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

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DOI: 10.33963/KP.a2023.0161

**Received:**
July 2, 2023

**Accepted:**
July 4, 2023

**Early publication date:**
July 14, 2023

In clinical practice, mechanical complications including left ventricular (LV) aneurysm formation (true or pseudoaneurysms) 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 mid-apical TTS who, following her discharge, was found to have an LV true aneurysm complicated by a giant LV pseudoaneurysm formation after several months. This case might yield important didactic 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 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 mimic 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) suggest an existing aneurysmal sac rather than apical ballooning [2]. Interestingly, these features 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 a transient apical ballooning pattern might have been superimposed on an already existing true apical aneurysm in that 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 from a 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 LV true aneurysm formation in TTS patients [4]. Therefore, a possible coronary embolism originating from the mural thrombus might have also led to LV aneurysm formation [3, 4] in that patient. Notably, 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 pseudoaneurysm formation [4]. This potentially suggests certain preventive strategies, including volume loading, up-titration 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 co-existing MINOCA and to examine precisely associated aneurysms in TTS patients [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 the 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 TTS patients, intraventricular mechanical factors (including midventricular gradient) and co-existing MINOCA might serve as clinical risk factors for LV aneurysm formation (both true and pseudoaneurysms) [2–4]. This potentially warrants timely and proper management of these risk factors to improve TTS outcomes.

## *Article information*

**Conflict of interest:** None declared.

**Funding:** None.



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