

## Tako-Tsubo cardiomyopathy in an older woman with hyperkinetic delirium

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Tako-Tsubo syndrome is a cardiological condition, mainly affecting postmenopausal women, that mimics an acute heart attack, because of a similar clinical presentation, ECG changes and altered laboratory tests. There is general consensus that the common etiology is a sudden and strong emotional or physical stress. We report here the case of an 80-year-old woman who developed a Tako-Tsubo syndrome during an episode of hyperkinetic delirium. Delirium is an acute, transient, usually reversible neuropsychiatric syndrome, considered a serious and stressful condition in older patients admitted to both medical and surgical settings. This clinical case expands our knowledge on the negative consequences of delirium in hospitalized patients and describes an additional risk factor for Tako-Tsubo syndrome in older people.

**Key words:** Tako-Tsubo cardiomyopathy, apical ballooning syndrome, elderly, hyperkinetic delirium, dementia

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## INTRODUCTION

Tako-Tsubo syndrome (TTS), also known as the broken heart disease, was first described in the 90's in Japan. The name of the disease has originated from the pot ("tsubo") with a narrow neck used as an octopus ("tako") trap. TTS is characterized by reversible left ventricular apical ballooning without a significant coronary artery disease. Its onset is acute with chest pain and/or dyspnea, occasionally shock and syncope, associated with electrocardiographic changes (ST segment elevation and/or deep symmetric T-wave inversion, QT prolongation). Cardiac enzymes may be normal or elevated. Atypical presentation includes epigastric pain, nausea and vomiting, seizures <sup>1</sup>.

There is general consensus in the scientific community that the common etiology is a sudden and strong emotional or physical stress, such as natural disasters, family mourning, subarachnoid hemorrhage, chemotherapy, surgical interventions, delirium tremens due to alcohol or drugs withdrawal. It is hypothesized that these stressful conditions might cause a surge in catecholamine levels that, in turn, induces microvascular spasm and endothelial dysfunction or a direct catecholamine-associated myocardial toxicity. The impaired microvascular reactivity is selectively localized at mid and apical segments of the left ventricle with the consequent transient apical ballooning. Its main characteristic is the hypokinesis of the apical and distal inferior wall, and the compensatory hyperkinesis of the basal

antero-inferior wall commonly assessed by transthoracic echocardiography<sup>2</sup>.

TTS mainly affects postmenopausal women, and mimics an acute heart attack, with its clinical presentation, ECG changes and altered laboratory tests. The prevalence is 1-2% among patients presenting to the hospital with suspected acute coronary syndrome, and the prognosis is usually good. TTS complications include pulmonary edema, heart failure, arrhythmias, hypotension. Malignant arrhythmias such as tachycardia and ventricular fibrillation, cardiogenic shock or free wall rupture and death are more severe and rare complications (< 1%). Moreover, TTS may happen again in another stressful event situation.

Generally, the management of the syndrome is conservative throughout supportive therapy, but in some cases may include aggressive treatment, such as inotropic agents or intra-aortic balloon pump.

Delirium, an acute, transient, usually reversible neuropsychiatric syndrome, is associated with different negative outcomes in older people. According to the available literature, the first therapeutic approach is non-pharmacologic based on the identification and treatment of medical or iatrogenic underlying causes<sup>3</sup>. In this case report we describe a case of Tako-Tsubo cardiomyopathy developed in an older hypertensive woman with hyperkinetic delirium.

## CASE PRESENTATION

An 80-year-old woman was admitted to the Geriatrics Unit with acute confusional state associated with aggressive verbal and physical behavior.

She had a clinical history of previous smoking habit, hypertensive cardiomyopathy and a recent diagnosis of atrial fibrillation. Moreover, few months ago she underwent oophorectomy for a voluminous ovarian cyst, complicated by heart failure with pericardial effusion.

The home therapy was the following: warfarin 19 mg/weekly, ramipril 5 mg/die, amlodipine 10 mg/die, bisoprolol 5 mg/die, furosemide 50 mg/die and canrenone 50 mg/die.

