A case of transient left ventricular apical ballooning. A condition simulating an acute myocardial infarction

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Transient left ventricular apical ballooning, sometimes associated with intraventricular pressure gradient, is a condition simulating an acute myocardial infarction and may occur in patients presenting with chest pain, electrocardiographic changes and minimal myocardial enzyme release typically without coronary angiographic stenosis. It was originally described in the Japanese population and is often associated with cerebrovascular accidents, surgical procedures and emotional and physical stress. We report the case of a 65-year-old woman presenting with chest pain typical of myocardial ischemia, dyspnea, electrocardiographic abnormalities and signs of hemodynamic instability, occurring after a severe emotional stress. Echocardiography and contrast ventriculography showed normokinesis confined to the basal segments of the left ventricle, with a markedly decreased ejection fraction. Scintigraphy was suggestive of a large perfusion defect. The electrocardiographic abnormalities and dyskinesis persisted for many hours. Coronary angiography, performed in the acute phase, was completely normal. Five months later, the functional and electrocardiographic abnormalities had totally disappeared.

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Introduction

Tako-Tsubo-like transient left ventricular dysfunction is a new syndrome simulating an acute myocardial infarction. It is characterized by chest pain, electrocardiographic changes and minimal myocardial enzyme release, transient left ventricular dysfunction[1] (sometimes with intraventricular pressure gradient and left apical ballooning[2]) and regional wall motion abnormalities and manifests in patients without angiographic stenosis at coronary angiography. Several cases have been reported in Japan and this syndrome is often subsequent to cerebrovascular accidents, surgery and emotional or physical stress. It is more common in women. The pathophysiology is not clear: multiple vasospasms of the epicardial coronary arteries or microvascular spasm causing myocardial ischemia are considered possible mechanisms. A key role seems to be played by the level of catecholamines during the events triggering this condition[3].

We report a case of transient left ventricular apical ballooning with electrocardiographic, echocardiographic and scintigraphic abnormalities (similar to those observed in patients with acute myocardial infarction) manifesting in a woman with typical chest pain and a normal coronary angiogram. We performed coronarography in the presence of ST-segment elevation and only documented slow flow in the left coronary artery, without stenosis or spasm of the epicardial branches. Furthermore, the creatine kinase levels were only slightly increased, even though the electrocardiographic and echocardiographic abnormalities persisted for hours and myocardial scintigraphy, performed within a few hours of symptom onset, showed a wide area of hypoperfusion. Five months later all the above-mentioned abnormalities had completely resolved.

Case report

A 65-year-old woman presented with chest pain typical of myocardial ischemia and dyspnea, after a severe emotional stress (a quarrel with her neighbors).

She had various cardiovascular risk factors including hypertension, elevated cholesterol levels, and a family history of sudden death.

On admission, the electrocardiogram, recorded while the patient was symptomatic for chest pain, revealed tachycardic sinus rhythm, signs of a slightly delayed right ventricular activation, with non-specific repolarization abnormalities (Fig. 1I). Her troponin and myoglobin quality assays were abnormal; the creatine kinase level was 236 U/l (normal value < 190 U/l) and that of creatine kinase-MB 29 U/l (normal value < 25 U/l).

Transthoracic echocardiography showed a mildly dilated left ventricle (the diameter of the left ventricle at the level of the mitral apparatus was 58 mm). The anterior wall, the middle and apical septum, the apical inferior segments and the lateral wall were akinetic, while the basal segments were hyperkinetic. These abnormalities resulted in a significant reduction of the overall left ventricular function (ejection fraction ≈ 35%). Doppler evaluation revealed severe mitral and tricuspid regurgitation as well as signs of pulmonary hypertension and of an abnormal diastolic relaxation.

A new electrocardiogram recorded 5 hours later (when the patient no longer complained of chest pain) showed ST-segment elevation in leads V_1-V_3 (Fig. 1II). Angiography, performed immediately after electrocardiography, was suggestive of a slow flow in the left coronary artery but did not reveal any coronary stenosis; no spontaneous spasm of the epicardial coronary arteries was documented; the length of the left anterior descending coronary artery was 145.5 mm. We did not perform any coronary spasm provocation test nor did we try to identify an intraventricular gradient because at that time our diagnostic hypothesis was a classic anterior myocardial infarction and not transient left ventricular apical ballooning.

