

The guidewire was directed toward the VSD and through it. A 7-Fr introducer with the dilator was passed through the defect with utmost care in order to avoid perforating the left ventricular wall. Once the introducer was in place and the dilator withdrawn, a 6/4 Amplatzer Duct Occluder II (AGA Medical Corporation) was loaded into the delivery device and implanted into the VSD by means of the 7-Fr introducer, in accordance with the standard technique (Fig. 2). The result was satisfactory; there was a minimal residual shunt in the device and, at the end of the procedure, the pulmonary pressure had decreased from 85 mmHg to 40 mmHg. The patient was discharged with no complications.

This hybrid procedure has advantages over surgical closure in that it does not require cardiopulmonary bypass, a circumstance that reduces the risk of myocardial and neurological damage. Another advantage is the continuous monitoring by means of TEE, which enables us to assess and correct, in real time, the position of the device and possible interference with the atrioventricular valves.<sup>1</sup>

With respect to percutaneous closure, the hybrid procedure offers several advantages such as the fact that it does not require arterial or venous access. Access to the defect and passage of the guidewire through it is simpler with the transventricular approach because the device penetrates at a right angle and the distance from the penetration site is short. This permits better control during delivery of the device, especially in tortuous VSD and those having irregular borders.<sup>1</sup> The technique employed in isolated percutaneous closure is complex; it requires access to right ventricle from left ventricle, the creation of an arteriovenous loop with the guidewire, and the use of a snare to trap the guidewire and achieve its externalization through the venous side, to ultimately introduce the delivery system and device into the arterial side from the venous side. In all these steps, difficulties often arise which, under conditions of severe hemodynamic deterioration, can prove fatal. Finally, should closure during the hybrid technique prove impossible or if serious complications develop, there always

remains the possibility of immediately incorporating a pump and performing surgical closure.

This case illustrates the fact that a rare complication such as VSD following myectomy can be treated easily and successfully by means of a hybrid procedure, especially in high-risk patients. Moreover, it points out the importance of the collaboration between cardiac surgeons and interventional cardiologists, as well as the need for hybrid operating rooms.

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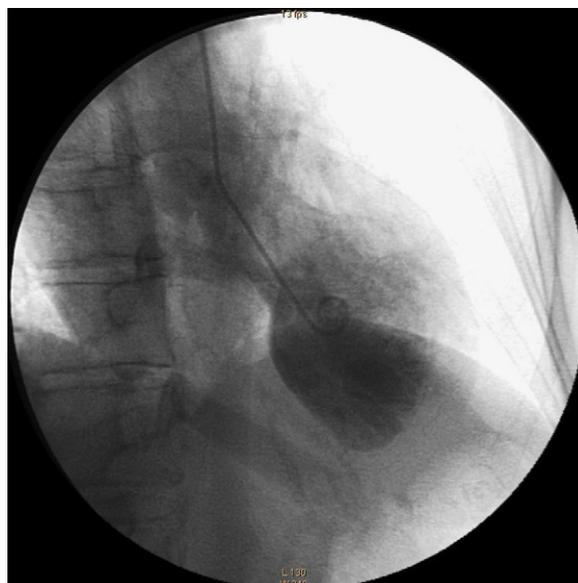
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## Recurrent Tako Tsubo Related to Subclinical Hyperthyroidism

