

Transient Left Midventricular Dyskinesia: Clinical Features of a New Variant

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We report 5 consecutive patients with transient midventricular dyskinesia, a recently described variant of transient apical dyskinesia. It is a syndrome that shares a broad clinical, prognostic and, probably, pathogenetic resemblance to the latter. The patients, the majority of whom were experiencing an episode of emotional or physical stress, were all admitted to an emergency department with a clinical presentation compatible with acute myocardial infarction. Evidence of dyskinesia or akinesia in midventricular segments was seen on ventriculography. These ventricular abnormalities resolved completely over a short period of time. No coronary artery abnormality was evident in any patient. The existence of this variant shows that segments other than apical ventricular segments, which are affected in tako-tsubo syndrome, can also undergo reversible change. The occurrence of new variants raises further questions about the relationship between the nervous system, catecholamines, and reversible myocardial lesions.

Key words: *Transient dyskinesia. Midventricular. Infarction. Coronary angiography. Tako-tsubo.*

Discinesia medioventricular izquierda transitoria: aspectos clínicos de una nueva variante

Presentamos 5 casos consecutivos de discinesia medioventricular transitoria, una variante recientemente descrita de discinesia apical transitoria. Se trata de un síndrome que presenta similitudes con la anterior en cuanto a características clínicas, pronósticas y probablemente etiopatogénicas. Los pacientes, la mayoría en relación con un episodio física o emocionalmente estresante, acudieron a urgencias con un cuadro compatible con infarto agudo de miocardio que incluía discinesia/acinesia de los segmentos medios en la ventriculografía. Las alteraciones ventriculares se resolvieron de manera temprana y completa. Las arterias coronarias no mostraron alteraciones en ningún caso. Su existencia denota que no sólo las porciones apicales, afectas en el síndrome *tako-tsubo*, pueden alterarse de manera reversible. La aparición de nuevas variantes abre nuevos interrogantes sobre la relación de sistema nervioso, catecolaminas y lesión miocárdica reversible.

Palabras clave: *Discinesia transitoria. Medioventricular. Infarto. Coronariografía. Tako-tsubo.*

INTRODUCTION

In Japan in 1990, Sato et al^{1,2} described a syndrome consisting of chest pain, electrocardiographic, and biochemical changes, all similar to those of acute myocardial infarction, and abnormalities of apical left ventricular contractility. The unusual component was the lack of lesions of the coronary vessels and the almost complete resolution of the segmental disorders after a

few days or weeks. This syndrome was called “apical ballooning,” “tako-tsubo,” or transient apical dyskinesia. Of note was the segmental involvement of various coronary territories and the excellent prognosis. Cases were later reported from all over the world and involving all races.³⁻⁵

METHODS

We report the first series of consecutive cases in Spain, all with similar symptoms, but with different segmental involvement, who had the same benign course. The patients all had midventricular abnormalities, similar to those recently described.⁶⁻¹⁰

The patients were all Caucasian women, with symptoms compatible with acute coronary syndrome (Table 1);

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TABLE 1. Characteristics of the Patients and Presentation

Characteristics/Case	1	2	3	4	5
Age	44	62	52	69	69
Sex	Female	Female	Female	Female	Female
Hypertension ^a	Yes	Yes	No	Yes	Yes
Dyslipidemia ^a	Yes	Yes	Yes	Yes	No
Diabetes mellitus ^a	No	Yes (insulin)	No	No	No
Smoker	10 cigarettes/day	No	No	No	No
Depression/anxiety ^a	Yes	No	No	No	No
Triggering factor	Psychic	Physical	Psychic	Physical	No
Presentation	Dizziness and syncope	Resting chest pain	Exertion chest pain	Epigastric oppression	Exertion chest pain
Symptom onset ^b	<6 h	In-hospital	<6 h	<6 h	12 h

^aDiagnosed before admission and treated pharmacologically.

^bUp to arrival at the hospital.

elevation (on serum analysis) of markers of myocardial necrosis; electrocardiographic abnormalities (ST segment elevation >1 mm in 2 leads and/or the appearance of negative T waves in 2 or more leads); left ventricular contractility disorders (severe dyskinesia/akinesia/hypokinesia) on ventriculography, but with no apical involvement; with no significant coronary vessel abnormalities (obstruction <50% and absence of unstable plaque that might cause the symptoms), with complete recovery of ventricular contractility. Patients with cranioencephalic abnormalities that might have caused the symptoms were excluded. The initial segmental classification was done with ventriculography (Figures 1 and 2), and transthoracic echocardiography was used for the follow-up (Table 2).

