

## Takotsubo syndrome-induced acute myocardial infarction

John E. Madias

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Dear Editor,

The elegantly documented case report by Kato et al. [1], published on line ahead of print on January 28, 2015 in the *Journal*, about a 71-year old woman who suffered mid-ventricular Takotsubo syndrome (TTS), did not seek medical attention immediately, and was 2 days later admitted with a small myocardial infarction due to an occluded septal branch of the distal left anterior descending coronary artery, exemplifies the state of TTS as a comorbidity in patients with acute myocardial infarction (AMI) [2], and more specifically in this case, the potential of TTC to induce an AMI. Although in cases when TTS and AMI coexist, authors engage in the proverbial causality dilemma of “which came first, the chicken or the egg?”, the case herein as discussed appears to this reader to be an example of an AMI caused by a preceding TTS [1], unlike of a recent case of typical TTT with apical ballooning precipitated by an inferior myocardial infarction [3]. The hypothesis of the authors of a “mechanical stress around the distal hinge point of anterior wall produced intimal injury of nearby small coronary arteries, which resulted in acute thrombosis”, appears plausible, as a pathogenetic mechanism of TTS. One wonders whether other phenomena, like e.g. coronary vasospasm are also consequences of the mechanical stresses resulting from TTS, and not the cause of TTS [4].

Recently an electrocardiogram (ECG) feature, characterized by transient attenuation of the voltage of the QRS complexes (AVQRS), attributed to myocardial edema (ME), has been reported in patients with TTS [5], and was found to be “myocardial territory involved”-specific, in the sense that patients with mid-ventricular TTS, had AVQRS in ECG leads I and aVL [6]. Since the present case included documentation of ME in the mid-ventricular myocardium by cardiac magnetic resonance imaging, did the patient had transient AVQRS in leads I and aVL, as assessed by a comparison of all her available ECGs?

**Conflict of interest** The author does not have any competing interests to disclose.

### References

1. Kato K, Sakai Y, Ishibashi I, Kobayashi Y (2015) Mid-ventricular takotsubo cardiomyopathy preceding acute myocardial infarction. *Int J Cardiovasc Imaging*. doi:10.1007/s10554-015-0602-5
2. Madias JE (2014) Why the current diagnostic criteria of Takotsubo syndrome are outmoded: a proposal for new criteria. *Int J Cardiol* 174(3):468–470
3. Koeth O, Zeymer U, Schiele R, Zahn R (2010) Inferior ST-elevation myocardial infarction associated with takotsubo cardiomyopathy. *Case Rep Med* 2010:467867. doi:10.1155/2010/467867
4. Madias JE (2014) Coronary vasospasm is an unlikely cause of Takotsubo syndrome, although we should keep an open mind. *Int J Cardiol* 176:1–5
5. Madias JE (2014) Transient attenuation of the amplitude of the QRS complexes in the diagnosis of Takotsubo syndrome. *Eur Heart J Acute Cardiovasc Care* 3:28–36
6. Madias JE (2013) Electrocardiogram lead-specific QRS attenuation in an atypical midventricular case of Takotsubo syndrome. *J Electrocardiol* 46:728–729

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J. E. Madias  
Icahn School of Medicine at Mount Sinai, New York, NY, USA

J. E. Madias (✉)  
Division of Cardiology, Elmhurst Hospital Center, 79-01  
Broadway, Elmhurst, NY 11373, USA  
e-mail: madiasj@nychhc.org