Rapid morphological transition during the course of Takotsubo syndrome: A mysterious phenomenon with subtle implications

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Factors and associated mechanisms that particularly predispose to the evolution of atypical morphological patterns [1–4] in the setting of takotsubo syndrome (TTS) have been poorly understood. As a general rule, it may be suggested that the higher the severity of initial adrenergic discharge, the more likely the evolution of atypical TTS variants (basal, global, etc.) [2, 3]. Therefore, these variants have been mostly associated with relatively extreme conditions such as pheochromocytoma with adrenergic crisis [2, 3]. On the other hand, the emergence of diverse morphological patterns (in a consecutive manner) [1–4] during a single TTS course seems to be even more atypical and enigmatic in the clinical setting. The recent article by Pan et al. [1] has described a case of TTS with a midventricular pattern (complicated by severe mitral regurgitation [MR]) that subsequently transformed into a classical apical ballooning pattern. Therefore, we would like to comment on further implications of this interesting case.

Notably, rapid morphological transition in the setting of TTS (during a single disease course) has been very rarely reported [1–4]. This may suggest it is underdiagnosed possibly due to certain factors such as lack of further serial echocardiographic imaging during the TTS course and late TTS presentation (after the established transition). Previously, TTS with a morphological transition pattern was also called "fast wandering TTS" [2, 4]. In particular, this phenomenon was observed in patients with pheochromocytoma-induced TTS [2, 3]. In general, pheochromocytoma-induced TTS has a higher likelihood of presenting with atypical morphological patterns mostly in the absence of an overt physical or emotional TTS trigger. It has worse in-hospital outcomes largely due to a variety of factors, including extreme adrenergic discharge, delayed diagnosis, and persistent myocardial abnormalities [2, 3]. Importantly, rapid transition from a regional to a global TTS pattern was also suggested to have prognostic implications in patients with pheochromocytoma-induced TTS [3].

Based on that, we suggest that the reported patient [1] needs to be further examined for potential pheochromocytoma (as the trigger of TTS) *via* imaging modalities and biochemical tests due to the suspicious findings (including the absence of a significant TTS trigger, relatively young age, initial presentation with an atypical TTS pattern followed by its rapid transition to another myocardial territory) [1]. Did the patient have signs of (or a history suggestive of) extreme adrenergic discharge such as coronary slow flow pattern on angiogram, paroxysmal severe hypertension, bouts of headache, and malignant arrhythmogenesis [2, 3]? If pheochromocytoma is identified as the trigger of the TTS episode, the presence of residual myocardial abnormalities may also be quite likely, and they need to be further investigated with advanced echocardiographic modalities (including strain, etc.), along with the management of pheochromocytoma [2].

Alternatively, the "fast wandering TTS" pattern may also arise in the absence of any organic source of extreme adrenergic discharge (including pheochromocytoma).

In certain TTS episodes, this dynamic pattern may simply emerge as a protective or counterbalancing mechanism against life-threatening mechanical complications such as acute MR, severe outflow tract gradient, and severe ballooning in the initially affected myocardial territory. In that patient [1], rapid transition of wall motion abnormalities from the mid-ventricle to the apical territory apparently terminated severe MR which, if persistent, might have led to acute pulmonary edema and/or hemodynamic compromise. In other terms, this "rapid transition pattern" [1] might have emerged as a critical physiological response aiming to abort acute MR, rather than being a coincidental phenomenon. Similarly, rapid transition of TTS-related wall motion abnormalities from the apex to other myocardial regions might possibly arise as a neutralizing mechanism against an impending or existing severe outflow tract gradient (a mechanical complication generally encountered in the setting of apical ballooning pattern [2]). On the other hand, rapid morphological transition may emerge in a small portion of patients with TTS [1–4] (even if they have mechanical complications or extreme adrenergic discharge) suggesting the pivotal role of patient-related factors in the evolution of this phenomenon. Notably, a significant individual variation may also exist in the clinical features of this phenomenon such as its temporal characteristics (early vs. late transitions, etc.), site of transition (to the neighboring or distant myocardial segment), and number of morphological transitions during a single TTS course (single vs. multiple). However, the above-mentioned ideas are largely speculative and need to be further investigated.

In conclusion, rapid morphological transition might have important pathogenetic and clinical implications in TTS patients [1–4] which still need to be established in detail.

Article information

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