RESULTS

Case 1

A 44-year-old woman who presented dizziness and syncope that lasted just seconds, in relation with a family problem. Because of generalized negative T waves and high troponin I levels coronary arteriography was carried out. No coronary lesions were seen, but anterolateral akinesia and inferior hypokinesia were detected (left ventricular ejection fraction, 32%) with moderate mitral insufficiency. A transthoracic echocardiogram performed 5 days later showed normalization of the earlier findings. Cardiac magnetic resonance imaging before discharge showed no segmental abnormalities or signs of myocarditis.

Case 2

A 62-year-old woman was admitted with increased dyspnea, respiratory infection, and bronchospasm; there were no data suggestive of myocardial involvement. Twenty-four hours later she reported central chest pressure and had ST elevation on the upper lateral surface, as well

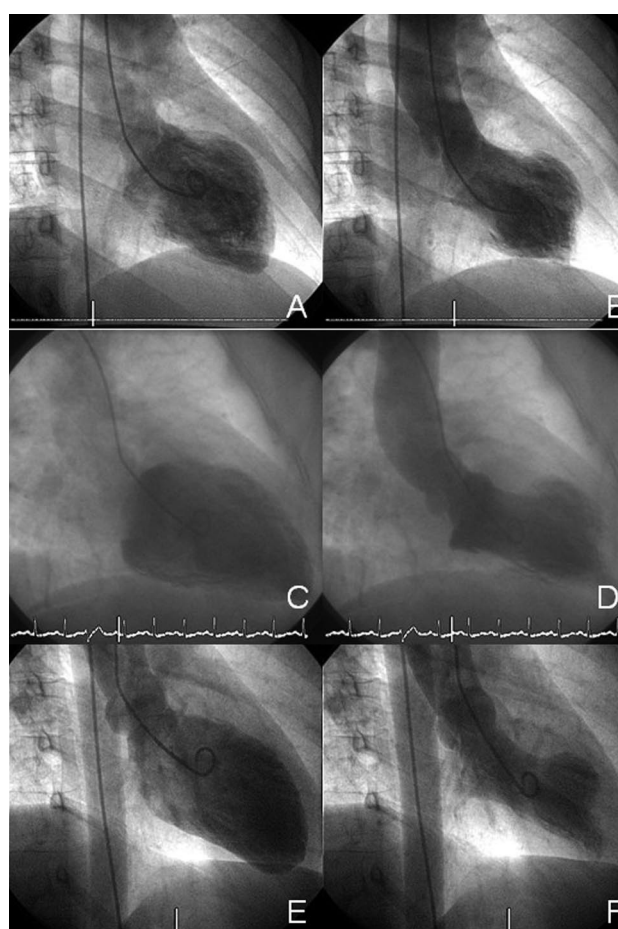


Figure 1. Ventriculograms. A and B: case 1. C and D: case 2. E and F: case 3. Diastole is shown on the left (A, C, and E) and systole on the right (B, D, and F).

as raised levels of markers of necrosis. A coronary angiogram showed normal coronary vessels, and a ventriculogram showed anterior dyskinesia with basal hyperkinesia. The follow-up echocardiogram carried out four months later showed normalization of the contractility abnormalities.

