

However, compliance is poor. There is no general consensus on dose in WE.

Diagnosis is clinical even though there are tests for assessment and support. The cerebral nuclear magnetic resonance is the modality of choice<sup>1,3</sup> thanks to its specificity (93 per cent) and positive predictive value (89 per cent) being the CT scan discarded due to its low sensitivity. The typical lesions are symmetrical and they affect the medial thalamus, the mammillary bodies, the tectal plate, and the periaqueductal region<sup>8</sup> (Fig. 1). The thiamine deficiency may be analysed using high resolution liquid chromatography or from the activity displayed by erythrocyte transketolase<sup>1,3,7</sup> where it acts as a cofactor. In our patient we were unable to obtain any levels due to an extraction mistake that made our sample useless. The fast recovery after the administration is the best diagnosis as it occurred with our patient.

Vitamins and other coexisting deficits should be supplemented. Magnesium is an important cofactor of the thiamine pathways. One recent study confirmed a significant increase of the activity of erythrocyte transketolase in the group treated with magnesium and thiamine compared to the group that received thiamine only, which means that the administration of magnesium plus thiamine may speed up the recovery of metabolic pathways.<sup>9,10</sup>

The prognosis of WE is closely associated with the precocity of diagnosis and treatment.<sup>1,3,5</sup> Its low cost and innocuousness support its early administration. The residual deficits are common as it occurred with the case presented here.

We would like to finish with a thought. The diagnosis of WE is clinical and the response to treatment with thiamine is the best test there is. We should always take it into consideration with non-related neurological manifestations in patients with risk factors (increased metabolic demand, or deficits). When in clinical suspicion, we should supplement with IV thiamine, since it has a good profile of safety and innocuousness that may prevent and even stop the devastating effects of WE.

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## Extracorporeal membrane oxygenation support for Takotsubo syndrome and long QT after cardiac surgery<sup>☆</sup>

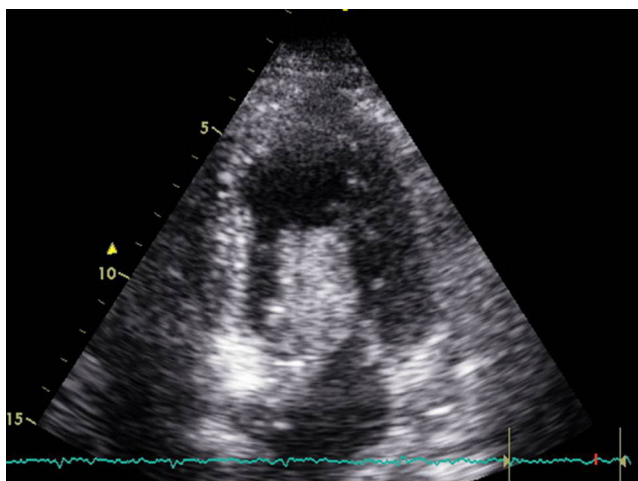


## SopORTE con oxigenación de membrana extracorpórea en un síndrome de Takotsubo y QT largo tras cirugía cardíaca

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Dear Editor,

Takotsubo syndrome is a type of cardiomyopathy characterized by transient left ventricular dysfunction associated to electrocardiographic changes similar to those of acute myocardial infarction, though without evidence of coronary disease, and with scantily altered myocardial enzyme levels. The syndrome is also known as transient apical dysfunction, "apical ballooning" or stress-induced cardiomyopathy, since it generally affects postmenopausal women following some stressing event. The diagnostic criteria of Takotsubo syndrome are: acute electrocardiographic anomalies such as ST-segment elevation or T-wave inversion; the absence of obstructive coronary disease; akinesia or transient dyskinesia of the left ventricle; and the absence of traumatic brain injury, intracranial hemorrhage, pheochromocytoma,



**Figure 1** Apical projection view of the four chambers, showing the atrial myxoma penetrating into the left ventricle through the mitral valve.

myocarditis or hypertrophic cardiomyopathy.<sup>1</sup> The incidence and prevalence of the syndrome are not known, and few cases have been reported in patients subjected to cardiovascular surgery. We present the case of a woman operated upon due to an atrial myxoma, who in the postoperative period developed cardiogenic shock secondary to Takotsubo syndrome.