Contrast left ventriculography confirmed akinesis of the apical, anterior and postero-lateral segments, with lack of diastolic relaxation, and hyperkinesis of the basal segments, with a globally decreased left ventricular function (left ventricular ejection fraction 22%) and moderate mitral regurgitation (Fig. 2); after the procedure, the patient developed signs of hemodynamic instability (systolic blood pressure 85 mmHg).

A few hours later we performed a rest perfusion tomoscintigraphy (^{99m}Tc-tetrofosmin) that revealed a large perfusion defect of the anterior-apical and postero-lateral segments (Fig 3).

The patient required intravenous inotropes (dobutamine) and diuretics for the first 3 days. Afterwards beta-blockers and ACE-inhibitors were prescribed and the patient's clinical status progressively improved. The peak serum levels of creatine kinase and creatine kinase-MB were 396 and 43 U/l respectively.

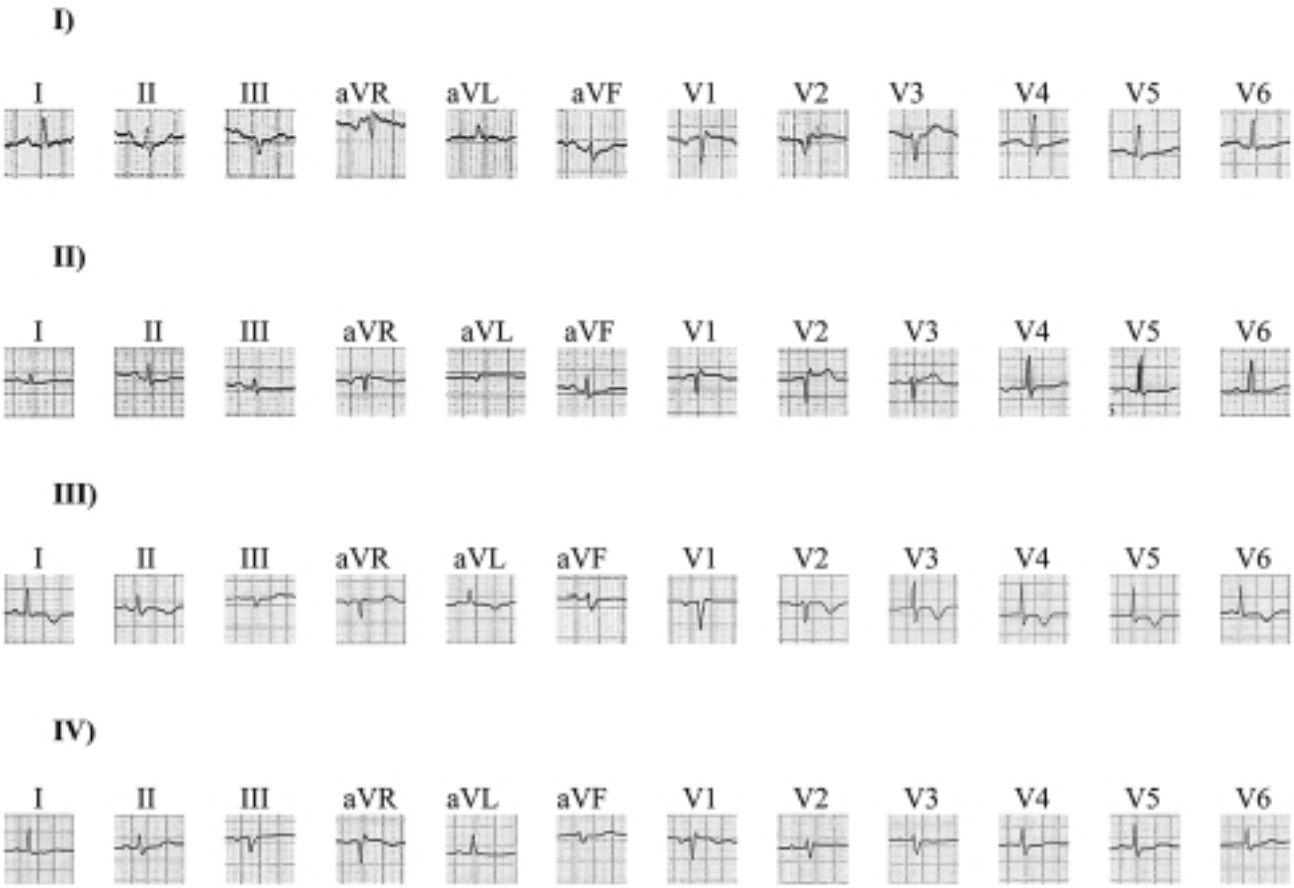

