A Case Report of “Tako-tsubo-like” Syndrome

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Citation

Abstract
The case report describes a 65-year-old woman presenting chest pain typical of myocardial ischemia, electrocardiographic abnormalities and unstable clinical and hemodynamic condition. Coronary angiography demonstrated the absence of coronary stenosis, thrombi and coronary spasm. Echocardiographic examination revealed apical and lateral wall akinesis, with severe depression of global systolic function. The electrocardiographic evolution, modest elevation of cardiac enzymes and complete recovery of the regional wall motion abnormalities few days after the event are suggestive of tako-tsubo cardiomyopathy. A sudden psycho-physical stress event may have caused a violent and rapid increase of catecholamines, which has a direct toxic effect on the cardiac muscle.

INTRODUCTION
“Tako-tsubo-like” left ventricular dysfunction or transient left ventricular apical ballooning is a new syndrome that presents characteristics and effects similar to acute myocardial infarction (AMI).

Transient left ventricular apical ballooning can be considered a rare form of cardiomyopathy, which was initially described a few years ago [1,2,3,4,5]. Actually some authors deal with “tako-tsubo syndrome” because all the acute kinetic alterations that involve the left ventricle give it a shape which is very similar to a fishing pot (tako-tsubo) once used by Japanese fishermen to catch octopus. From a clinical point of view, precordial symptoms, together with electrocardiographic alterations, namely ST-T segment elevation in the anterior precordial leads, relate to the angiographic finding of a left ventricular apex aneurysm associated with hypercontractility of the basal segments and the absence of coronary atherosclerosis of the epicardial arteries.

CASE REPORT
A 65-year-old woman was admitted to a local Emergency Department due to chest pain in the retrosternal region associated with severe dyspnea. Anamnesis revealed that the woman had been suffering from hypertension, obesity and dyslipidemia for 10 years. Before the onset of the symptoms, the patient reported a significant stress episode following a serious quarrel with her husband. Due to persistent chest pain (about 3 hours), the woman went to the nearest hospital Emergency Department, where an electrocardiogram revealed ST-segment elevation in the anterior-lateral leads. Hypertension and acute pulmonary oedema were associated and the patient underwent intravenous thrombolytic treatment (full dose TNK), heparin and furosemide.

Transthoracic echocardiography showed the left ventricle normal in size, akinesia of the anterior interventricular septum, the apex and the near lateral wall; ejection fraction of 20%, mild mitral regurgitation, normal right ventricular size and function; absence of pericardial effusion or pulmonary hypertension. Cardiac enzymes, on admission to the Emergency Department, showed an increase in troponin I (22.4 ng/ml, normal < 0.1 ng/ml) and creatine kinase-MB 48 u/L.

After intravenous thrombolytic treatment, the woman became asymptomatic and the clinical condition was stable. But one hour later the patient suffered a recurrence of dyspnea; her systolic blood pressure dropped to 75 mmHg and heart rate became tachycardic (100 beats/min). There were clear signs of bilateral pulmonary edema. Because of the ineffectiveness of the therapy, the woman was transferred to our department.

At admission she was tachypnoeic, her blood pressure was 100/70 mmHg, her pulse heart rate was 110 beats/min; lung auscultation revealed signs of pulmonary edema up to the bilateral middle lung fields.
Blood tests were normal as regards hemoglobin, coagulative parameters, glycaemia, creatinine, serum electrolytes and liver function. The following values were altered: CK-MB (maximum 38 u/L) and troponin T (maximum 22 ng/ml). Electrocardiogram performed on admission to our department showed persistent ST elevation in anterior and inferior regions (Fig 1) and echocardiogram confirmed apex expansion associated with akinesia of anterior interventricular septum, anterior-lateral wall and the mid-distal inferior-posterior wall; the ejection fraction was 15-20% (Fig 2). Mild mitral regurgitation and a pulmonary arterial pressure (PAP) of about 40 mmHg were also noted; there was no evidence of inferior vena cava congestion.

**Figure 1**
Figure 1: The electrocardiogram performed on admission

**Figure 2**
Figure 2: The echocardiogram revealing apex expansion, akinesia of some segments of the left ventricle, with a severe reduction in the ejection fraction

In the coronary intensive care unit, the patient was treated with dopamine and furosemide. Urgent coronary angiography was performed, which revealed no signs of coronary stenosis. Ventriculography showed apical ballooning (fig 3).

