Clinical note

Transient apical dysfunction syndrome (Tako-Tsubo) simulating acute myocardial infarction

M.J. Carrero Lérida, a M.C. Mariscal Cerrato, a,* C. Dávila Arias, a A. López Ruiz, a J. Caballero Güeto b

a Servicio de Medicina Nuclear, Hospital Clínico Universitario San Cecilio, Granada, Spain
b Servicio de Cardiología, Hospital Clínico Universitario San Cecilio, Granada, Spain

Introduction

The transient apical dysfunction syndrome (TADS), Tako-Tsubo, apical ballooning, broken heart syndrome, stress cardiomyopathy or neurogenic disturbance was described by Hiauru Sato in 1990.1 In 2006, the American Heart Association included this syndrome in its classification of cardiomyopathies as primary acquired cardiomyopathy. This syndrome simulates one of acute myocardial infarction (AMI) and is characterized by the presence of ischemic chest pain accompanied by an elevation in the markers of myocardial necrosis and electrocardiographic alterations as well as hypokinesis, akinesis or anteroapical dyskinesis, in absence of significant coronary disorders. It generally resolves in days or weeks with individualized support measures. We present the case of a female patient referred to our service for a myocardial perfusion imaging study due to a history suggestive of an acute coronary syndrome after a stressful event.

It usually resolves within days or weeks with individualized means of support. This syndrome predominantly affects the female sex with an age range of 60 to 80 years and is related to a picture of physical or emotional stress. The etiology is unknown despite several pathophysiological mechanisms having been described, among which involvement secondary to the discharge of catecholamines triggered by stress on a heart unable to maintain an adequate inotropic response is of note.3

Clinical case

A 78-year-old hypertense woman receiving treatment without other cardiovascular risk factors arrived to the emergency department for long duration oppressive pain in the left hemithorax which irradiated to the throat after having been robbed in the street.

Physical examination found a bad general status, tachycardia with a trend to gallop and normal respiratory auscultation. Arterial pressure was 110/60 mm Hg. Electrocardiogram showed sinusal rhythm and T wave inversion in V2-V6 precordial without Q wave (fig. 1). Blood analysis showed a maximum peak of troponine of 0.6 ng/dL (range of normality 0.1) and myoglobulin of 69 ng/mL. Cardiological examination by echocardiography showed marked

* Corresponding author.
E-mail: carmina_mariscal@hotmail.com (M.C. Mariscal Cerrato).
medial dysfunction on all the sides with severe ventricular dysfunction (ejection fraction (EF) of 15 %) (fig.2). During the stay in the emergency department observation unit she presented a picture of hypotension and was admitted to an intensive care unit where cardiac insufficiency and an episode of rapid auricular fibrillation at 130 beats per minute began. The following morning and after having reverted to a sinus rhythm a gated-SPECT study was performed showing normal perfusion while septal akinesis-dyskinesis was observed with an important alteration in the left ventricle EF (LVEF) of 26 % (figs. 3 and 4).

The following day the patient was transferred to the hemodynamics unit for coronaryography which showed epicardic coronary arteries without significant angiographic stenosis as well as anteropical akinesis and hypercontractility of the basal segments with a moderately depressed LVEF (figs. 5 and 6).

The clinical course was favorable and the patient was transferred to the cardiology ward to continue the control of evolution. At three weeks a new study was carried out with a control gated-SPECT which showed complete recovery of contractility as well as an improvement in the LVEF (58 %) (fig. 4).
Discussion

The TADS was described by Hiuru Sato in 1990. In 2001 it was typified as a clinical entity with the finding of a series of 88 cases in Japan and in 2003 it the first cases were described in the US. In 2006 the American Heart Association incorporated the syndrome in its classification of cardiomyopathies as primary acquired cardiomyopathy.

In reference to TADS, it has been hypothesized that there may be a hyperactivity of the local sympathetic system secondary to a hyperadrenergic discharge with a brusque elevation of catecholamines accompanied by an “asymmetric” myocardial response. In addition,
clear similarities have been observed between TADS and myocardial disturbances. The discharge of catecholamines reported in both diseases is also detected in entities such as subarachnoid hemorrhage or pheochromocytoma crises.4 Another argument in favor of the adrenergic thesis is the discovery of an experimental model of TADS in which continued stress provokes hyperkinesis of the anteropapical region of the LV with the changes in animals receiving supplementary estrogens remaining attenuated or normalized after blockade of the adrenoreceptor α or β.5

Other causes which have been suggested are an abnormality in the coronary anatomy in the apical region with the presence of an anterior descending coronary artery with a large recurrent segment,6 the presence of a sigmoid septum, dynamic medioventricular obstruction, the appearance of an intraventricular gradient7 or viral myocarditis. A reduction in apical and septodistal coronary flow and normality of the flow in the mediobasal zone have been demonstrated with techniques such as PET and echocardiography.