She did not drink alcohol, or use illicit drugs. She did not refer any allergy.

In the last month a progressive psycho-organic decay was reported with impairment in instrumental (IADL) and basic (BADL) activities of daily living ability, need for caregiver, behavior disorders such as psychomotor agitation, delusions (poisoning), aberrant sleep-wake cycle, fluctuating disorientation, rejection of therapy and food refusal.

At admission physical examination, clinical signs of malnutrition, dehydration and muscular hypotrophy

were pointed out. A suspicious attitude and opposition to the medical examination have been also shown. Vital signs were: blood pressure 130/70 mmHg, heart rate 90 bpm regular, body temperature 36.5 °C, peripheral oxygen saturation 96% without any respiratory support. Electrocardiogram and chest radiograph were normal. Laboratory tests showed a renal function slightly altered with creatinine level 1,8 mg/dl (reference range 0,5-1,2 mg/dl), eGFR according to CDK-EPI 25.1 ml/min (reference range  $\geq$  90 ml/min), and urea 154 mg/dl (reference range 17-43 mg/dl); a normocytic-normochromic anemia condition with hemoglobin 10,2 g/dl (reference range 11,5-16,5 g/dl) was also present.

At second day of hospitalization, the patient developed syncope, without chest pain or dyspnea, associated with electrocardiographic abnormalities: T-wave inversion in leads V1-V6, DI-II-III, aVL, aVF; QT prolongation – 648 ms (reference range 360-480 ms) (Fig. 1). Apical hypokinesia with severe left ventricular failure (ejection fraction 35-40%) was present at transthoracic echocardiography. Blood tests revealed cardiac enzymes increase: troponin values 4284 ng/l (reference range < 12 ng/l), CK-MB 8,10 ng/ml (reference range 0-5 ng/ml), BNP 1379 pg/ml (reference range 0-100 pg/ml).

The other exams excluded myocarditis, pneumonia, SARS-CoV-2 infection, cancer, electrolytes and thyroid disorders. Neuroimaging did not show acute ischemic lesions but only microvascular chronic pathology. The electroencephalogram ruled out epileptic abnormalities.

Beta-blocker, ACE-inhibitor, and diuretic were administered, together with adequate hydration and nutrition.

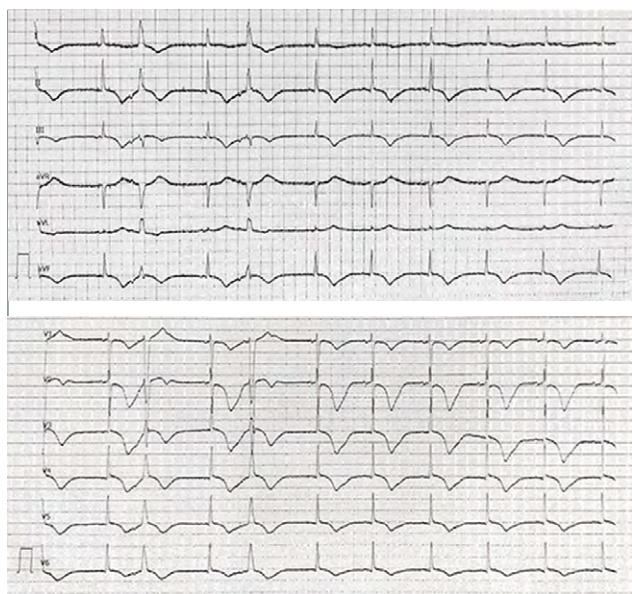
After five days, a second transthoracic echocardiography was performed and showed regression of segmental wall motion abnormalities, with normalization of the ejection fraction values (55%). A chronic, ubiquitous pericardial effusion was also shown.

During the hospitalization we gradually witnessed an improvement of behavioural symptoms.

Blood tests also showed an improvement, in particular troponin decreased until to 42 ng/l, CK-MB to 3,00 ng/ml, BNP to 120 pg/ml and creatinine levels to 0,81 mg/dl.