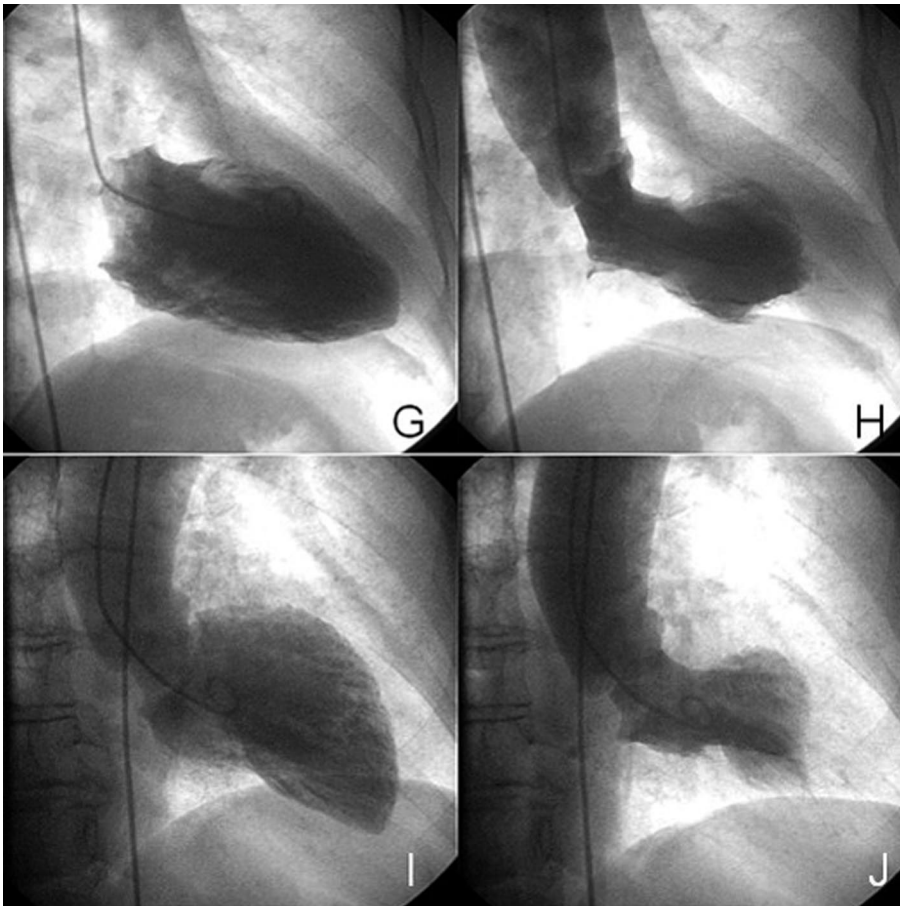


Figure 2. Ventriculograms. G and H: case 4. I and J: case 5. Diastole is shown on the left (G and I) and systole on the right.

Case 3

A 52-year-old woman presented to the emergency department with a 3-hour history of typical exertion pain that ceased with nitroglycerin. She had negative T waves and raised troponin I concentrations. A coronary angiogram showed normal coronary vessels and a ventriculogram showed an area of severe hypokinesia localized on the anterolateral surface. The echocardiogram on discharge was normal and the patient remains asymptomatic.

Case 4

A 69-year-old woman, who had had asthma since childhood, peripheral facial palsy, and a history of paroxysmic atrial fibrillation, presented to the emergency department with a hypertensive crisis which ceased with no further complications. The following day she reported epigastric oppression accompanied by rapid palpitations and cephalgia. Her blood pressure was 180/90 mm Hg. Troponin was raised (2.11 ng/mL). The coronary angiogram showed normal epicardic arteries and the ventriculogram revealed anterior hypokinesia. Following catheterization, the electrocardiogram showed negative T waves in the precordial leads, which were later corrected.

An echocardiogram 5 days after her admission showed no abnormalities. She has since presented with episodes of rapid atrial fibrillation. She has been admitted once, but with no clinical involvement, increase in markers of necrosis or new electrocardiographic abnormalities indicative of ischemia.

Case 5

A 69-year-old woman who had had olivopontocerebellar atrophy for several years presented to the emergency department with a 12-hour history of central chest oppression accompanied by dyspnea. She had ST elevation of 1 mm in leads I and aVL, with later T wave inversion at the same level, and raised markers. Coronary arteriography showed no abnormalities of the coronary tree, with akinesia localized to the anterolateral region with normal apical contractility. An echocardiogram prior to discharge showed no segmental abnormalities.

