

A Case of Transient Left Ventricular Apical Ballooning with an Unusual Complication

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A retrospective study of patients with acute myocardial ischemia syndrome, mainly middle-aged and elderly women, was published in July 2001. The main features of acute myocardial ischemia were typical triggering circumstances, initial ECG mimicking acute myocardial infarction (AMI with transient appearance of Q waves and large negative T waves), mild or no enzymatic changes, and a combination of a normal coronary angiogram and transient left ventricular apical dyskinesia that normalized within days. The clinical course and prognosis were completely different from those of conventional AMI, with an increased number of acute-phase complications such as acute pulmonary edema, cardiogenic shock, and ventricular tachycardia. Despite this, the long-term outcome was better than that of AMI. We report a typical case diagnosed in a Spanish woman who developed embolic stroke, a complication not previously described, most likely related with her apical dyskinesia.

Key words: *Myocardial infarction. Myocardial ischemia. Stroke.*

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Síndrome de discinesia apical transitoria con una complicación inhabitual

En julio de 2001 se ha publicado un estudio retrospectivo de pacientes, sobre todo mujeres de edad mediana y avanzada, que presentaban un síndrome de isquemia miocárdica aguda precedido de un desencadenante típico, con evolución inesperada en el electrocardiograma (aparición transitoria de ondas Q, ondas T negativas gigantes), leve o inexistente elevación enzimática y la combinación de coronarias normales y discinesia apical transitoria en la fase aguda, con normalización de la función ventricular días más tarde. El curso y el pronóstico son completamente diferentes de los del IAM convencional, con más complicaciones iniciales, como edema agudo pulmonar, shock cardiogénico y taquicardia ventricular y, sin embargo, mejor pronóstico a medio-largo plazos. Describimos un caso típico en una enferma de nuestro medio que presentó una embolia cerebral, complicación aún no descrita, probablemente relacionada con su discinesia apical.

Palabras clave: *Infarto de miocardio. Isquemia miocárdica. Accidente cerebrovascular.*

INTRODUCTION

Around 6% of acute myocardial infarctions (AMI) in which coronariography is performed occur in the presence of normal coronary arteries. The etiology of these conditions varies widely and includes coronary spasm, coronary embolism, arteritis, cardiac trauma, aortic valve disease, blood dyscrasia, and cocaine use.¹

A recent series² of 88 Japanese patients with transient left ventricular apical ballooning syndrome, or transient apical dyskinesia (TAD), has been published. The epidemiology, trigger factors, clinical, electrocardiographic, and enzymatic evolution, as well as the existence of TAD with normal coronary arteries differentiate it from the conditions described above. Although cases have been described in Japan since 1990,³ it is exceptional in other regions.

Rapid recognition of the syndrome can modify the diagnostic and therapeutic attitude (avoiding thrombolysis and performing catheterization in the acute phase), so we think that it is useful to report a typical case that was recently seen in our center. We also report an embolic complication of the syndrome that has not been described in the literature.

