

**B) BRIEF CASE REPORTS**

**Acute kidney failure in the context of a Tako-Tsubo syndrome**

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**To the Editor,**

Tako-Tsubo syndrome, stress cardiopathy or transient apical ballooning, is a very rare clinical condition, and consists of a reversible acute heart failure. It is common in middle-aged women, following intense periods of psychological or physical stress.<sup>1</sup>

As described in the nineties, it consists of an acquired cardiomyopathy that simulates all clinical, analytical, electrocardiographic and echocardiographic aspects of an acute, usually apical, attack.<sup>2</sup> It is usually presented as an acute heart failure, with typical or atypical chest pain. It also presents with TS segment alterations and/or repolarisation on the electrocardiogram (ECG) and positive troponin curve. The electrocardiogram shows a severe myocardial dysfunction with a reduced ejection fraction. However, studies for detecting coronary ischaemia (stress ergometry or coronary angiography) are, by definition, negative.<sup>3</sup>

The most accepted histopathological theories discuss transient microvascular spasms caused by catecholaminergic discharge in predisposed patients. More recent studies champion a neurocardiogenic theory.<sup>4</sup>

Prognosis is almost always benign, with complete myocardial function recovery, and the relapse rate is between 2% and 10%.<sup>5</sup>

As with all acute heart disorders, Tako-Tsubo syndrome can induce acute or chronic renal failure through the classic mechanisms of secondary kidney involvement. We describe the case of a partially recovered acute kidney failure after resolving the heart disorder.

The patient is a 51-year-old female, smoker, with no relevant history of events. She came to the hospital in March 2010 with clinical symptoms of oedemas in her lower limbs that had developed over a month. She did not have dyspnoea, chest pain or any other symptoms. The emergency department found that she had renal function deterioration with 1.54mg/dl creatinine (baseline creatinine 1.1mg/dl) and microalbuminuria (95mg/day). The rest of the analyses were within the normal range and the ECG showed negative T waves for leads V5 and V6.

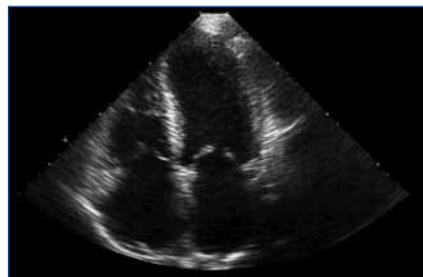
She was admitted for examination; the most noticeable result was from a transthoracic echocardiogram that revealed a severely depressed systolic function (ejection fraction of the left

ventricle of 25%), with overall hypokinesia and slight hyperechogenicity of the ventricular wall (Figure 1). The other complementary tests were negative. The abdominal ultrasound found small kidneys (10cm and 11cm) with good corticomedullary differentiation (Figure 2).

Two weeks later, cardiac magnetic resonance imaging was performed showing that the ventricular function and heart contractility were normal. A subcutaneous fat biopsy was performed to verify possible amyloidosis, which was also negative. Stress ergometry performed three months later was negative for myocardial ischaemia.

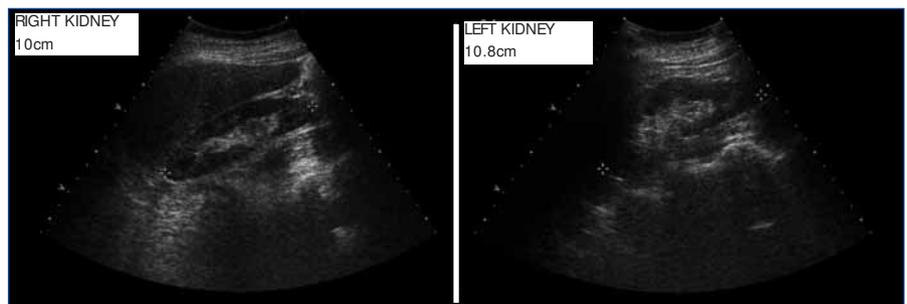
In view of this, the patient was diagnosed with transient myocardial dyskinesia. Performing the ventriculography was not considered given that she had good myocardial recovery, and so as to not worsen the kidney failure. When the doctor spoke to her again, she mentioned that her family situation had been extremely stressful days before her symptoms started.

After the renal function reached a maximum creatinine level of 2mg/dl, it started to improve. It was 1.4mg/dl in the last examination performed in the nephrology department. Furthermore, ventricular function continued to be normal in later ultrasound scans.



Transthoracic echocardiogram performed while the Tako-Tsubo syndrome was in acute stages, with hypokinesia in the myocardium's most distal regions

**Figure 1.** Transthoracic echocardiogram



Kidney ultrasound that shows small kidneys with no echogenicity alterations.