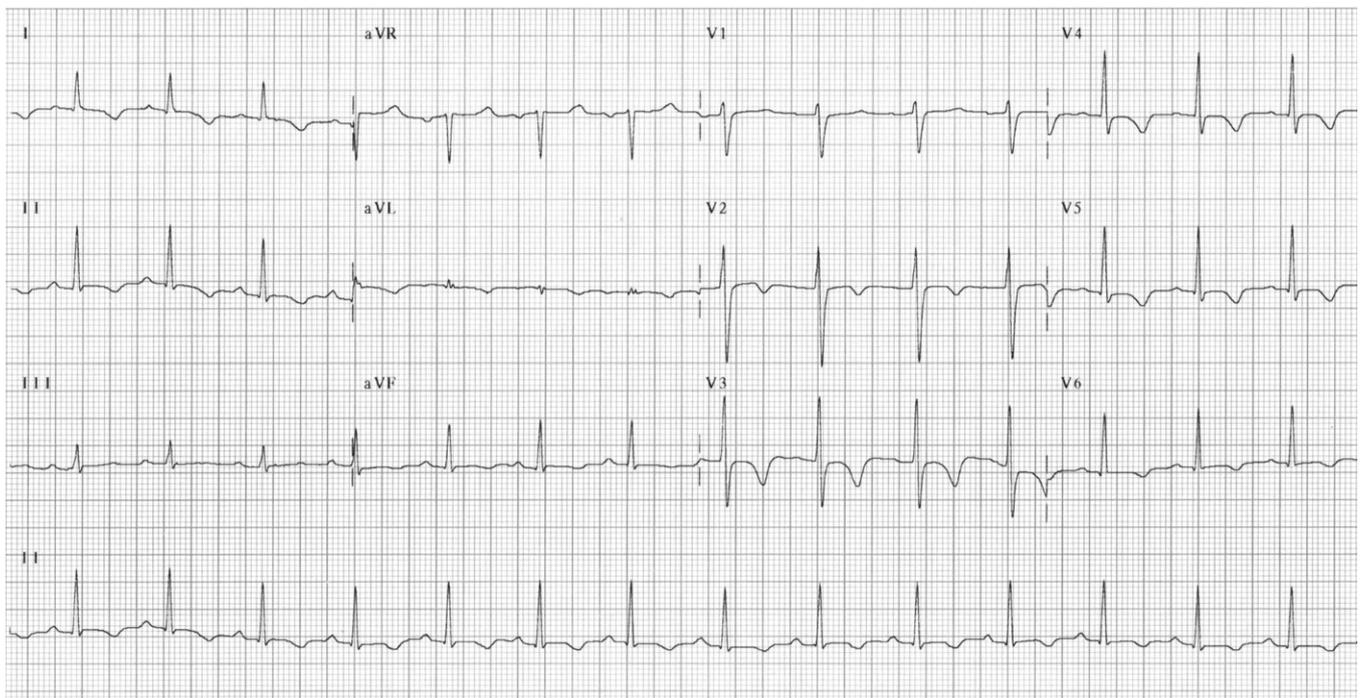
### Síndrome de tako-tsubo recurrente asociado a hipertiroidismo subclínico

#### To the Editor,

We report the case of a 53-year-old woman referred to our hospital for a 2-h history of constrictive chest pains. She had no risk factors and no recent emotional stress. She only had a history of undifferentiated nasopharyngeal carcinoma 4 years ago treated by radiation and chemotherapy with total recovery. Clinical examination was normal, with a blood pressure of 129/83 mmHg and blood oxygenation of 98%, except for a sinus tachycardia of 109 beats per minute. The electrocardiogram (ECG) showed an accelerated sinus rhythm with infero-apico-lateral ST-segment elevation. A diagnosis of acute myocardial infarction was made and she was taken to the hemodynamic laboratory. The coronary angiography demonstrated nonobstructive coronary atheroma. The left ventricular angiography confirmed a severe impaired left ventricular systolic function with apical ballooning and elevated left ventricular end diastolic pressure of 32 mmHg (Fig. 1). She was assisted with intra-aortic balloon pump counterpulsation. The 6-h



**Figure 1.** Left ventriculography (right anterior oblique 30°) showing apical ballooning during systole.



**Figure 2.** Percritical electrocardiogram showing diffuse deep T waves inversion with prolonged corrected QT interval.

troponin T rose to 4 mg/L. The first cardiac ultrasound confirmed severe systolic dysfunction (left ventricular ejection fraction of 30%) with large apical ballooning. An optimal medical treatment based on beta-blockers, diuretics, and angiotensin converting enzyme inhibitors was begun. She evolved well clinically and the circulatory assistance was removed 2 days later. She was discharged at day 10. The follow-up cardiac ultrasound 10 days after discharge showed a total recovery and the ECG had normalized.

