Takotsubo syndrome in hypertrophic cardiomyopathy: Mechanical versus neurohumoral factors

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In clinical practice, left ventricular (LV) apical ballooning may be associated with purely mechanical factors including acute intraventricular obstruction [1–4]. This is in strict contradistinction with the widely recognized neurohumoral ballooning (classical takotsubo syndrome [TTS] pattern) strongly associated with adrenergic activation [2, 4]. The recent report by Ołpińska et al. [1] describes a case of apical ballooning in a young man with hypertrophic cardiomyopathy (HCM) with LV outflow tract obstruction (LVOTO) [1]. Accordingly, we would like to underscore a few points regarding this interesting case.

First, we hold the opinion that this case might also be regarded as a true TTS episode rather than an imitation of TTS (as suggested by the authors [1]). Apical stunning with subsequent ballooning due to sudden intraventricular obstruction (either in the form of LVOTO or midventricular obstruction) has been largely attributed to myocardial supply-demand mismatch associated with extreme apical intraventricular pressure, particularly in the presence of disturbed myocardial energy utilization [2–4]. This form of stunning has been generally encountered in patients with HCM or hypertensive heart disease mostly characterized by small LV cavities and septal hypertrophy, both of which significantly predispose to the evolution of sudden intraventricular gradient [2, 3]. On the other hand, severe hypertrophy was previously suggested to have a protective role against mechanical ballooning of the apex largely through a reduction in myocardial tension [2, 3]. Therefore, this form of ballooning usually emerges in the context of modest hypertrophy [2, 3]. Clinically, hemodynamic compromise is more likely to occur in patients with mechanical ballooning compared with those suffering from neurohumoral ballooning [2, 3]. Therefore, we wonder about the patient's hemodynamic status and the values of LV cavity dimensions and septal thickness [1]. What was the authors' therapeutic strategy for the prevention of extreme LVOTO recurrence?

Second, superimposed neurohumoral ballooning complicated by subsequent LVOTO may also have been possible in this patient [1]. Prolonged QT interval [1] may denote potential neurohumoral implications in this context. However, the patient reportedly had no physical or emotional stressor that might support the involvement of neurohumoral mechanisms. On the other hand, internal subtle stressors may also have pathogenetic implications in TTS evolution [4]. Notably, since most patients with mechanical ballooning have also been elderly and female [2, 3, 5], the presence of typical TTS demographic features is of no value to support the diagnosis of neurohumoral TTS in this context. On the other hand, certain clues including severe hypertrophy, absence of intraventricular gradient (at rest or provoked with challenge tests) following complete TTS recovery, and normal (or near-normal) mitral and papillary muscles may primarily suggest neurohumoral rather than mechanical factors in TTS evolution in those with HCM or hypertensive heart disease [2, 3, 5]. Did the authors [1] plan to provoke LVOTO recurrence following TTS recovery with challenge tests including dobutamine or exercise stress echocardiogram in their patient [2, 3]?

In summary, TTS evolution in patients with HCM or hypertensive heart disease might be associated with mechanical or neurohumoral factors (or both) [2, 3]. In these patients, the

identification of intraventricular mechanical factors as the substantial contributor to TTS evolution strongly warrants the management of these factors (*via* medical or invasive approaches) to prevent future TTS recurrences [1–3].

Article information

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