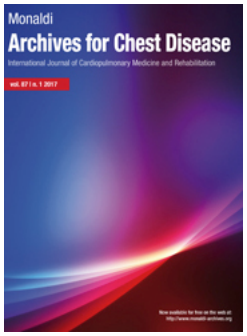

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TakoTsubo secondary to acute kidney disease

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Abstract

We report the case of a 62-year-old man who was admitted to the cardiac department for TakoTsubo and ACC by torsades de point, secondary to acute kidney disease. We decide to discharge with a portable defibrillator. One month after Cardiac Magnetic Resonance showed a complete recovery of left ventricular function.

Case Report

A 62-year-old man with chest pain described as sharp, left sided, associated with nausea and vomiting, anorexia and consequently weight loss during the past week. Because of an episode of acute gout he assumed a NSAID (Nimesulide). On physical examination he had blood pressure 80/40 mmHg, heart rate 54 bpm. He presented also an anasarcatic state (including pleural and pericardial effusion) caused by hypoalbuminemia (Albumin 2,9 g per milliliter) and severe anemia (Hb 7,9 g per deciliter), associated with oligo-anuria. He was oriented, apiretic and eupnoic.

The patient had arterial hypertension treated with pharmacological therapy (ramipril and amlodipin), hyperuricemia, hypercholesterolemia. He suffered from seizures in the past not investigated by the physicians.

Laboratory tests showed acute kidney injury (creatinine 17,3 mg per deciliter, azotemia 590 mg per deciliter), acute metabolic acidosis (pH 7.18, HCO3- 15 mEq per liter), hyperkalemia (K 5.1 mEq), caused by FANS and ACE- inhibitors abuse. His electrocardiogram showed sinus bradycardia without ischemic changes. In the emergency room, a transthoracic echocardiogram (TTE) was performed and showed normal biventricular size and function (LVEF 60%), absence of valvulopathies. During hospitalization for renal failure, he had a progressive worsening of heart function with echocardiographic signs of apex and anterolateral wall akinesia (LVEF 30%) and normal electrocardiogram. A coronary angiography was performed and documented the absence of CAD. We made diagnosis of TM confirmed also through cardiac magnetic resonance (EF 39%) (Figure 1).

Acute kidney injury was treated with albumin and bicarbonates infusion. An ultrafiltration therapy was not needed. Due to severe anemia, a transfusion was performed. After the diagnosis of TM, the patient was treated with loop diuretic, betablocker, anti-aldosterone agent. In the following hours after the diagnosis, the electrocardiogram showed also a progressive lengthening of the QT tract and consequently had a cardiac arrest due to torsades de pointes treated with a single DC shock.

After 2 hour stabilized clinical conditions, he suffered from a tonic-clonic epileptic crisis resolved after valium administration. The post-crisis electroencephalogram showed nonspecific slow center-anterior electrical changes and the magnetic resonance imaging brain was negative for acute injury. A therapy with betablockers and anti-aldosterone agent was performed and continued.

Due to acute renal failure, a treatment with ace inhibitors was not performed. Subsequently, improved kidney function, improved cardiac contractility with consequent progressive reduction of the QT tract and absence of further arrhythmic events (QT max 620 msec QT at dimission 460 msec).

Upon discharge, the echocardiogram showed apical hypokinesia, with partial improvement of systolic function (EF 48%).

The kidney also showed progressive improvement (creatinine at dimission 1.5 mg per deciliter, GFR 49 ml/min). Therefore, the patient was discharged with a portable defibrillator and in clinical good conditions.

One month after discharge he was subjected to a second cardiac magnetic resonance showed a complete recovery of left ventricular function (EF 58%) (Figure 2), so the portable defibrillator was deleted.

Discussion

TM is a reversible cardiomyopathy characterized by systolic abnormality of the left ventricle's apical area resulting in "apical ballooning" appearance in the absence of coronary artery disease. Catecholamines play an important role in the pathogenesis and pathophysiology of TM. In fact, the most accepted theories are catecholamine-induced cardiotoxicity and microvascular dysfunction, in addition to the complex and integration of neuroendocrine physiology involving the cognitive centers of the brain and hypothalamic-pituitary-adrenal axis. The prevalence of TM is 1.0-2.5% which especially occur in post-menopausal women [1-3].

TM was responsible of numerous arrhythmic events including QT elongation with numerous torsades de pointes and an episode of seizure. The absence of acute lesions at magnetic resonance imaging brain confirmed the hypothesis that TM reduced the epileptogenic threshold and was responsible for a seizure in the patient who has never suffered from epilepsy, so he didn't start an anti-epileptic therapy.TM was a secondary event of acute kidney injury which was also responsible for anasarcatic state and a severe anemia and resided in chronic kidney disease. Probably this situation was the trigger on which a TM developed. Patients with TM had a higher prevalence of neurologic or psychiatric disorders [4], moreover patients with TM and chronic kidney disease more often experience severe complications in the acute phase of the disease, particularly sudden cardiac arrest [5].

