Pathophysiological insights from dobutamine-induced Takotsubo syndrome

To the Editor,

I enjoyed immensely the case report by Hajsadeghi et al. (1) regarding the 74-year-old woman with Takotsubo syndrome (TTS) in the setting of dobutamine stress echocardiography (DSE) and the associated comprehensive meta-analysis of 22 similar patients from the international literature. The particulars of DSE-induced TTS are well presented and should act as a spring board for contemplating about the pathophysiology of TTS, which remains elusive, using data as the ones presented herein (1). I would like to engage the authors with some inquiries for their kind consideration: 1) Why do the authors refer to "catecholamine surge and alteration of responses to different types of receptors on the endocardium leading to microvascular dysfunction" (1), as opposed to receptors on cardiomyocytes throughout the ventricular wall thickness? 2) When we perform DSE, a baseline echocardiogram is obtained, followed by an echocardiogram at the peak pharmacological effect of dobutamine; one wonders about a stage of hypercontractility preceding the stage of regional wall motion abnormalities of TTS. Did the authors' review of the literature disclose any such information? 3) The authors documented that imaging in younger patients with TTS, undergoing DSE, revealed the reverse and mid-ventricular variants, rather than the apical variety of TTS (1); consequently do the authors have any thoughts about the effect of dobutamine, in particular, and the topographic distribution of the various types of β-adrenergic receptors in the ventricular myocardium, as a function of age?

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Reference

1. Hajsadeghi S, Rahbar MH, Iranpour A, Salehi A, Asadi O, Jafarian SR. Dobutamine-induced takotsubo cardiomyopathy: A systematic review of the literature and case report. Anatol J Cardiol 2018; 19: 412-6. [CrossRef]

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Author`s Reply

To the Editor,

We all know that a unifying explanation about the mechanism of takotsubo cardiomyopathy (TTS) remains questionable, but the main explanation is the overstimulation of beta 1 receptors in the heart (no difference between myocardium and endocardium) due to catecholamine surge. This phenomenon leads to microcirculatory dysfunction and direct cardiotoxicity, which results in severe myocardial morphological alterations (1). The observed alterations occur as myocardial histological changes, including focal mononuclear inflammatory areas of fibrotic response and characteristic contraction bands.

Based on observations in our patient and the literature review, a phase of hypercontractility preceding the stage of regional wall motion abnormalities is noted in most patients.

Observations of reverse and mid-ventricular types of TTS in younger patients have raised questions about the underlying mechanisms for us too, but further studies and literature review are needed to clarify the exact explanation.

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