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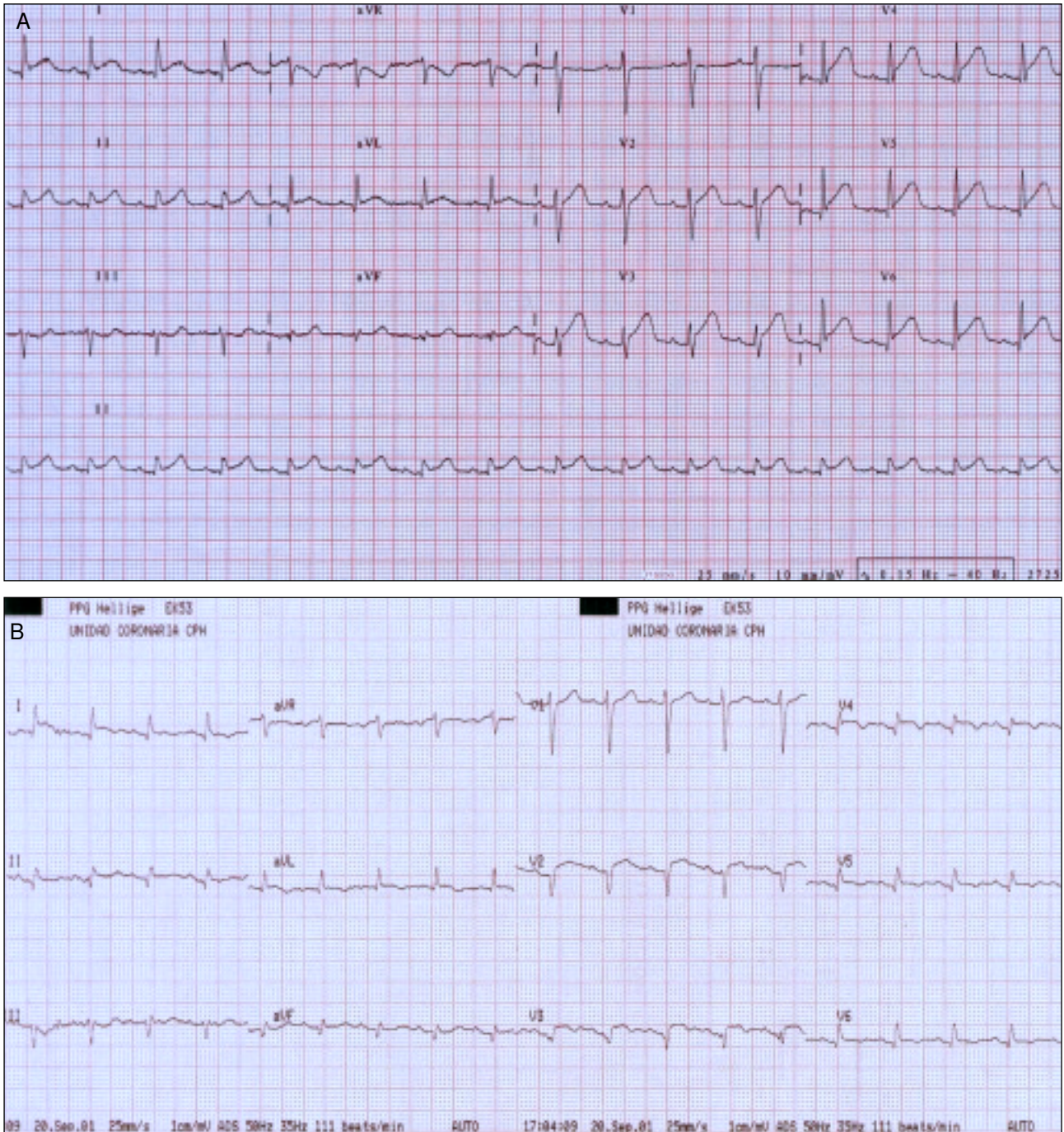


Fig. 1. A: ECG of the patient on arrival to the emergency room. Observe the elevation of the ST segment in leads V2-V6. B: ECG of the patient a few hours after the onset of the condition. Q waves have appeared in V2-V6 and elevation of the ST segment persists.

CLINICAL CASE

A 65-year-old woman with mild hypertension, smoker of 20 cigarettes/day until the age of 45, was seen in the emergency room of our hospital for palpitations and nausea after an accidental fall with injury to the head and right elbow. In the emergency room, she presented precordial oppression lasting 3-4 minutes.

The physical examination showed blood pressure

134/74 mm Hg, heart rate 103 beats/min, and post-traumatic hematoma of the right frontal region. The blood tests disclosed a normal differential blood count, kidney function, and electrolyte concentrations. The initial creatine phosphokinase was 222 U/L (normal, 24 U/L-170 U/L). The initial troponin I was 11.8 µg/L (normal, up to 0.4 µg/L). The chest radiograph was normal.