As concern the epileptic threshold reduction, it is known hormones influence brain excitability, however, both epileptic seizures and antiepileptic drugs may alter hormone secretion and metabolism. Progesterone, testosterone, adrenocorticotropin and desoxycorticosterone are responsible for an increase in seizure threshold. Therefore, after an epileptic seizure, an increase in serum concentrations of prolactin, cortisol, adrenocorticotropin, triidothyronine, thyroxin, thyrotropin, luteotropin, follicular stimulating hormone and growth hormone is found.

These changes may persist for two hours, while prolactin concentration even for 24 hours after a seizure. Recognition of the relationship between epilepsy and hormonal system is necessary to obtain better understanding the epileptic threshold reduction [6]. Therefore, hyperazotemia

was associated with various disorders (such as hepatic encephalopathy), due to varied pathophysiological mechanisms among wich false neurotrasmitters [7].

Conclusions

We present an unusual case of TM associated with acute kydney injury and subsequent chronic kidney disease. Evidences confirm that TM could be responsible of acute kidney injury, but cases of TM secondary to an acute kidney injury have not been described in literature.

Acute kidney injury should be considered as a trigger for TM that was responsible of arrhythmic events and seizure.

In our opinion false neurotransmitters (related to hyperazotemia) could induce an adrenergic stress to myocardiocytes determining the development of TM.

Further studies to be necessary to confirm the link between acute kidney injury and TM. In our patient, the outcome in TM is favorable, with completely recovery both of cardiac and kidney functions.

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Figure 1. Different sections of cardiac magnetic resonance during the hospitalization. The typical apical ballooning of TakoTsubo syndrome is visible.

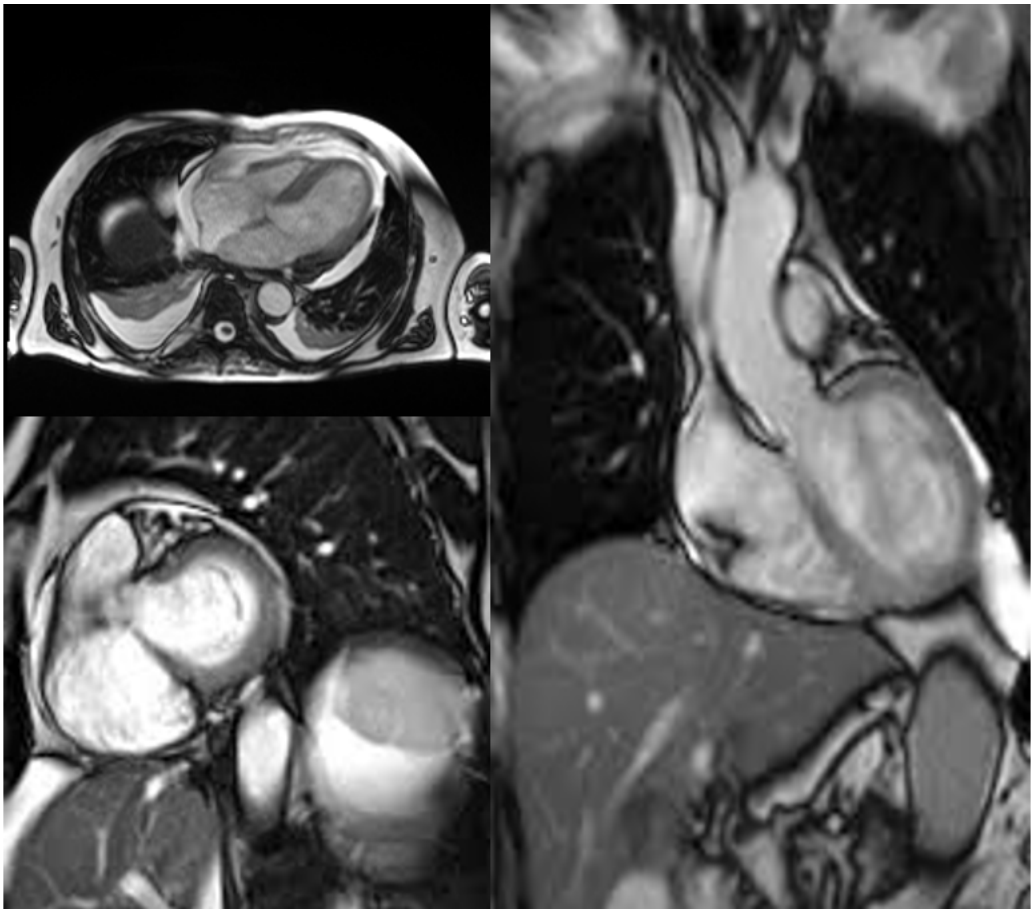

Figure 2. Different sections of cardiac magnetic resonance after one month of discharge. A complete recovery of left ventricular function is visible.