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Tako-Tsubo Syndrome Induced by Emotional Stress Leading to Cardiogenic Shock and Cardiocirculatory Arrest in a 56 Old-Year-Old Patient with Deficit of Protein C

Mayssem gabsi*, Dr Sarra chenik chenik, Kenza zniber kenza, Dr Karima taamallah cardio, Bouthaina besbes, Pr Wafa fehri wafa

Service of Cardiology of the Military Hospital of Tunisia

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***Corresponding author:** Mayssem Gabsi

Abstract **Case Report**

Transient left ventricular (LV) dysfunction syndrome, or Tako-tsubo syndrome, occurs following intense emotional or physical stress and simulates the clinical presentation of an acute myocardial infarction. The prognosis is favorable with normalization of wall motion abnormalities within weeks. In this case we report a young female patient who developed sudden chest pain and consciousness disorder after a profound stress (mother's death).The patient had characteristic feature of progressive pulmonary edema. Her symptom worsened gradually leading to herintubation. An immediately performed echocardiography showed akinetic mid-to-distal portion of the left ventricular chamber and hyperkinetic activity in basal segment with an ejection fraction (EF) of 25%. Coronary angiography showed normal coronary arteries. After 6 days of monitoring she was extubated and had a cardiac MRI that confirmed the diagnosis of tako-tsubo.

Keywords: Tako-Tsubo syndrome; cardiogenic shock; deficit of protein C.

INTRODUCTION

Tako-tsubo syndrome (TTS) is characterized by reversible left ventricular (LV) apical wall motion abnormalities with chest symptoms and electrocardiographic changes and relatively minor myocardial enzymatic release, which mimics acute coronary syndrome in patients without angiographic stenosis in the epicardial coronary artery. Although no predictors of clinical outcome have been established, the general prognosis is considered to be favorable, as previously reported. This condition is considered to be related to disproportionate catecholamine secretion, which may stun the myocardium. As a result, catecholamine infusion can worsen the phenomenon and therefore TTS presenting with cardiogenic shock is difficult to handle. However, some investigators have reported cases with various complications cardiogenic shock Fortunately, Cardiogenic shock rarely complicates TTS hence the interest of our case.

CASE REPORT

A 56 year- old- woman was transferred to our hospital and admitted to the emergency department with the acute onset of chest pain and dyspnea in a context of psychological situation (her mother's death). She had been given medication to treat type 2 diabetes mellitus since 1998 and had repeated profound venous thrombosis. Therefore, she has been treated with Sintrom (acenocoumarol) for thrombophilia related to a deficit of C protein.

During admission, the patient's pulse rate was 80bpm, her blood pressure was 100/60 mm Hg, and her temperature was 37.8°C.Routine laboratory studies disclosed the following values (reference ranges shown parenthetically): leukocyte count, 7.5 × 109 /L; hemoglobin, 12.7 g/dL; platelet count 237 × 109 /L; aspartate aminotransferase 24 U/L potassium 4.3 mEq/Land C-reactive protein 20.55 mg/dL a mild increase in troponin I (500 ng/mL) TP 23% INR 4.45.

An electrocardiogram showed a sinus tachycardia and inverted T waves in inferoapicolateral. An acute coronary syndrome was suspected, and a loading dose of dual antiplatelet therapy was administered (250 mg Aspirin and 600 mg Clopidogrel).

The patient rapidly deteriorated (her hemodynamic status) and required intubation, norepinephrine infusion (1mg /h) and fluid expansion.

Transthoracic echocardiography (Figure1) showed severe left ventricular dysfunction (left ejection fraction of 25%), with akinetic apical and midventricular segments while basal segments were hyperkinetic, and a mild (< 10 mm) pericardial effusion localized in front of the right ventricle. Emergent coronary angiography (Figure 2) showed no significant lesions in the epicardial arteries. Note that the patient presented a circulatory cardiac arrest at the catheterism room which was recovered with 1mg adrenaline and 1 min of cardiac massage.

The patient was extubated at the 7th day with success. In post extubation she presented confusion, delirium episodes and extreme agitation. She was treated with halopéridol and benefited from angioMRI exploration which showed: endoluminal defect at the left sinus sigmoid level framing with cerebral thrombophlebitis, diffuse micro bleeds sus tentoriels related with lesions. She benefited from curative dose of HNF.

This presentation revealed a case of mid-ventricular TTS complicated with concomitant cardiogenic shock. The patient benefited at distance from cardiac MRI (Figure 3) which confirmed the diagnosis.

An echocardiography after one week revealed restoration of left ventricular function and the patient was discharged.

