# Takotsubo syndrome in the absence of an overt stressor: A glimpse into its mechanistic and clinical aspects

### To the Editor,

In the clinical setting, takotsubo syndrome (TS) is well known to be associated with adrenergic discharge mostly attributable to various emotional and physical triggers (1, 2). A recently published article by Taghavi et al. (1) has reported an interesting case of TS in the absence of an apparent stressor. This form of TS might be termed as "spontaneous TS" and might potentially mimic other cardiovascular conditions including myocarditis clinically (1). Accordingly, a few comments were made on this interesting case particularly emphasizing on its mechanistic and clinical implications:

First, evolution of "spontaneous TS" might be related to certain mechanical factors regardless of existing stressors particularly in patients with preexisting hypertensive heart disease or hypertrophic cardiomyopathy (HCM) (2, 3). That is, sudden increases in midventricular gradient (MVG) (possibly due to abrupt physiological changes) might elicit myocardial stunning in the left ventricular (LV) apical segments (leading to an apical ballooning pattern) possibly as a consequence of excessive myocardial wall tension in these segments (2). Therefore, the evolution of TS in this case (1) seemed possible to have a potential mechanical basis (2, 3). In this case, right ventricular (RV) dysfunction on initial echocardiogram might have been a secondary pathology (rather than direct TS involvement) associated with substantial LV morphological changes induced by the TS episode. Accordingly, we wonder whether the severity of left ventricular hypertrophy was also assessed along with resting and provoked MVG (if any) values in the patient.

Second, "spontaneous TS," though less likely, might also emerge due to subtle pathologies of the central or peripheral nervous system leading to bouts of adrenergic storm. In particular, involvement of the cardiovascular center in the brain stem (medulla oblongata) might potentially trigger TS episodes in the setting of multiple sclerosis (4), and possibly other central neurological conditions even if they are subclinically presenting with vague or no neurological deficit. Accordingly, we wonder whether any subtle neurological findings on physical examination or on imaging modalities were considered along with potential findings suggestive of an autonomic neuropathy (bouts of hypertension, diaphoresis, etc.) in the patient.

Lastly, "spontaneous TS" might rarely arise as a complication of acute myocarditis (5) that initially goes undetected due to its mild symptomatology. Accordingly, the disease process in this case (1) might have commenced as a mild form of myocarditis (as might be consistent with little or no late gadolinium enhancement on imaging) that ultimately turned into a true TS episode during the later stages. In conclusion; TS without an overt stressor (spontaneous TS) might be regarded as an interesting, yet; potentially underdiagnosed phenomenon with various mechanistic and clinical implications. However, further implications of this phenomenon still need to be established in clinical practice.

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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-