A 68-year-old woman with arterial hypertension and non-insulin dependent diabetes reported to the emergency service due to clinically manifest heart failure for the last two weeks. Upon admission she presented resting dyspnea and palpitations. Rapid atrial fibrillation was detected, requiring pharmacological cardioversion. The echocardiographic study (Fig. 1) revealed a large left atrial mass ( $1.8 \times 7$  cm in size) protruding toward the left ventricle and causing secondary mitral valve stenosis and atrial dilatation, with a preserved left ventricular ejection fraction (LVEF). Preoperative coronary angiography showed no lesions.

Surgery was performed four days later, with transeptal removal of the myxoma. There were no relevant incidents other than a tendency toward hypoxemia. The clamping and extracorporeal circulation times were 52 and 83 min, respectively. Intraoperative echocardiography confirmed the absence of any remnant myxoma tissue, with a normal LVEF. The patient remained stable and subjected to mechanical ventilation during her first hours of stay in the Intensive Care Unit (ICU) due to the aforementioned hypoxemia, with  $\text{FiO}_2 = 0.6$  and  $\text{PEEP} = 10\text{--}12$  cmH<sub>2</sub>O. Eight hours after surgery she suffered a sudden hypertensive crisis (systolic blood pressure  $>220$  mmHg) followed by hypotension and cardiac arrest with pulseless electrical activity (electromechanical dissociation). Resuscitation maneuvering restored the pulse within a few minutes. The patient was in cardiogenic shock refractory to high-dose vasoactive medication (adrenalin, dobutamine, noradrenalin); as a result, venous-arterial extracorporeal membrane oxygenation (V-A ECMO) was therefore finally decided.

Transthoracic echocardiography revealed severe left ventricular dysfunction (LVEF  $<20\%$ ), hyperdynamic basal segments and akinesia of the middle and apical seg-

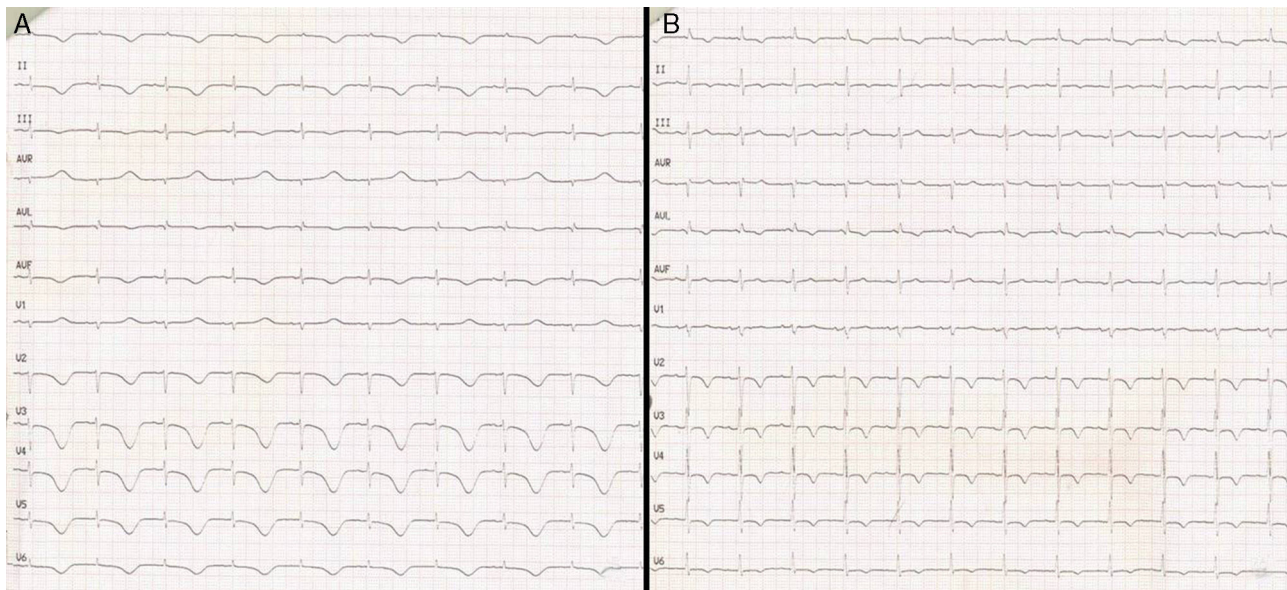
ments—these findings being typical of Takotsubo syndrome. There were no significant cardiac enzyme elevations over the following days (taking surgery into account), with a maximum ultra-sensitive troponin I concentration of 4000 pg/ml (normal 2–15.6 pg/ml). In contrast, the electrocardiographic tracing showed significant changes with respect to the previous recordings, with a long QTc (680 ms) and the appearance of inverted T-waves on precordial leads during several days (Fig. 2). The electrolyte profile and rest of the laboratory test parameters were normal.