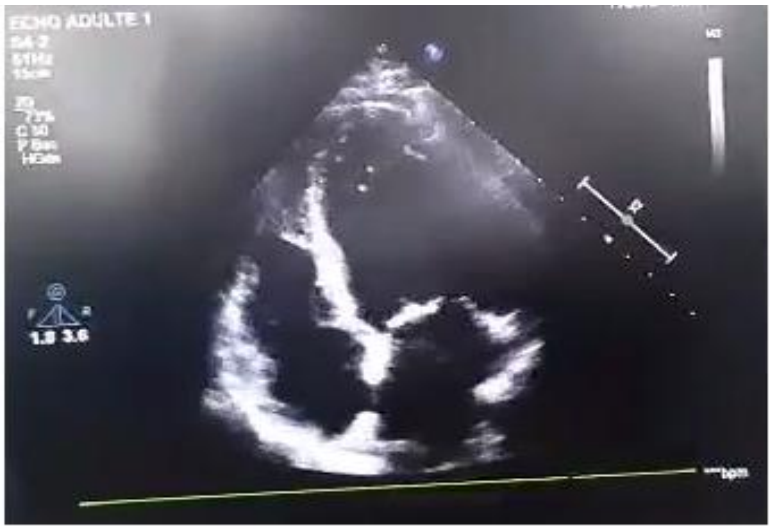

Fig-1: Transthoracic echocardiography (Apical4 cavities view)