**Figure 2.** Kidney ultrasound

This is a representative case of a type 1 cardiorenal syndrome (kidney involvement secondary to acute heart failure) in the context of a recently reported complex pathology, such as the Tako-Tsubo syndrome. It is a prerenal failure, in which an adequate recovery of renal function should be expected.

While there are no good early markers available for kidney dysfunction, renal function must be closely monitored for any patient with an acute heart disease for an early diagnosis. If the cardiologist and the nephrologist work together in acute management of the treatment, patient prognosis could improve.

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## Chronic pulmonary bleeding as the first sign of microscopic polyangiitis associated with autoimmune thyroiditis

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### To the Editor,

Microscopic polyangiitis (MPA) is defined as a non granulomatous, anti-neutrophil cytoplasmic antibodies (ANCA)-positive systemic necrotising vasculitis which affects small vessels. Kidneys and lungs are the most frequently main affected organs.<sup>1</sup> Pulmonary bleeding in MPA is usually acute or even fulminant.<sup>2,3</sup> Pulmonary bleeding as part of chronic MPA is not described as often. Autoimmune thyroiditis is associated with glomerulopathies, especially with membranous nephropathy.<sup>4</sup>

We describe a patient with chronic pulmonary bleeding as a sign of MPA; the examination also showed that she presented with autoimmune thyroiditis.

A 54-year-old woman under examination for a year and a half in another hospital for iron deficiency anaemia of several years. She was being treated with orally-administered iron and needed transfusions. They found that she had renal failure, proteinuria and microhaematuria during this examination, and she was referred to us. Patient history: arterial hypertension for a year. Clinically, she presented with important asthenia and medium-effort dyspnoea. Physical examination: she did not present with fever, blood pressure was 140/96mm Hg, and had no oedemas on lower limbs. The analytical study showed: haemoglobin: 8.6g/dl; iron: 33.5µg/dl; total iron-binding capacity (TIBC): 264µg/dl; ferritin: 137ng/ml; urea: 67mg/dl; creatinine: 1.50mg/dl; estimated glomerular filtration rate (eGFR) (according to Modification of Diet in Renal Disease [MDRD4]): 38.46ml/min/1.73m<sup>2</sup>; uric acid: 7.19mg/dl; total cholesterol: 233mg/dl (LDL: 163; HDL: 33.7);

triglycerides: 178mg/dl. Normal/negative immunoglobulins, antinuclear antibodies (ANA), anti-DNA, rheumatoid factor (RF), anti-glomerular basement membrane antibody treatment (anti-MBG) and anti-transglutaminase antibodies (ATA); positive myeloperoxidase (MPO)-ANCA, negative PR3-ANCA. High resolution electrophoresis (HRE) of serum: no monoclonal band. Thyroid-stimulating hormone (TSH): 326mIU/l (0.350-4.940), free thyroxine (T4): 0.11ng/dl (0.7-1.48); anti-thyroid peroxidase antibodies (anti-TPO) >1000IU/ml. Negative Mantoux test. Negative hepatitis B surface antigens (HbsAg), hepatitis B anti-core antibodies (anti-HBc), hepatitis C antibodies (anti-HCV) and human immunodeficiency virus (HIV) tests. Cytomegalovirus (CMV) serology test and Epstein-Barr virus (EBV): previous exposure. Urine showed a proteinuria of 1.3g/24hr (moderately selective glomerular proteinuria), sediment with 60-100 red blood cells (30%-50% dysmorphic) and hyaline-granular casts. Urine culture: negative. Chest X-ray and electrocardiogram (ECG) were normal. Abdominal ultrasound: kidneys of a normal size with slight cortical thinning. Chest computerised tomography (CT) showed images of 'ground-glass opacity' on both sides in the middle and lower fields and a bronchoscopy with a bronchoalveolar lavage was indicated, showing 80% siderophores. The malignant cell cytology and cell cultures were negative. The patient was interviewed, revealing that she sporadically presented with a slight, dark-coloured expectoration. The renal biopsy (Figure 1) showed a focal and segmental necrotising glomerulonephritis, and approximately a third of the glomeruli were sclerotic, interstitial fibrosis and tubular atrophy in 30% of the cylinder. The immunofluorescence study showed slight deposits of IgM, C3 and C4 on capillary walls and the mesangium. Given the pulmonary bleeding, the positive ANCA, and the focal necrotising glomerulonephritis with scarce immune deposits, MPA was diagnosed, associated with autoimmune thyroiditis-induced hypothyroidism. Treatment with prednisone at 50mg/day was started for 8 weeks, gradually reducing the dosage, and six 750-mg cyclophosphamide boli, which