

Clinical Case

Ultrarapid recovery in Takotsubo Syndrome: a case report

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ABSTRACT

Takotsubo syndrome (TTS) is a transient cardiomyopathy characterized by acute ventricular dysfunction that mimics acute coronary syndrome, usually recovering within days to weeks. We present the case of a 45-year-old woman with cardiovascular risk factors who, after an emotional stress event, developed chest pain and ST-segment elevation, initially treated as acute myocardial infarction with thrombolysis without reperfusion. Coronary angiography revealed unobstructed epicardial arteries, and ventriculography showed the classic "apical ballooning" morphology. Remarkably, the patient achieved complete clinical, electrocardiographic, and echocardiographic recovery in less than 24 hours under conservative management. This outcome represents an unusually rapid course compared to the literature. Our report expands the clinical spectrum of TTS and highlights the importance of recognizing ultrarapid presentations, as well as the need for multicenter studies to identify predictors of accelerated recovery.

INTRODUCTION

Takotsubo syndrome (TTS), also called stress-induced cardiomyopathy or "broken heart syndrome", is a transient cardiomyopathy characterized by acute ventricular dysfunction, usually with apical hypokinesis and preserved basal contraction, which resembles an acute coronary syndrome. It usually presents as chest pain, electrocardiographic changes and elevated cardiac biomarkers, but without significant obstructive lesions in coronary arteries. Pathophysiology is mainly associated with intense catecholaminergic discharges, coronary vasospasm and microvascular dysfunction. It mainly affects postmenopausal women, and ventricular function recovery typically occurs within days or weeks. We present a clinical case of TTS of ultrafast resolution, enhancing the spectrum of classical presentation of this entity.

CLINICAL CASE PRESENTATION

Female patient, aged 45, with cardiovascular risk factors: sedentary life, menopause, smoking and overweight. She presented to a first-level hospital with clinical symptoms characterized by precordial pain of cardiac characteristics after a verbal discussion. Initially, she received treatment with nonsteroidal anti-inflammatory drugs (NSAIDS) with no improvement, so when symptoms persisted, she went to a second-level hospital center six hours after the onset of the pain. Upon admission, the electrocardiogram (ECG) showed ST segment elevation in leads DI, AVL, V5 and V6 (*Figure 1*). Due to suspicion of ST-segment elevation acute myocardial infarction (STEMI), and the impossibility of performing primary coronary angiography within 120 minutes, thrombolysis was carried out with tenecteplase in a single 40 mg dose. Before the absence of indirect criteria for coronary reperfusion, the patient was referred to this hospital

for rescue percutaneous coronary interventionism (PCI). In the center, besides corroborating the electrocardiographic findings, Troponin I elevation of 16 ng/ml was documented (TriageTrue, Quidel; cutoff point <1 ng/ml). Coronary angiography showed epicardial arteries without significant obstructive lesions. In the ventriculography, basal hypercontractility and apical hypokinesis became evident, with classical morphology of apical ballooning (*Figure 2*). Thus, diagnosis of TTS was established, and the patient was admitted for clinical surveillance. During her stay she did not present new episodes of precordial pain, troponins decreased progressively, and ECG showed ST-segment resolution within just 16 hours (*Figure 3*). The echocardiogram carried out 24 hours after coronary angiography showed left ventricle with no alterations in segmental motion, with preserved systolic function (ejection fraction 54.1% by Simpson's biplane method) and ventricular geometry with concentric remodeling, so the patient was discharged the following day with hygienic and diet measures and no other pharmacological treatment. Currently, she is in NYHA functional class I (*Figure 4*).

DISCUSSION

TTS, also called stress-induced cardiomyopathy or "broken heart syndrome", is an increasingly identified entity in clinical practice. Although initially it was described in the Japanese population, currently there have been reports in different regions of the world, including Latin America. Its incidence is estimated to be 1-2% of patients admitted with suspicion of acute coronary syndrome (ACS), reflecting the significance of considering it in differential diagnosis.^{1,2}

Clinically, TTS usually presents with chest pain, dyspnea or syncope, accompanied by electrocardiographic changes and mo-



