

On The Nature of Transient Left Atrial Dysfunction in Patients with Takotsubo Syndrome

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One of the hallmarks of takotsubo syndrome (TTS) is the regional contraction abnormalities transiently affecting the left and occasionally the right ventricles. One wonders whether the left and right atria are involved in TTS, and if not, why the atria are spared. A recent relevant paper [1] rekindled these thoughts about a possible left atrial (LA) involvement in TTS. The authors compared 125 patients with TTS with 125 patients admitted with an anterior ST-segment elevation myocardial infarction, employing cardiovascular magnetic resonance (CMR)-based conventional LA volumetric indices, derived from fractional volume changes, and found lower total LA emptying fraction (EF), passive LA-EF, and active LA-EF in the former. Also in a subgroup of 20 of their TTS patients, who underwent a repeated CMR at follow-up, they detected recovery of LA function, as shown by the significant improvement in all 3 LA volumetric indices, between the CMR assessments carried out at the initial acute/subacute phase of the illness and at follow-up at a “median 3.3 months (IQR 3-5months) after the initial event” [1].

The authors appeared to be delving on the issue of the nature of the transient LA secondary dysfunction by first referring to the well-known pathological phenotypes of LA dysfunction in association with hypertension, cardiomyopathy, and heart failure. Also they analyzed the LA function in a few patients with TTS who had rapid restoration of left ventricular (LV) function to ascertain whether the LA dysfunction in these patients was a primary or secondary problem, and indeed these patients had milder LA dysfunction. All in all the authors felt that effect of TTS on the left atrium cannot be supported” by their data “and remains to be proven in future trials” [1].

The authors may be right that the transient LA dysfunction in patients with TTS is probably due to the associated LV dysfunction, and thus such dysfunctions move *pari passu*, when they emerge, progress, abate, and finally are restored to normalcy. However we should persist in exploring

whether there is transient LA dysfunction of the nature seen in association with the LV and right ventricle in TTS. Perhaps we should be exploring whether there are patterns of regional wall motion abnormalities in LA, which will qualify for “real” or “primary” TTS involvement of the LA, as opposed to LA dysfunction due to LV dysfunction. Histological study on the distribution of autonomic nerves in the human heart [2] has shown that the sympathetic autonomic innervation density of the atria is higher than the one of the ventricles, and thus the sympathetic autonomic seethe associated with TTS must theoretically have an injurious impact on the atria. Perhaps our imaging methods are insensitive to discriminate the emergence of regional wall motion abnormalities in LA in TTS, or the atrial β -adrenergic receptors are less sensitive than the ones of the LV, particularly of its apex, to norepinephrine [3], or circulating blood-borne epinephrine [4]. No matter what is the case, we should be continuously looking!

Conflict of Interest

There are no conflicts of interest to disclose.

References

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