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**Author's Reply**

To the Editor,

We appreciate your interest and your comments in this case report detailing differential diagnosis of takotsubo syndrome (TS) especially in the absence of an obvious major stress (1).

TS is well known as the result of forced release of catecholamines secondary to psychogenic or physical stressors (2).

Based on the International Expert Consensus Document on TS, the stressor is not a requisite for the diagnosis of TS (3). Ac-

tually, absence of stressors has been reported in about one-third of patients with TS (4).

The pathophysiology of TS involves two main factors: first, the cognitive centers of the brain, the HPA (hypothalamic-pituitary-adrenal) axis, and the amount of catecholamines that are released in response to a stress and, second, the response of the cardiovascular and sympathetic nervous systems to this catecholamine surge (5). Based on these facts, the level of each component could be different among individuals leading to a wide variation of manifestations between them. Simply put, the threshold of that stressor could be lower in some patients.

Considering these facts, the term "spontaneous TS" seems inappropriate due to possible undetermined causal agents.

In fact, this patient had a familial quarrel before the presentation that could not be categorized as a major stress, but it might be severe enough for her which induced the takotsubo phenomenon.

This patient had no history of hypertension or evidences of hypertrophic cardiomyopathy, including septal hypertrophy and small left ventricle cavity. Any mid cavity gradient in the left ventricle was not detected during evaluation of the patient by echocardiography or ventriculography.

Any neurologic abnormality or any finding suggestive of autonomic disorder was not observed. However, the absence of these disorders was not observed precisely due to the absence of any neurologic sign that leads to further evaluation.

The last proposed mechanism also seems somewhat unusual due to some reasons: First, absence of robust evidences indicating myocarditis; second, extensive involvement of both ventricles that seems not in favor of mild form of myocarditis; third, complete resolution of cardiovascular magnetic resonance findings and absence of myocarditis evidences including late gadolinium enhancement; and finally, absence of myocarditis is one of the TS diagnostic criteria (6). In fact, if the patient had any evidence of possible myocarditis, the diagnosis of TS might be under debate and questionable. However, this mechanism was not completely ruled out as a triggering factor considering that possible coexistence of these two conditions makes the diagnosis more challenging even more due to different therapeutic and prognostic implications (7).

 **Sepideh Taghavi**,  **Maryam Chenaghloou<sup>1</sup>**,  
 **Marzieh Mirtajaddini<sup>2</sup>**,  **Nasim Naderi**,  **Ahmad Amin**  
**Department of Heart Failure and Transplantation, Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences; Tehran-Iran**  
**<sup>1</sup>Cardiovascular Research Center, Tabriz University of Medical Sciences; Tabriz-Iran**  
**<sup>2</sup>Cardiovascular Research Center, Kerman University of Medical Sciences; Kerman-Iran**

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**Address for Correspondence:** Maryam Chenaghloou, MD, Cardiovascular Research Center, Tabriz University of Medical Sciences, Tabriz-Iran  
Phone: +09144012182  
E-mail: mchenaghloou@yahoo.com  
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