The patient was discharged after 28 days, in a nursing home, with the following diagnosis: "Probable Tako-Tsubo syndrome during hyperkinetic delirium in patient affected by pre-renal kidney failure, hypertensive cardiomyopathy, atrial fibrillation, pericardial effusion, vascular chronic encephalopathy". She was asymptomatic with normal cardiac enzymes blood level.

At discharge, the comprehensive geriatric assessment revealed a complete dependence in BADL (2/6 – able to walk and feed autonomously) and IADL (0/8). The



**Figure 1.** Standard 12-lead electrocardiogram performed at second day of hospitalization.

Short Portable Mental Status Questionnaire was 6/10 with a suspect of major neurocognitive disorder onset.

## DISCUSSION

The case presented here, is the first description of TTS case triggered by an episode of hyperkinetic delirium in 80 years old woman, in a suspect major neurocognitive disorder, in the absence of chest pain and shortness of breath<sup>4</sup>.

As mentioned above, TTS is often caused by stressful and extreme events like intense physical illness, surgery or psychological stress, as seen in our patient<sup>1,2</sup>.

In our case a cumulative emotional stress, namely repeated episodes of agitation and probably dementia onset, precipitated left ventricular wall-motion abnormalities with acute heart failure and TTS. The repeated echocardiographic examinations showed the transience of the left ventricular pathologic condition.

For the final diagnosis of TTS, we referred to the modified Mayo Clinic Criteria: transient hypokinesia or akinesia of the left ventricle with apical involvement; a stressful trigger present; absence of obstructive coronary disease; ECG abnormalities and cardiac troponin level elevation; exclusion of myocarditis or pheochromocytoma<sup>5</sup>.

After interventional cardiologic consult, given the discrepancy between signs (syncope), symptoms (absence of chest pain and dyspnea) and ECG abnormalities, was decided not to perform coronary angiography. Although TTS is still underrecognized, the incidence has steadily

increased, probably due to a combination of more effective diagnostic criteria and a likely rise incidence.

We ruled out acute pericarditis, myocarditis, pulmonary embolism, pneumonia, acute ischaemic event through-out blood and urine cultures, echocardiography, chest X-Ray and brain scan respectively.

Regarding on delirium, we supposed that it was in turn caused by acute kidney failure (eGFR 25.2 ml/min) due to a dehydration state, attested at clinical examination on admission. Indeed, the patient, that probably suffered by an undiagnosed dementia with behavioral and psychological symptoms of dementia (BPSD), was unable to sufficiently take care of herself at home.

TTS is a clinical diagnosis of exclusion therefore a high suspicion is needed to identify and manage it earlier. The differential diagnosis with life-threatening conditions is significant because TTS has more favourable prognosis and a complete recovery with supportive therapy.

Thus, in clinical practice, the underlying cause of delirium, a common acute pathological condition observed in older people, should be timely recognised and treated to prevent the associated negative outcomes, such as the development of TTS.

## CONCLUSIONS

In our case, the chronology of events suggests that hyperkinetic delirium, a major emotional stress trigger in a geriatric patient, induced Tako-Tsubo cardiomyopathy.

The peculiarity of the current clinical case is that TTS was diagnosed in an 80-year-old woman undergoing hyperkinetic delirium, probably triggered by pre-renal kidney failure in dehydration state, linked to a progressive loss of functional autonomy in the context of new onset dementia.

This case report reinforces the role of delirium as a negative prognostic factor in older patients and describes a new complication of this common geriatric syndrome.

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### Conflict of interest statement

The Authors declare no conflict of interest.

### Authors' contributions

Conceptualization: AV, FR, AS, MGC, GG, AZ, and SV; writing - original draft preparation: AV, FR, AS, MGC, and GG; writing - review and editing: AZ, and SV.

### Ethical consideration

The study has been performed in accordance with the ethical standards as laid down in the 1964 Helsinki

Declaration and its later amendments or comparable ethical standards.

#### Statement of human and animal rights

This article does not contain any studies with animals performed by any of the Authors.

#### Informed consent

Written informed consent for the publication of their clinical details was obtained from the patient.

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