Figure 1. *Electrocardiogram performed at the time of admission to the emergency room (I), with ST-segment elevation in leads V_1-V_3, before coronary angiography (II), at discharge (III), and 5 months later (IV).*

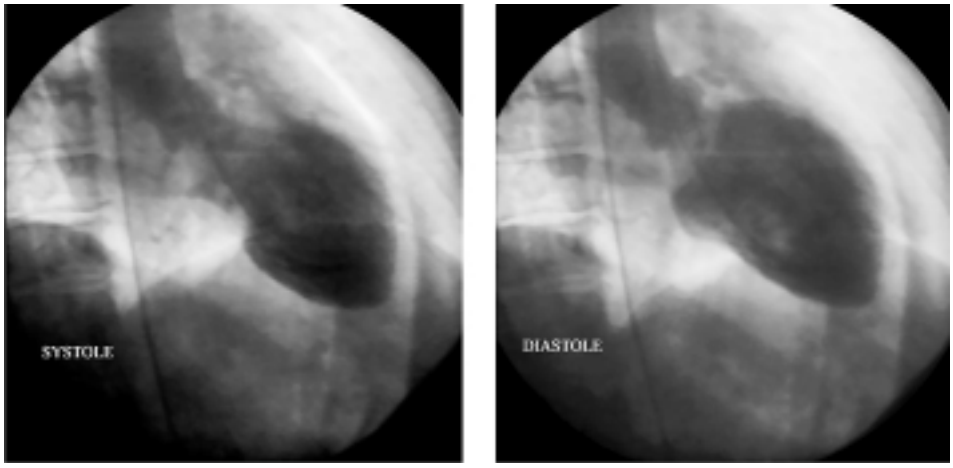

Figure 2. *Left ventriculogram during diastole and systole.*

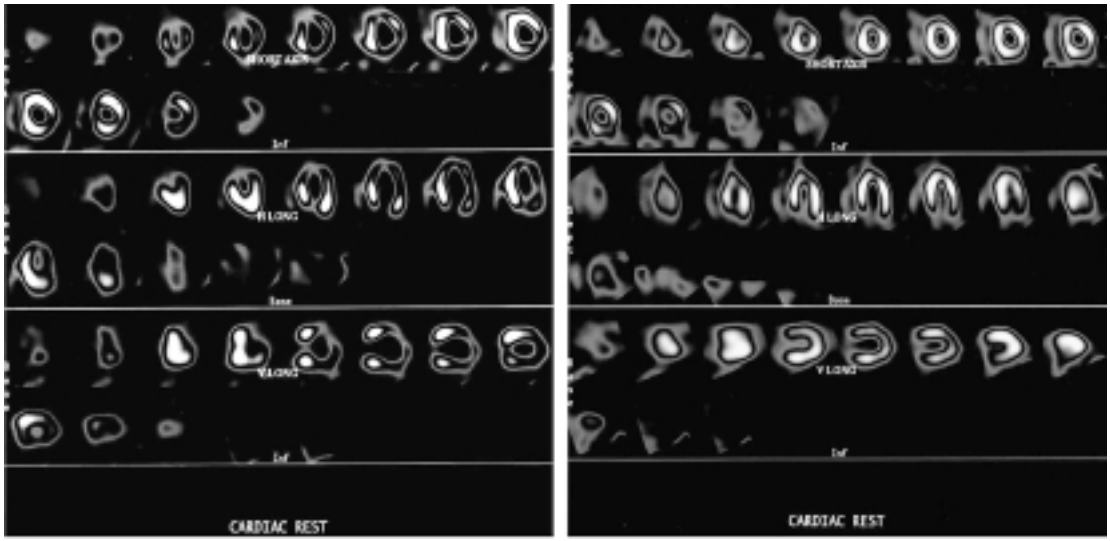

Figure 3. *Rest perfusion scintigraphy performed 3 hours following angiography and showing perfusion defects in the apical, anterior and postero-lateral segments and the same technique performed 40 days later and confirming the resolution of the perfusion defects.*

Echocardiography, performed 10 days after admission, showed normal left ventricular dimensions with moderate hypertrophy, akinesis limited to the apical segments and mild mitral and tricuspid regurgitation. The overall left ventricular function was only slightly impaired (left ventricular ejection fraction 42%).

