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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 cardiocirculatory arrest by torsades de point, secondary to acute kidney disease. We decide to discharge with a portable defibrillator. One month later, cardiac magnetic resonance showed a complete recovery of left ventricular function.

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Case Report

A 62-year-old man described his chest pain as sharp and 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 nonsteroidal anti-inflammatory drug (NSAID) (nimesulide). On physical examination, he had a blood pressure of 80/40 mmHg and a heart rate of 54 bpm. He also presented an anasarcatic state (including pleural and pericardial effusion) caused by hypoalbuminemia (albumin 2.9 g per mL) and severe anemia (hemoglobin 7.9 g per dL), associated with oligo-anuria. He was oriented, apiretic and eupnoic.

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

Laboratory tests showed acute kidney injury (creatinine 17.3 mg per dL, azotemia 590 mg per dL), acute metabolic acidosis (pH 7.18, bicarbonate 15 mEq per liter), hyperkalemia (K 5.1 mEq), caused by nonsteroidal anti-inflammatory drugs and angiotensin-converting enzyme (ACE) inhibitors abuse. His electrocardiogram showed sinus bradycardia without ischemic changes. In the emergency room, a transthoracic echocardiogram was performed and showed normal biventricular size and function [left ventricular ejection fraction (LVEF) 60%] and 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 a normal electrocardiogram. A coronary angiography was performed and documented the absence of coronary artery disease. We made a diagnosis of TakoTsubo cardiomyopathy (TM) confirmed also through cardiac magnetic resonance [ejection fraction (EF) 39%] (Figure 1).

Acute kidney injury was treated with albumin and bicarbonate 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 a loop diuretic, β-blocker, and anti-aldosterone agent. In the following hours after the diagnosis, the electrocardiogram also showed a progressive lengthening of the QT tract, and consequently had a cardiac arrest due to torsades de pointes treated with a single defibrillator cardioverter shock.

After 2 hours of 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 of the brain was negative for acute injury. A therapy with β-blocker and anti-aldosterone agents was performed and continued.

Due to acute renal failure, 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) were observed.

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

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

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 an "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 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 occurs in post-menopausal women [1-3].

TM was responsible for numerous arrhythmic events including QT elongation with numerous torsades de pointes and an episode of seizure. The absence of acute lesions in the magnetic resonance imaging brain confirmed the hypothesis that TM reduced the epileptogenic threshold and was responsible for a seizure in the patient who had never suffered from epilepsy, so he did not start an antiepileptic therapy.TM was a secondary event of acute kidney injury, which was also responsible for an anasarcatic state and 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 a concern for 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 2 hours, while prolactin concentration may persist for 24 hours after a seizure. Recognition of the relationship between epilepsy and the hormonal system is necessary to obtain a better understanding of epileptic threshold reduction [6]. Therefore, hyperazotemia was associated with various disorders (such as hepatic encephalopathy) due to varied pathophysiological mechanisms, among which false neurotrasmitters [7].

Conclusions

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

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

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

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

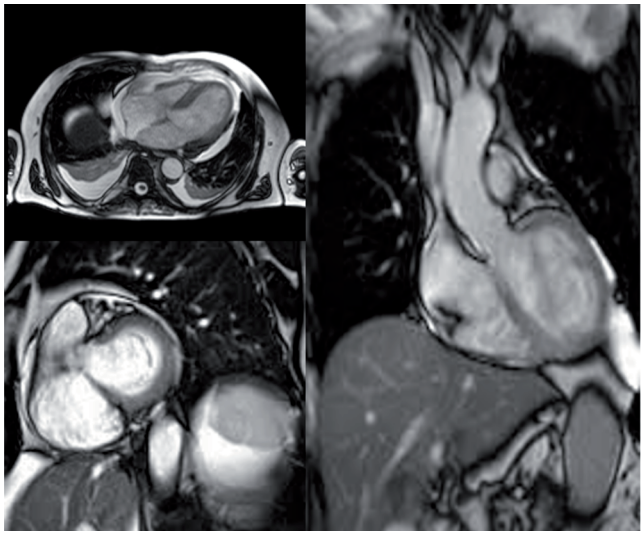

Figure 1. Different sections of cardiac magnetic resonance during the hospitalization. The typical apical ballooning of TakoTsubo syndrome is visible.

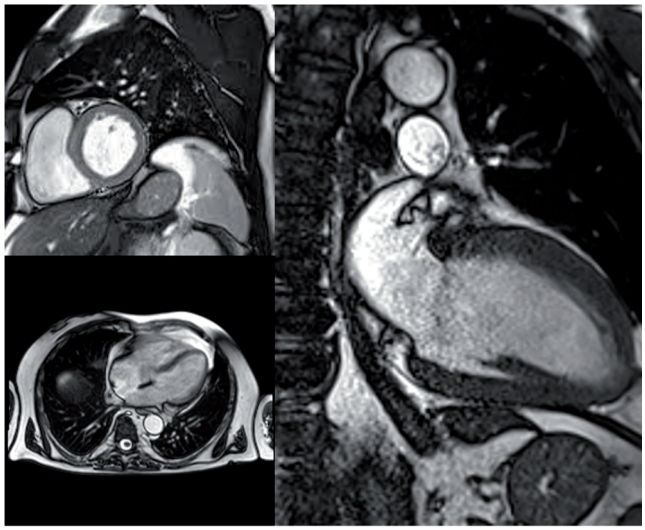

Figure 2. Different sections of cardiac magnetic resonance after one month of discharge. A complete recovery of left ventricular function is visible.

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