Venous-arterial ECMO was maintained for four days, allowing the withdrawal of vasoactive drug support, and followed by a favorable clinical course. The echocardiographic studies revealed gradual improvement of ventricular function. The patient was extubated a few days later and moved to the hospital ward, where she remained until discharge in good functional condition. The last echocardiographic exploration showed slight anterior septal and inferior hypokinesia, with globally preserved systolic function.

In this case the diagnosis of Takotsubo syndrome was established from the echocardiographic findings, the absence of previous coronary lesions, scant alteration of the cardiac enzyme levels despite the ventricular dysfunction and surgery, and the precordial electrocardiographic tracings (inverted T-waves and long QT syndrome, with normal electrolyte levels). There has been an increase in the number of diagnoses of this syndrome in recent years,<sup>2</sup> also in cardiovascular surgery patients,<sup>3–5</sup> though Takotsubo syndrome has not been previously described in the postoperative period of atrial myxoma resection.

The etiology and pathophysiology of Takotsubo syndrome have not been fully established, though a number of mechanisms have been proposed, such as coronary vasospasm, coronary microvascular functional anomalies, and particularly catecholamine-mediated cardiotoxicity. Probably because of this, the main risk factor is considered to be stress (both physical and mental), which is present in different diagnostic tests and surgical procedures—especially those of an emergent or urgent nature.<sup>6</sup> Cardiac surgery and extracorporeal circulation generate important stress, and may intrinsically account for the appearance of Takotsubo syndrome, particularly if the epidemiological profile of the patient and the echocardiographic and electrocardiographic features are consistent with the diagnostic criteria, as in our case. The differential diagnosis includes coronary embolism or poor myocardial protection. The typical echocardiographic findings, with scant enzyme alterations, brief surgery without incidents, and previous hemodynamic stability with a normal first electrocardiographic tracing allow us to reasonably discard both of the aforementioned disorders. In recent years, evidence has been gained suggesting that magnetic resonance imaging may be useful in establishing a differential diagnosis with other disorders such as myocarditis or coronary embolism,<sup>7</sup> though this technique could not be used in our case.

Of note is the appearance of a long QT interval on the electrocardiographic tracing. The association between Takotsubo myocardial pathology and transient prolongation of the QT interval has been documented,<sup>8,9</sup> being more frequent in patients with some form of heart disease, previous long QT syndrome, or genetic alterations. In Takotsubo syndrome,



**Figure 2** (A) Twelve-lead electrocardiographic tracing corresponding to day two after onset of the condition, with a QTc interval of 680 ms. (B) Electrocardiogram corresponding to evolutive day 10, showing a QTc interval of 475 ms, but with persistent T-wave inversion.

electrocardiographic normalization occurs approximately two months after the episode. Although malignant ventricular arrhythmias, sudden death or cardiac block are infrequent, there have been reports of such situations. Increased QT prolongation (QTc > 500 ms) has been identified as a risk factor for complications of this kind; close monitoring of the cardiac rhythm is thus required, with the prevention or treatment of arrhythmias and the implantation of a pacemaker if necessary.

In conclusion, Takotsubo syndrome is an infrequent cause of cardiogenic shock in the postoperative period of heart surgery, though it must be considered in patients with apparently uncomplicated surgery, normal coronary vessels and compatible echocardiographic findings, since the prognosis is generally good when adequate supportive treatment is provided.

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