization of the acute phase, the patient was transferred to another service, where, after one month, he died due to multisystem organ failure secondary to sepsis.

## Discussion

The patient reported thoracic discomfort of acute onset, which evolved with electrocardiographic alterations compatible with acute myocardial infarction with an ST segment elevation, accompanied by severe left ventricular dysfunction. Complete spontaneous reversion of those alterations occurred after 10 days. This clinical cardiac syndrome was first described in 1990 by Satoh et al <sup>2</sup>, and, in 1991, by Dote et al <sup>4</sup>. On that occasion it was called Takotsubo-type cardiomyopathy, due to the similarity, on ventriculography, to a device used for trapping octopuses in Japan. Most cases were described in that country, and this is the first case reported in Brazil under that name. In 2000, Franken et al <sup>5</sup> reported on a similar case, which they called "myocardial pseudo-infarction during an episode of herpes zoster"; these authors, however, did not characterize their case as the one here reported.

The etiology and clinical characteristics of that syndrome have not been completely clarified. However, in a recent review, Tsuchihashi et al <sup>6</sup> retrospectively studied 88 patients with that diagnosis. In that series, several conditions triggered the syndrome. Psychological factors were observed in approximately 20% of cases, exacerbation of a systemic disease in 3%, neurogenic causes in 7%, pulmonary causes in 8%, gastrointestinal disorders in 7%, renal disease in 6%, and other unspecific factors in 23 (15%) patients. None of the patients had myasthenia gravis, and the number of women was 6 times greater than that of men. Systemic arterial hypertension was the most frequent (48%) coronary risk factor. The only risk factor of our patient was hypertension, although he was an ex-smoker. Initially, most patients (67%) had precordial discomfort, 56% had an enzymatic elevation, and 90% had an ST segment elevation in the acute phase. Persistent Q waves occurred in only 10%. Cardiogenic shock was reported in 15%, use of intra-aortic balloon in 8%, and dopamine or dobutamine, or both, in 19%. In-hospital mortality was 1%. Only one patient had heart failure on hospital discharge. According to the criteria of the MONICA project <sup>7</sup>, 39% of the patients met 3 criteria for acute myocardial infarction, and in 43% of patients that diagnosis



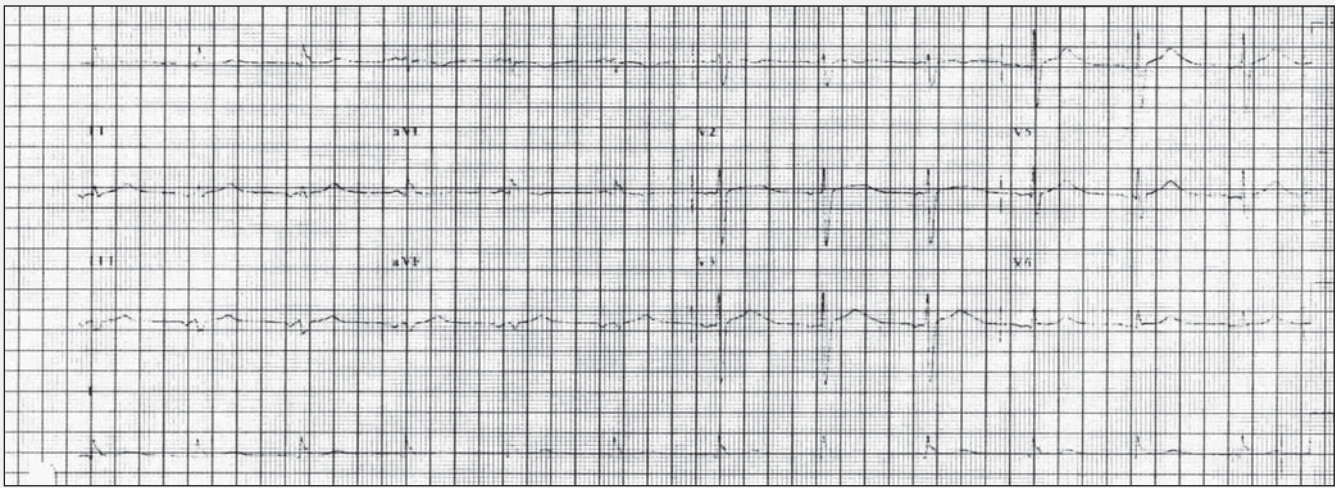


Fig. 4 - Electrocardiogram showing complete recovery of the R waves in the anterior wall.

was considered probable. Our patient met the criteria of the MONICA project for acute myocardial infarction<sup>7</sup> and the new guidelines of the American Heart Association<sup>8</sup>.

Complete recovery of our patient's ejection fraction was similar to that in the series described in this review, in which the mean ejection fractions in the acute and late phases were  $41\% \pm 11\%$  and  $64\% \pm 10\%$ , respectively.

The exact mechanism responsible for the transient ventricular alteration has not yet been elucidated. Thus, similarly to the cases reported by Tsuchihashi et al<sup>8</sup>, in our patient, early coronary angiography did not show total arterial occlusion, which, therefore, did not characterize a situation that could be explained by stunning or hibernating myocardium.

One peculiarity of the syndrome described is that its triggering is related to some extremely variable factors, stress being one of

them. All stressful situations show an elevation in catecholamines that can also cause ventricular dysfunction, such as in pheochromocytoma. In none of these cases were the catecholamines assessed; however, in a case reported by Kawai et al<sup>9</sup>, myocardial alterations similar to those of catecholamine-induced cardiomyopathy were found.

Therefore, although no defined etiology exists for that syndrome, we should keep ourselves well aware of that possibility in patients with clinical findings of acute myocardial infarction in the presence of a situation of systemic stress, because the specific treatment should be instituted, and, eventually, thrombolysis may not have the classic benefits known in such a situation, in which total occlusion of the coronary artery does not occur, but transient ventricular dysfunction of another etiology mimicking acute myocardial infarction does.

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