Electrocardiography, performed immediately before discharge, showed a negative T wave in leads V_1-V_6 (Fig. 1III). Even these abnormalities were found to have resolved at clinical evaluation performed 5 months following discharge (Fig. 1IV).

Forty days later, repeat basal perfusion tomography (^{99m}Tc-sestamibi electrocardiographic-gated scintigraphy) documented the absence of perfusion defects with hypokinesis of the inferior segments and a normal ventricular function (left ventricular ejection fraction 59%) (Fig. 3). The hypokinesis of the inferior segments is not specific; indeed, the exams performed during the acute phase did not show hypoperfusion of the inferior segments and echocardiography was not suggestive of segmental kinetic abnormalities.

Discussion

Tako-Tsubo "cardiomyopathy" is a new condition mimicking acute myocardial infarction. It is characterized by transient apical ballooning and severe left ventricular dysfunction (sometimes with intraventricular pressure gradient), normal coronary angiography, and subsequent complete recovery. Several cases have been described in Japan, but it has received little attention in other countries (Europe and the United States) even if it should be rather frequent in intensive care units, particularly after cerebrovascular accidents or following surgery.

Even though above the normal range, the creatine kinase peak level and troponin titers are out of proportion to the extent and duration of the electrocardiographic abnormalities and to the left ventricular dyskinesis. This is typical of this syndrome. In the present case, the peak level of creatine kinase was only 396 U/l in spite of the fact that the electrocardiographic abnormalities persisted for hours and that echocardiography showed severe left ventricular dyskinesis with clinical signs of hemodynamic instability lasting for days.

The pathophysiology is still unknown; multivessel vasospasm of the epicardial coronary arteries and microvascular vasospasm have been both hypothesized. Our patient underwent angiography during the acute phase. No spontaneous vasospasm of the epicardial coronary arteries was identified. We only documented a slow flow in the left coronary artery, compatible with the hypothesis of microvascular spasm. Elsewhere, other authors documented, during the acute phase, vasospasm of the epicardial coronary arteries. Scintigraphy, performed during the acute phase, showed perfusion defects, probably consequent to prolonged microvascular abnormalities. In 2001 Di Chiara et al.[4] published 4 cases of patients with dynamic left ventricular outflow tract obstruction. This condition simulated an anterior myocardial infarction with cardiogenic shock. We could assume that those cases were actually examples of transient left ventricular dysfunction. According to these authors, the left ventricular dysfunction is due to a left ventricular outflow tract obstruction in patients with ventricular hypertrophy and excessive sympathetic stimulation. A strong sympathetic stimulation may cause left ventricular outflow tract obstruction, hyperkinesis of the basal segment and an apical dilation of left ventricle with signs of hemodynamic instability. For this reason, a high level of catecholamines secondary to the trigger event plays, in our opinion, an important role in the pathophysiology of this syndrome, and beta-blockers could be the optimal therapy for such patients. Obviously, not all patients with hemodynamic instability tolerate beta-blockers; the most severe cases need inotropic agents and/or mechanical support. We have noted, treating another case, that norepinephrine is better than other inotropic agents, probably because it allows for an increased afterload thus contrasting the outflow tract obstruction. Elsewhere, our experience showed that it is necessary to start beta-blocker therapy as soon as possible.

The recovery of the left ventricular function is usually complete; we documented a normal left ventricular kinesis and a normal perfusion after 40 days, while the electrocardiographic abnormalities resolved (T wave) after 5 months.

A more sensible approach coupled with the widespread use of echocardiography in the emergency room and in the intensive care unit could increase the recognition of this syndrome. Therefore we strongly recommend that the Tako-Tsubo cardiomyopathy be borne in mind so as to improve the diagnostic accuracy.

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