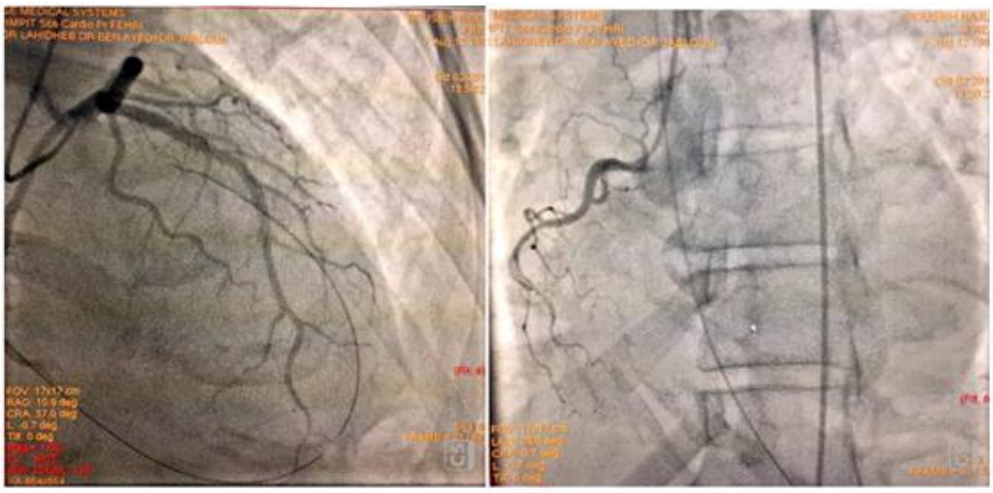

Fig-2: Coronarography A: Left coronary artery B: Right coronary artery

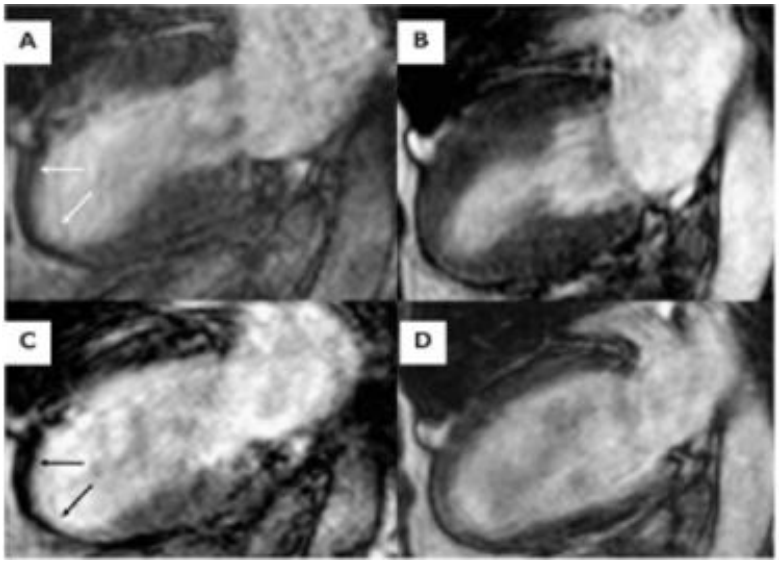

Fig-3: MRI

DISCUSSION

Our patient had Tako-Tsubo syndrome induced by emotional stress leading to cardiogenic shock and cardiocirculatory arrest. Most patients with TTS are usually paucisymptomatic and have a favorable evolution in-hospital course. However, adverse events due to hemodynamic instability (e.g., acute heart failure or cardiogenic shock) may occur in up to one-fourth of patients, even during the first hours after clinical onset. A study showed that higher WMSI and lower LVEF were associated with pulmonary edema and cardiogenic shock (CS), confirming the fact that marked LV systolic dysfunction contributes to the development of hemodynamic instability. Increased sympathetic stimulation seems to play a central role in the occurrence of systolic impairment related to myocardial stunning in TTS patients, through avariety of pathophysiological mechanisms, including myocyte calcium overload, oxidative stress, and micro vascular dysfunction. Lower peak levels of cardiac enzymes are observed in TTS than in acute coronary syndrome, despite more pronounced myocardial dysfunction. The slight elevation of troponin and CK-MB levels seems to be associated with myocardial stunning rather than necrosis, as abnormal troponin levels do not necessarily indicate true myocardial damage.

Wittstein *et al*. [1] reported that sudden elevations in plasma epinephrine levels after emotional or physiological stress are possibly involved in the pathogenesis of TTS, while the pathophysiologic mechanism underlying the deteriorating effect of cathecolamines is not fully understood. Similarly, we previously reported that a unique pattern of transient myocardial dysfunction can occur after severe emotional or physical stress which may induce abnormal activation of adrenoceptors and catecholamine induced myocardial damage. In our case, her anxiety and tensionafter her mother's death may be considered as a trigger of acute emotional stress. However, in this case, we can't confirm the association between a surge of catecholamines and the onset of TTS because we did not examine the plasma concentration of catecholamines during her hospital day.

In our case the deficit of C protein may have an impact in this acute coronary syndrom. A study investigated the possible involvement of the prothrombotic gene variants as risk factors of AMI in different gender and age subjects. The data indicate that the FV Leiden and FII G20201A variants in females represent risk factors for young AMI, namely, patients who developed the first episode of AMI before reaching the age of 45. The FV Leiden is associated to resistant inhibitory effect of protein C that enhances a procoagulant activity, while the FII G20210A variant causes higher levels and more pronounced procoagulant activity of FII, both of which may predispose to ischemic diseases [2]. Therefore, this study reported a significant association between some prothrombotic gene variants and the occurrence of young AMI in females.

In the literature a case of TTS complicated by cardiogenic shock that was successfully managed with Milrinone has been reported. The major pathophysiological phenomenon involved in TTS is considered as disproportionate catecholamine discharge in response to stress. This "catecholamine rush" is thought to saturate beta receptors and to literally stun the myocardium. At supraphysiological concentrations, catecholamines could result in beta-receptor paradoxical negative inotropic effects[1] [3]. Cardiogenic shock is mostly treated with dobutamine or epinephrine. But in TTS patients, these catecholamines could, given their beta agonist action, worsen myocardial stunning. Milrinone is a well-known non-

catecholamine inotrope that is used less than catecholamines. By inhibiting type III phosphodiesterase, Milrinone increases the calcium influx and improves myocardial contraction without any beta agonist action. This drug also decreases the systemic vascular resistance and pulmonary capillary wedge pressure. In our case she was just put on norepinephrine infusion (1mg /h) and fluid expansion.

Also the paper by Almendro-Delia *et al*., [4] published in a recent issue reporting on the outcome of TTS patients who experienced, CS elicited great interest among researchers in the present authors' working group. The authors found that CS is a common complication in TTS, with a prevalence of 11.4%. They concluded that CS is associated with a decline in short- and long-term prognosis.

Of note, the pathophysiology of TTS remains lacking. A hypothesis of acute catecholaminergic myocardial stunning causing wall motion abnormalities has been debated[1]**.** Recently published data suggest that catecholamine use reduces the short- and long-term survival of TTS patients[5]. It has been recommended that catecholamine use in TTS patients be avoided or withdrawn because it prolongs or exacerbates the acute phase of the disease[6]. However, the need for alternative circulatory support in CS among TTS patients has been evaluated, and it showed a different rate of mortality depending on the sort of catecholamine used. Studies investigated the successful use of the calcium sensitizer levosimendan in a series of TTS patients.

Finally, there are limited data for CS in TTS. A recent study published by the American college of cardiology [7] demonstrated that there are significant differences in clinical characteristics and outcomes of patients with CS due to TTS compared to that due to acute myocardial infarction.

CONCLUSION

Complications of TTS are thought to be infrequent and different from those of the acute coronary syndrome, although there is inadequate literature evaluating the true incidence of these complications such as heart failure, cardiogenic shock, ventricular arrhythmias, ventricular rupture, and death. The prognosis of patients experiencing TTS is generally favorable, with normalization of wall abnormalities within weeks.

In conclusion, patients who present acute coronary syndrome with normal coronarography should be hints for us to consider TTS more often as it can occasionally lead to CS.TTS presenting with CS represents a difficult challenge in intensive care units that's why early diagnosis is necessary for an appropriate medical care.

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