Left ventricular aneurysm formation in patients with takotsubo syndrome: A peculiar phenomenon with subtle implications

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Short title: Ventricular aneurysm formation in Takotsubo syndrome

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In clinical practice, mechanical complications including left ventricular (LV) aneurysm formation (true or pseudo aneurysms) have been very rarely reported in patients with takotsubo syndrome (TTS) [1]. In their recently published article, Lozo et al. [1] have reported an elderly female patient with a mid-apical TTS who, following her discharge, was found to have an LV true aneurysm apparently complicated by a giant LV pseudoaneurysm formation after several months. This didactic case might yield important implications:

Besides the widely recognized theory of “adrenergic myocardial stunning”, certain mechanical factors including acute midventricular or outflow tract obstruction might also trigger TTS evolution in certain subjects particularly in those with hypertensive heart disease or hypertrophic cardiomyopathy [2]. Mechanistically, sudden increases in LV apical wall stress might possibly lead to apical ballooning due to mechanical myocardial stunning [2]. On the other hand, chronic severe midventricular gradient might also account for progressive apical aneurysm formation through augmented myocardial remodeling in this group of patients [2]. In this context, transient apical ballooning and progressive apical aneurysm formation might strongly mimick each other on initial imaging [2]. However, certain features including lack of regression (or even progressive expansion) on follow-up, considerable wall thinning and substantial late gadolinium enhancement (LGE) on magnetic resonance imaging (MRI)
suggests an existing aneurysmal sac rather than apical ballooning [2]. Interestingly, they may also co-exist in certain patients with an intraventricular mechanical trigger. Accordingly, the patient reported by Lozo M, et al. might have harbored an intraventricular gradient (resting or provoked) potentially giving rise to both TTS evolution and apical aneurysm formation (possibly at different times) [1]. In other words, true aneurysm formation in the setting of TTS might not be regarded as a direct complication of TTS itself (that is well known to be devoid of myonecrosis), yet might be labeled as a separate entity potentially associated with the above-mentioned mechanical factors [2]. It seems quite possible that transient apical ballooning pattern might have superimposed on an already existing true apical aneurysm in the patient [1]. However, it seems challenging to detect a co-existing true aneurysm located within the territory of apical ballooning particularly in the presence of a substantial mural thrombus formation (as in the patient [1]). These mechanically-triggered true aneurysms are more likely to be diagnosed as residual structures following complete recovery of the TTS episode, and are very rarely complicated by a pseudoaneurysm formation (as in the patient [1]) due to the absence of substantial myonecrosis.

Furthermore, co-existing acute coronary syndromes (ACSs) mostly in the form of MINOCA [1, 3] might alternatively lead to the LV true aneurysm formation in patients with TTS [4]. Therefore, possible coronary embolism originating from the mural thrombus might have also led to the LV aneurysm formation [3, 4] in the patient. Of note, these necrotic aneurysms might not necessarily involve the apex, and might also arise in other territories including the posterobasal segment [4] in which case their detection might be relatively easier on echocardiogram. MINOCA-related true aneurysms might occasionally be complicated by pseudoaneurysm formation largely depending on the extent of myonecrosis [4]. Enhanced myocardial fragility and/or adverse impact of increased intraventricular pressure (due to intraventricular mechanical factors or hypertensive episodes) may serve as important triggers of both true and pseudo aneurysm formation [4] potentially suggesting certain preventive strategies including volume loading, uptitration of beta blockers and withdrawal of agents impeding myocardial healing in patients with a co-existence of TTS and possible MINOCA. In these patients, pseudoaneurysms may either arise as a ‘de novo’ phenomenon or may complicate true aneurysms (as in the patient [1]) [4]. Finally, MRI serves as a perfect tool to confirm a co-existing MINOCA, and to examine the details of associated aneurysms in patients with TTS [3, 4]. Taken together, we wonder about signs of MINOCA including coronary slow flow phenomenon, etc. and pattern of mural LGE on MRI (around the base of pseudoaneurysm)
along with findings of specific myocardial disease (small LV cavity dimension, any resting or provoked intraventricular gradient, etc.) in the patient [1].

In conclusion, LV aneurysm formation in the setting of TTS may be regarded as a rare phenomenon with poorly understood mechanisms [1]. In patients with TTS, intraventricular mechanical factors (including midventricular gradient) and co-existing MINOCA might serve as clinical risk factors for LV aneurysm formation (both true and pseudo aneurysms) [2–4]. This potentially warrants timely and proper management of these risk factors to improve TTS outcomes.

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