The most extended opinion is that TADS is due to a multiple and as yet unclear etiology with the possible causes ranging from spasms of the responsible epicardic coronary arteries also from another clinical entity of ischemic heart disease: Prinzmetal angina. The difference between the two entities lays in the triggering factors, the risk factors and the variations in the clinical manifestations. The TADS is more frequent in women over the age of 60 years associated with stress as the triggering factor. It simulates an acute myocardial infarction producing a transient ventricular dysfunction and is usually a benign disease, although it may present complications (cardiogenic shock, arrhythmias, mural thrombi, wall rupture). Prinzmetal angina however, is more frequent in young men and is related to smoking but is not associated with stress and does not produce ventricular dysfunction.

Moreover, the differential diagnosis should extend to the previously mentioned diseases: subarachnoid hemorrhage, pheochromocytoma, myocardial disturbances, toxic substances such as cocaine and myocarditis.8

The clinical presentation of the picture is similar to that of ACS. The onset may include chest pain (53–71 %) with similar characteristics and irradiation as that of ischemic cardiopathy with dyspnea (7–20 %) and, more rarely, with cardiogenic shock (5 %).9

The electrocardiogram shows over elevation of the ST segment on the anterior side in up to 90 % of the cases. Around 25 % of the patients show pathological Q waves with a mirror image in the inferior side being rare. The electrocardiographic changes may last days or weeks and evolve to disappearance of the Q wave (90–100 %), normalization of the ST segment and the presence of deep negative T waves (84–97 %).9

Only 50 % of the patients present an elevation in the myocardial necrosis markers and this is much lower than that expected in relation to the alterations described.10

The echocardiography shows an alteration in the EF, mediopapical hypokinesis or akinesis and a normal or hyperkinetic base which normalizes in days or weeks.11

Coronarography shows the arteries to be normal or without significant stenosis, although at present, and without demonstrated clinical evidence, the role of coronary angiography with multislice CT without the need for a hemodynamic study should be considered in the evaluation of the coronary tree.

Although it is true that there are no clear therapeutic recommendations and that those that are present limit the means of individualized support for each patient, the importance of achieving a rapid differential diagnosis with ACS lays in the possibility of avoiding fibrinolytic treatment and the risk it carries,12 and the benefit of administering alpha-adrenergic agonists.

The echocardiographic findings provide differential diagnosis with acute myocardial infarction with elevation of the ST segment by the total occlusion of a dominant anterior descending artery (surrounding the apex and irrigating part of the inferior wall). The transient character of the alterations in contractility determines the differentiation between the two entities which is why clear diagnosis of TADS is always late and never in the acute phase. Thus, the use of a gated-SPECT study demonstrating the recovery of the alterations in contractility and LV EF aids in establishing the definitive diagnosis.

The TADS presents more initial complications than ACS: acute pulmonary edema (22 %), cardiogenic shock (15 %), ventricular arrhythmias (9%) but a better short and medium term prognosis.13

Conclusion

In the presence of clinical manifestations suggestive of ACS in elderly patients (mainly women) with a trigger related to physical or emotional stress, electrocardiographic alterations suggestive of involvement of the anterior side, null or minimum elevation of biochemical data and important hypokinesis, akinesis and even septoapical dyskinesia not correlated with a slight alteration in the myocardial markers of necrosis, TADS should be suspected. Coronarography continues to support the diagnosis since no significant stenosis is observed. Nonetheless, definitive diagnosis is obtained on demonstrating the recovery of contractility. Myocardial perfusion studies with gated-SPECT obtained in the first phase in the absence of perfusion defects suggestive of necrosis and associated with important disorders in septoapical motility may aid in establishing the diagnosis of TADS. Although it is true that diseases with minimum necrosis may adopt a similar perfusion pattern, it is very difficult for the motility study to reveal important hypokinesis, akinesis and even dyskinesia. Late gated-SPECT is of great utility is demonstrating the recovery of LV functionality and regional contractility.

Although it has not been completely demonstrated, the management of these patients should consider the foreseeable benefit of careful administration of beta-blockers, IECA and alpha-adrenergic agonists.14

References