FIGURE 1.
Electrocardiography upon admission



FIGURE 2.
Images obtained in ventriculography

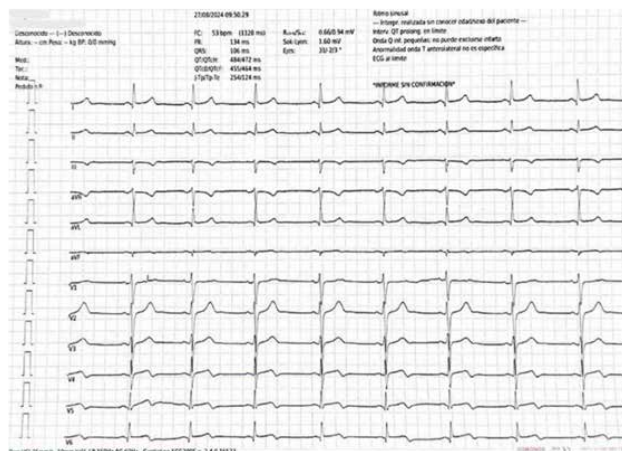


FIGURE 3.
Electrocardiogram 16 hours after admission

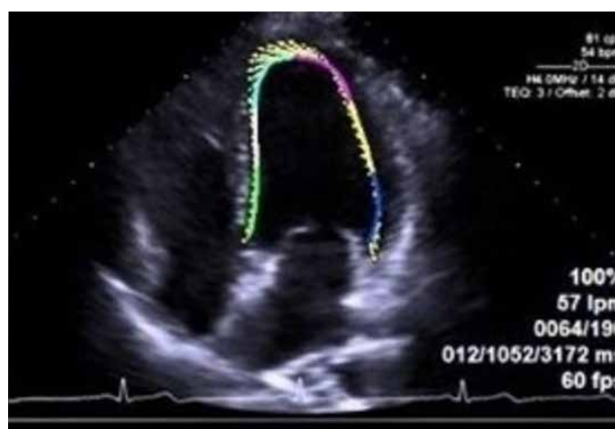


FIGURE 4.
Images of echocardiography 24 hours after admission

derate elevation in cardiac biomarkers, mimicking STEMI. In this regard, the absence of significant obstructive coronary lesions in angiography, together with transient ventricular alterations in ventriculography or echocardiography (generally the classical apical dyskinesia with basal hypercontractility), guide the diagnosis.^{3,4}

Pathogenesis is still being debated. The most widely accepted hypothesis links excessive catecholaminergic discharge with the trigger of transient myocardial dysfunction, although mechanisms related to coronary vasospasm, ventricular dysfunction and genetic susceptibility have also been described.^{5,6} Most cases occur in postmenopausal women, probably due to the loss of the protective effect of estrogens on the endothelium and adrenergic response.⁷ This clinical case coincides with this epidemiological profile, but presents a remarkably atypical outcome.

In most reports, recovery occurs in a period from days to weeks, defining an early resolution when ventricular function improves within the first 10 days after the initial event, reporting cases of improvement in up to the first three days; however, in this

patient, full resolution was documented within the first 24 hours under a conservative management, and only with clinical surveillance, representing a more rapid course than what is classically described.⁸ This finding suggests that there may be subgroups of patients with transient myocardial dysfunction of ultrashort course, probably influenced by factors such as adrenergic stimulation magnitude, previous myocardial reserve, or mechanisms yet to be clarified.⁹

The global prognosis of TTS is usually favorable, although not exempt from severe complications like heart failure, cardiogenic shock, ventricular arrhythmias or free wall rupture, with reported in-hospital mortality of up to 5%.¹⁰ In this regard, this case reinforces the significance of close monitoring in the cardiovascular care unit, even in patients with good clinical evolution, since variability in the natural course could be significant.

CONCLUSIONS

Takotsubo syndrome represents a diagnostic challenge due to its similarity with acute coronary syndrome. Although most ca-

ses show ventricular recovery within days or weeks, this patient presented a complete resolution in less than 24 hours under conservative management, constituting an unusually fast course. Reports like this one provide valuable information about the clinical range of TTS, particularly in the Latin American population, and highlight the need for multicenter studies to identify ultrafast recovery predictors in comparison with the usual course.

BIBLIOGRAPHY

1. Singh T, Khan H, Gamble DT, et al. Takotsubo syndrome: pathophysiology, emerging concepts, and clinical implications. *Circulation* **2022**; 145: 1002 – 1019.
2. Templin C, Ghadri JR, Diekmann J, et al. Clinical features and outcomes of Takotsubo (stress) cardiomyopathy. *N Engl J Med* **2015**; 373: 929 – 938.
3. Lyon AR, Bossone E, Schneider B, et al. Current state of knowledge on Takotsubo syndrome: A Position Statement from the Taskforce on Takotsubo Syndrome of the Heart Failure Association of the ESC. *Eur J Heart Fail* **2016**; 18: 8 – 27.
4. Pelliccia F, Kaski JC, Crea F, et al. Pathophysiology of Takotsubo Syndrome. *Circulation* **2017**; 135: 2426 – 2441.
5. Gianni M, Dentali F, Grandi AM, et al. Apical ballooning syndrome or takotsubo cardiomyopathy: a systematic review. *Eur Heart J* **2006**; 27: 1523 – 1529.
6. Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Takotsubo or stress cardiomyopathy): A mimic of acute myocardial infarction. *Am Heart J* **2008**; 155: 408 – 417.
7. Deshmukh A, Kumar G, Pant S, et al. Prevalence of Takotsubo cardiomyopathy in the United States. *Am Heart J* **2012**; 164: 66 – 71.
8. Jurisic S, Gili S, Cammann VL, et al. Clinical predictors and prognostic impact of recovery of wall motion abnormalities in Takotsubo syndrome: Results from the International Takotsubo Registry. *J Am Heart Assoc* **2019**; 8: e011194.
9. Bybee KA, Prasad A. Stress-related cardiomyopathy syndromes. *Circulation* **2008**; 118: 397 – 409.
10. Ghadri JR, Wittstein IS, Prasad A, et al. International expert consensus document on Takotsubo syndrome (Part I): Clinical characteristics, diagnostic criteria, and pathophysiology. *Eur Heart J* **2018**; 39: 2032 – 2046.