**Figure 3**
Figure 3: Left ventriculogram in systole (3a) and diastole (3b) to illustrate the ballooning

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**Figure 4**
The clinical course showed a rapid improvement in her status; therefore therapy with ACE-inhibitors and later beta blockers was started: captopril 25 mg/day and carvedilol
Three days after admission and prescribed therapy, the patient was totally asymptomatic. An echocardiogram revealed the left ventricle normal in size and wall thickness and no segmental wall motion abnormalities; ejection fraction had improved to 54%. Left ventricular filling pattern was normal; mild mitral regurgitation persisted.

Considering the angiographic report of normal coronaries, further investigations were made in order to determine the exact etiology of the acute ischemic event.

Initially, since viral myocarditis was suspected, serum anti-CMV, Parvovirus, Chlamidya, Coxackie, Echovirus and Adenovirus antibodies were measured, yielding a negative result; thrombophilia was then considered, but the result was again negative. Adrenergic hypersecretion was suspected, as there are many cases in literature showing that catecholaminergic increase may cause left ventricular dysfunction, with normal coronaries, characterised by a quick resolution. Urinary catecholamine was estimated in an attempt to define this condition, with the following results: adrenaline < 0.005 mg/L (normal < 0.018), noradrenaline 0.025 mg/L (normal < 0.107), dopamine 0.106 mg/L (normal < 0.460), vanillyl mandelic acid 1.8 mg/L (normal < 7.5). The following plasma hormones levels were also estimated: cortisol 11 ug/l (normal 3-20), clinostatic aldosterone 50 pg/ml (normal 50-150) and orthostatic aldosterone 46 pg/ml (normal 50-300), clinostatic plasma rennin activity (PRA) 0.8 ng/ml/h (normal 0.2–2.7) and orthostatic PRA 0.9 ng/ml/h (normal 1.5–5.6).

The patient was re-examined one month later had normal homodynamic status. An electrocardiogram revealed normal sinus rhythm, normal atrio-ventricular conduction and no significant ventricular repolarization alterations (Fig. 4); echocardiography showed a left ventricle normal in size and function (EF 54%); mild mitral valvular regurgitation and slight pulmonary hypertension (PAP 37 mmHg) persisted.

Considering the above results, the therapy was changed to the following: captopril 50 mg/day, carvedilol 12.5 mg/day, simvastatin 20 mg/day and acetylsalicylic acid 100 mg/day.

**DISCUSSION**

The main features that characterize the Japanese “tako-tsubo-like syndrome” are: the peculiar acute left ventricular dyskinesia that regress within a few weeks; presence of an electrocardiographic features compatible with anterior AMI, together with modest chest symptoms, mainly as the result of physical and/or psychological stress and normal coronaries. Among the latest case studies of non-Japanese patients — though limited - in 2002 and 2003, the “tako-tsubo-like syndrome” accounted for 2.2% of acute coronary syndrome with ST elevation. This particular cardiomyopathy does not appear to be related to a single pathophysiological factor, probably due to the very small number of cases that have occurred up to now. In the light of these considerations, it is clear that the above clinical picture we could prove is hardly to be referred to a classic picture of acute, antero-septal myocardial non-transmural necrosis, associated with atherosclerotic coronary involvement.

One of the most interesting aspects of the clinical case presented here is the intense psycho-physical stress that preceded the acute event; a study by Abe et al has shown that 94% of cases are preceded by severe stress. A sudden catecholamine release which has been demonstrated in some patients affected by this syndrome may determine the onset of transitory ventricular dysfunction as a direct toxic effect, as shown during subarachnoid haemorrhage or other serious non-cardiac diseases.

From an experimental point of view, similar apical left ventricular ballooning has been observed in rats as a response to emotional stress and prevented with beta-blockers.

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**Figure 5**

Figure 4: The electrocardiogram performed 1 month later
Further physiopathologic studies will be able to explain the reason why this peculiar post-catecholaminic myocardial stunning is usually located at the left ventricular apex.

The characteristic of such cases of “tako-tsubo” is the quick restoration of the left ventricular function which has also remained so in repeat evaluations stretching up to 6 months from the event. In our case the left ventricular function was restored in 3 days; in literature it usually takes a few weeks\textsuperscript{8,11,12} before the complete recovery of the ventricular contractility.

In conclusion, the left ventricular transitory dysfunction reported here, which occurred following psycho-physical stress, can be considered a typical case of “tako-tsubo-like syndrome”.

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