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

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Resumen

El síndrome de discinesia apical transitoria es una entidad indistinguible clínicamente de un síndrome coronario agudo que cursa con alteraciones electrocardiográficas y elevación de los marcadores de necrosis miocárdica acompañado de un cuadro de hipocinesia, acinesia o discinesia anteroapical, en ausencia de alteraciones coronarias significativas. Suele resolverse en días o semanas con medidas de soporte individualizadas. Presentamos el caso de una paciente remitida a nuestro servicio para estudio de perfusión miocárdica por cuadro sugerente de síndrome coronario agudo tras un episodio de estrés emocional.

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medioapical 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 sinusal rhythm a gated-SPECT study was performed showing normal perfusion while septoapical 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 coronariography 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).

Figure 1. Electrocardiogram demonstrating a rise in the ST segment which was more accentuated in the V2 and V3 derivations as well as inversion of the V5-V6 T wave.

Figure 2. Echocardiographic images. The left in systole and the right is diastole showing an optimum movement in the lateral side and akinesis at the septoapical level.

Figure 3. Perfusion study obtaining slice and polar maps showing the normality of the distribution of the tracer without evidence of signs of ischemia or necrosis.
Discussion

The TADS was described by Hiauru 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. 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 anteroapical region of the LV with the changes in animals receiving supplementary stressors remaining attenuated or normalized after blockage of the adrenoreceptor α or β.3

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,4 the presence of a sigmoid septum, dynamic medioventricular obstruction, the appearance of an intraventricular gradient5 or viral myocarditis. A reduction in apical and septodistal coronary flow and normalcy of the flow in the medibasal 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 spasm of the responsible epicardiac 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, mediapodial hypokinesis or akinesis and a normal or hyperkinetic base which normalizes in days or weeks.11

Coronariography 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 LVEF aids in establishing the definitive diagnosis. 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, nul or minimum elevation of biochemical data and important hypokinesis, akinesis and even septoapical dyskinesis not correlated with a slight alteration in the myocardial markers of necrosis, TADS should be suspected. Coronariography 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 dyskinesis. Late gated-SPECT is of great utility is demonstrating the recovery of LV functionality and regional contractility.

With respect to TADS, the medical literature does not provide clear therapeutic recommendations and what is available is limited to means of individualized support for each patient. The importance of achieving a rapid differential diagnosis compared with ACS lays in the possibility of avoiding the use of inotropic drugs and nitrates which may increase the dynamic gradient and fibrinolytic treatment due to its inherent risk.7

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