Twenty days after discharge, the patient again presented episodes of chest pain. Clinical examination was unchanged except for a remarkable weight loss of 10 kg. She complained of 7 days of diarrhea. The ECG showed diffuse deep T waves inversion with prolonged corrected QT interval (Fig. 2) and the cardiac ultrasound showed typical apical ballooning. The laboratory analysis showed no urinary catecholamine abnormalities but depressed thyroid-stimulating hormone, elevated free triiodothyronine, free thyroxine, thyroglobulin antibody, and thyroid peroxidase antibody led to the diagnosis of Basedow (Graves) disease by thyroid ultrasound. She was treated with carbimazole and the cardiac abnormalities reversed. A cardiac magnetic resonance imaging done 2 months later was normal and she remained event-free at 2 years.

Tako-Tsubo cardiomyopathy (TTC) is a syndrome of profound myocardial stunning precipitated by acute emotional and/or physical stress.<sup>1,2</sup> The growing number of clinical cases of TTC show a wide field of possible etiologies beyond emotional or physical stress.

In our case, we believed that hyperthyroidism was the cause of the TTC. Hyperthyroidism, which was subclinical and not diagnosed during the first episode of TTC, could explain the early recurrence of the apical ballooning. Moreover, the patient was event-free after correction of the hyperthyroidism. Our patient had a classical evolution of apical ballooning syndrome after the first episode, which remained questionable because the cause was not treated, but we thought that medical therapy, especially beta-blockers, could explain the initial favorable evolution. Beta-

blockers are recognized as the main treatment of TTC<sup>3</sup> and could have limited the consequences of hyperthyroidism.

Although the exact cause of TTC remains unknown, several pathomechanisms have been suggested, including epicardial coronary spasm triggered by catecholamines, microcirculatory dysfunction, or direct cardiotoxicity of catecholamines.<sup>1,3,4</sup> The active thyroid hormone T<sub>3</sub> has direct effects mediated by stimulation of specific nuclear receptors such as  $\alpha$ 1,  $\beta$ 1, and  $\beta$ 2 receptors, which in turn leads to specific mRNAs production. Secondly, thyroid hormones interact with the sympathetic nervous system so that hyperthyroidism is associated with hypersensitivity to catecholamines.<sup>5</sup> These actions of thyroid hormone could have induced a catecholamine-mediated cardiotoxicity, which remains the most widely proposed mechanism in TTC, but also coronary vasospasm, which has been associated with hyperthyroidism.

Recurrences of TTC are rare.<sup>2</sup> For the first time, we reported a case of a patient with recurrent episodes of TTC related to primary hyperthyroidism. This is of importance in suggesting a hard link between the two pathologies and establishing a proper treatment plan. Therefore, we think that thyroid function should be assessed in all patients with TTC.