DISCUSSION

This new variant of transitory dyskinesia is unusual because it does not apparently affect the apex of the left ventricle, unlike the classic form of tako-tsubo. In spite of the little experience with the 2 conditions, it would

TABLE 2. Complementary Tests and Evolution

Data/Case	1	2	3	4	5
Troponin I, maximum ^a /h ^b	0.33/8	10/48	0.37/8	2.11/6	2.81/6
Creatine kinase, maximum/h ^b	Normal	318/48	Normal	175/6	812/24
Electrocardiogram ^{c,d}					
ST elevation	No	I, aVL	No	No	I, aVL
Negative T waves	V ₂ -V ₆	I, aVL	V ₂₋₃	V ₂₋₃	I, aVL
Maximum QTc (Bazett), ms	450	470	510	468	519
Coronary arteries	Normal	Normal	Normal	Normal	Normal
Peculiarities (left anterior descending)	Recurrent	Slow flow Short recurrent	Recurrent	Recurrent	Slow flow Double left anterior descending system
Dominance	Right	Right	Left	Right	Right
LVEF (catheterism)	32%	50%	65%	65%	66%
Coronary angiography time ^e	<12 h	72 h	<24 h	24 h	24 h
Left ventricular gradient	No	No	No	No	No
Normalization time ^f	5 days	90 days	4 days	5 days	8 days
Later LVEF	50%	60%	60%	60%	70%
Complications ^g	Complete reversible atrioventricular block	Hematuria	No	Femoral pseudoaneurysm	No
Admission time	12 days	5 days	4 days	9 days	9 days
Further admissions	No	No	No	Yes, due to atrial fibrillation	No
Follow-up	1 year	7 months	9 months	1 year	18 months

LVEF indicates left ventricular ejection fraction.

^aNormal troponin I <0.2 ng/mL; normal creatine kinase <170 U/L (curve of markers).

^bApproximate time of arrival at the emergency department and maximum troponin/creatinine kinase.

^cDuring the admission.

^dMost notable electrocardiographic changes near the event.

^eApproximate time between onset of pain and catheterization.

^fUntil noting recovery, normally with transthoracic echocardiogram.

appear that, as well as sharing a lack of coronary lesions and the distribution of the motility abnormality in various vessels, they both have a benign prognosis with no complications, at least over the short term.⁷ The incidence of transient apical dyskinesia is unknown, although it could account for 1% of infarctions.³ However, its frequency may be greater but go unnoticed due to its lack of clinical expression. The midventricular variant appears to occur less often.⁷ Apical dyskinesia occurs predominantly in women,³ whilst Western persons tend to have inverted T waves rather than ST elevation, which is more common in Asian patients.¹⁰

Many etiological and pathological mechanisms have been suggested as explanations for these transitory ventricular abnormalities, which can resemble acute myocardial infarction in their clinical, analytical, electrocardiographic, and echocardiographic aspects.³⁻¹⁶ Of note are myocarditis,^{3,4} non-obstructive plaque rupture with spontaneous thrombolysis, anatomic abnormalities (very long left anterior descending artery),¹⁶ and geometric ventricular predisposition¹⁴ (gradient with left ventricular outflow tract obstruction), sepsis,¹⁵ and the most commonly proposed lately: elevated concentrations of catecholamines and neuropeptides due to stress.¹³ This latest theory is based on the clinical, anatomical, and

pathological association between transitory dyskinesia due to psychic stress (death of a relative) or physical stress (asthma, surgery), and that which is neuromediated in ictus, subarachnoid hemorrhage, cranioencephalic trauma, and pheochromocytoma. The mechanism between sympathetic stimulus and myocardial contusion is far from being discerned: spasm of the epicardic coronary vessels, microvascular spasm with reduction of coronary reserve, and endothelial dysfunction, or direct myocardial lesion (calcium overload mediated by cyclic adenosine monophosphate, free radicals).

No conclusive theories yet exist to explain the exclusive abnormality of the midventricular segments. It could reflect the temporal evolution of apical dyskinesia in the process of recovery, although given the haste with which cardiac catheterization was done this hypothesis seems questionable. The low values of the markers of necrosis and the rapid recovery of the patients (4 out of 5 prior to discharge) could suggest that it may be an abortive or attenuated variant of the classical form. Whatever the case, no demographic, clinical, biochemical, or angiographic differences have been noted between this and the classical form, although regional metabolic abnormalities coinciding with abnormalities in motility have been found.⁷

Currently, no clear therapeutic recommendations exist, and those recommendations available are restricted to the usual management of the complications and propose the use of beta-blockers.

In conclusion, further studies are required to explain the mechanisms associated with this type of involvement and to design efficient diagnostic and therapeutic attitudes.

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