The initial ECG, performed while the patient was

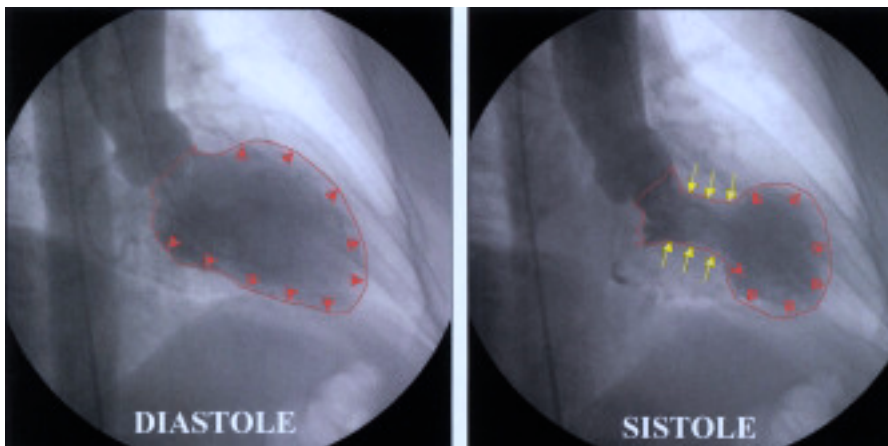


Fig. 2. Photographs of the patient's left ventriculogram in diastole and systole. The dark arrowheads mark the left ventricular contour in diastole and the zone of apical ballooning in systole. The light arrows indicate zones of normal contractility in systole, corresponding to the basal portions of the left ventricle.

experiencing chest pain, showed (Figure 1A) elevation of the ST segment of 1-3 mm in V2-V6, I, aVL, and II.

The patient was hospitalized in the coronary unit, where an ECG (Figure 1B) made hours later showed Q waves in the anterolateral region, with elevation of the ST segment in V2-V5 and Q waves on the inferior face. With the diagnosis of an extensive anterolateral necrosis of 6 hours of evolution and an absolute contraindication for thrombolytic treatment, coronariography was performed in preparation for primary angioplasty. The test revealed a normal coronary tree. Left ventriculography demonstrated a large ballooned apical zone of aneurysmal appearance, with good contractility of the basal segments and an ejection fraction of 39% (Figure 2).

Treatment was begun with platelet antiaggregants, sodium heparin, and captopril. Heparin was discontinued due to an increase in the size of the subcutaneous frontal hematoma. The patient had no new episodes of chest pain or clinical manifestations of heart failure. The maximum creatine phosphokinase and troponin I concentrations were 303 U/L and 31 µg/L, respectively. Unexpectedly, the ECG evolved (Figure 3) toward the disappearance of Q waves and the appearance of strong negativization of T waves in V2-V6, with prolongation of the QT interval (520 ms). At 7 days of admission, isotopic ventriculography showed normal contractility, with no zones of abnormal regional motility.

The patient showed an abrupt reduction of consciousness on day 5 of admission. Cranial CT demonstrated a small acute left frontal ischemic infarction. Doppler study of the supra-aortic trunks was suggestive of embolism to the left anterior cerebral artery. Cerebral arteriography performed one week later was normal.

The patient was released 15 days after admission with platelet antiaggregant treatment and sublingual

nitrates. She had no neurological sequelae or clinical incidents in the next 6 months.

DISCUSSION

In July 2001, Tsuchihashi et al² published a retrospective analysis of 88 Japanese patients, almost all of them women (86%), with a syndrome characterized by the presence of trigger factors (mental stress, acute disease, or non-cardiac surgery), dyspnea or chest discomfort, electrocardiographic disturbances that were initially identical to those of conventional AMI, minimal or no elevation of creatine phosphokinase and troponin, normal coronary arteries, and apical ballooning of the left ventricle that normalized in a few days.

The electrocardiographic evolution was different from the typical evolution of AMI because the Q waves disappeared and prominent negative T waves appeared in the subacute phase.

As occurred in our patient, in 73% of the cases in the original series the condition was preceded by mental stress, acute disease, or non-cardiac surgery. The pathophysiology of TAD is unknown, and it is postulated that it can be mediated by an increase in sympathetic activity due to the massive discharge of catecholamines,^{4,6} as occurs in episodes of pheochromocytoma and subarachnoid hemorrhage. In the acute phase of subarachnoid hemorrhage, abnormalities in segmental contractility have been described without evidence of coronary lesions or vasospasm, with improvement in the chronic phase, as occurs in stunned myocardium.⁵

For some authors,³ the mechanism that leads to myocardial ischemia could be a transient, diffuse coronary spasm. Rubio Caballero et al⁷ reported a case of variant angina after the subcutaneous administration of epinephrine, in which the epicardial coronary arteries were angiographically normal, which is why the condition was attributed to vasospasm. However, the arti-