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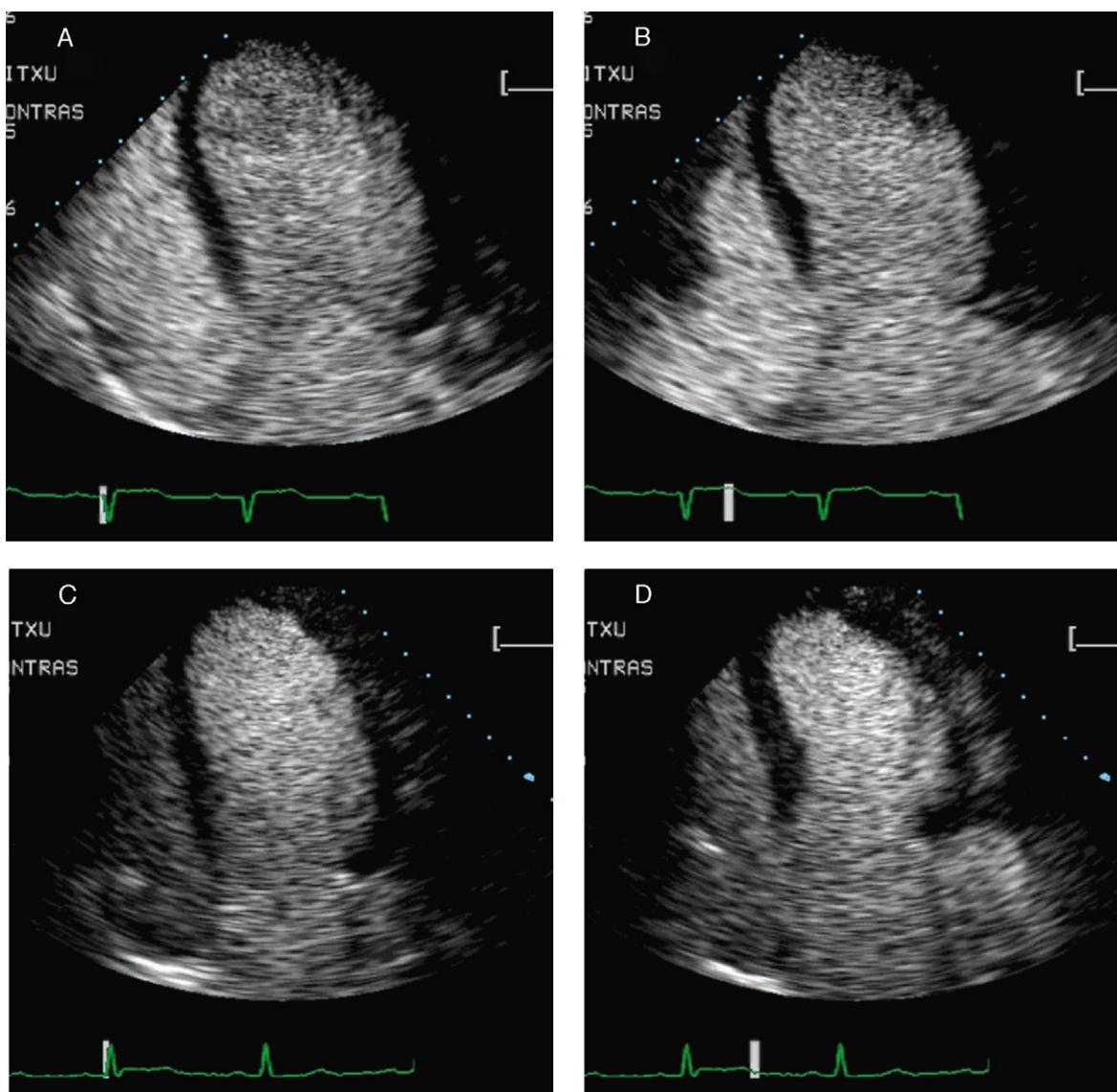
## Contrast Echocardiography and ST-Segment Elevation

### Ecocardiografía de contraste y supradesnivel del segmento ST

#### To the Editor,

We present the case of a 53-year-old man, diagnosed with an anterior acute myocardial infarction treated with fibrinolysis, with natural killer T cells, presenting reperfusion criteria and a maximum creatine kinase value of 516 IU/L. For the exact

quantification of systolic function, contrast echocardiography was performed by the manual injection of a 0.5 mL bolus of SonoVue<sup>®</sup> in 5 mL of physiological saline over 30 s. Simpson's biplane method was used to measure left ventricular end-diastolic index (LVEDI; 168 mL/m<sup>2</sup>), left ventricular end-systolic index (LVESI; 115 mL/m<sup>2</sup>) and left ventricular ejection fraction (32%) (Fig. 1). Coronary angiography showed a 90%-99% lesion in the origin of the left anterior descending artery (LAD). There were no lesions of significance in the circumflex artery and a dominant right coronary artery. The LAD was treated using a Taxus<sup>®</sup> stent.



**Figure 1.** Left ventriculography in end-diastole and end-systole in the apical 4-chamber view during the hospital phase of acute myocardial infarction (A and B) and instants before the anaphylactic reaction (C and D).