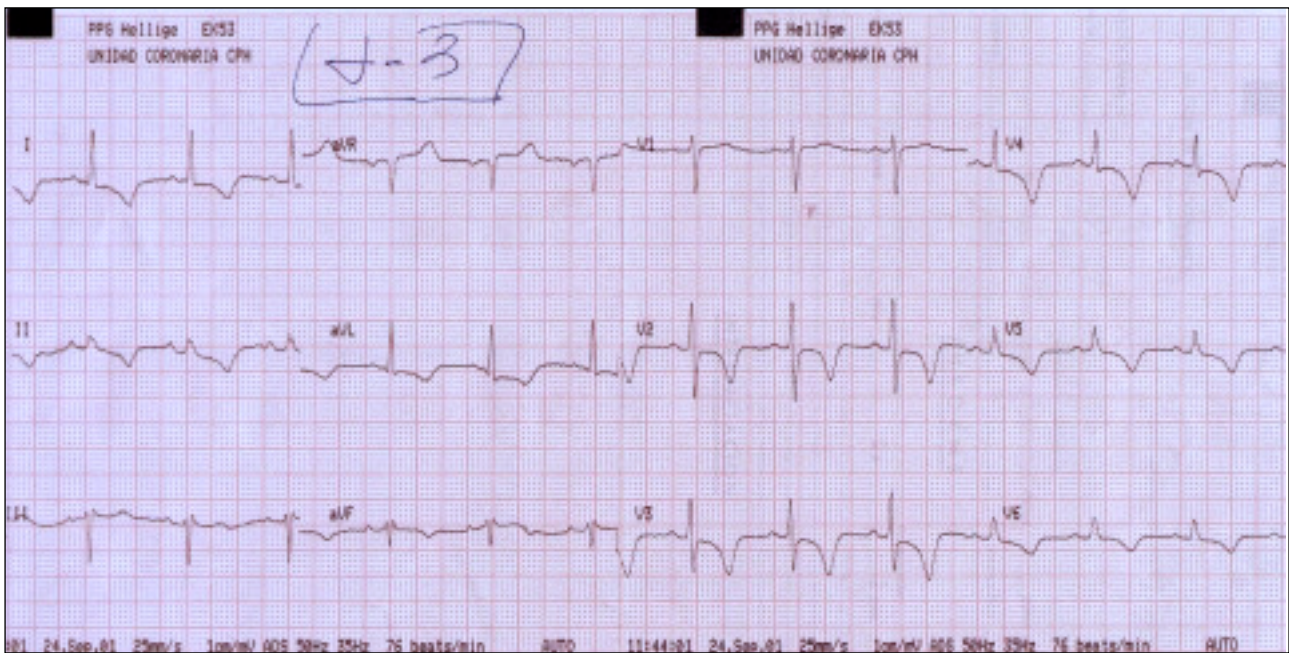


Fig. 3. ECG of the patient on day 4 of admission; the Q waves have disappeared and deep negative T waves are observed in V2-V6.

cle by Tsuchihashi et al² reported vasospasm in only 21% of the 48 patients with TAD in which a pharmacological challenge was used, which is a strong argument against vasospasm as the pathophysiological mechanism of this disorder.

In the article by Tsuchihashi et al, various complications associated with TAD are described, including acute pulmonary edema (22%), cardiogenic shock (15%), and atrial and ventricular arrhythmias. These complications occur more frequently in TAD than in conventional AMI.⁸⁻¹⁰ Paradoxically, the long-term prognosis of TAD syndrome is more favorable. In the series of 88 patients mentioned above, only one death took place during the hospital phase and there was 2% mortality in a mean follow-up of more than a year.²

Cerebral infarction as that which occurred in our patient has not been described previously in association with this syndrome. We think that the embolic origin of the complication has been sufficiently demonstrated by its clinical characteristics and cerebral arteriography without lesions, typical Doppler findings in the supra-aortic trunks, and the exceptional nature of focal cerebral vasospasm,¹¹ which could be invoked as an alternative cause. On the other hand, the thromboembolic phenomena associated with left ventricular dyskinesia of ischemic origin are well known,¹² generally in anterior transmural infarction with apical involvement (which produce emboli mainly in the first 2 weeks of evolution of the infarction).

The shortage of descriptions of TAD in our part of the world may suggest that this disease does not occur in our population. However, the finding of a typical

case soon after the condition was described as a separate entity suggests that it may in fact be underdiagnosed in western countries. For that reason, we think that it is important to suggest that when this syndrome is suspected (a woman with a typical trigger factor and elevation of the ST segment), emergency coronariography should be performed to confirm the diagnosis and eliminate the risk of unnecessary thrombolysis. The prognostic and therapeutic implications of TAD differ from those of conventional AMI and can lead to a different clinical management.

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