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EDITORIAL COMMENT

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Inquiries about a patient with a "snail-like" takotsubo syndrome variant

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This editorial accompanies the article on page 1051

A fascinating case report by Genc et al. [1], accompanied by a very impressive video of a patient with a "snail-like" focal takotsubo syndrome (TTS) variant underscores the contribution of videos in the appreciation of regional myocardial contraction abnormalities (RMCAs) in patients with TTS, and particularly in patients with "focal" abnormalities where such RMCAs are expected to be limited in extent. In addition, an inherent differential diagnosic problem (not encountered in the present case) is that "focal" TTS often needs to be differentiated from RMCAs related to an acute coronary syndrome, conventionally associated with a single culprit coronary artery occlusion or atherosclerotic plaque destabilization, while RMCAs in patients with non-focal TTS are more extensive occupying myocardial regions subtended by more than one coronary artery (e.g., apical, midventricular, and reverse TTS variants) comprise component RMCAs involving septal, anterior, inferior, and lateral myocardial territories, which would not be expected to be associated with a blood flow-limited occlusion or critical stenosis of a single coronary artery. I understand that the format of the paper presenting this case report was such that did not permit the authors [1] to include more details regarding this 70-year-old woman with TTS; however due to the rarity of the "focal" TTS variant, a few details provided by the authors, may be of value to the readers and investigators alike. This belief prompts me to request the authors' kind response on the following comments and inquiries: 1) The patient's lack of symptoms fits with the low troponin I and B-type natriuretic peptide, particularly the latter, value levels; 2) Was there a repeat echocardiogram done during hospitalization, and what were the findings?; 3) Was there a regional longitudinal strain (Bull's Eye Plot) carried out in the follow-up echocardiogram(s)?; 4) Was the patient on any drug therapy prior to her presentation, including hormones like estrogens or progestins [2]?; 5) Did the QTc shorten in subsequent electrocardiograms, and when, in the follow-up course?; 6) The electrocardiogram changes of ST-segment elevation in leads II, III, and aVF are incongruent with the topography of the left ventricular "focal" RMCAs (e.g., one would have expected such RMCAs to be associated with some changes in the precordial electrocardiogram leads and/or leads I and aVL [3, 4]; 7) What was the electrocardiogram evolution during hospitalization?; 8) Did the patient develop inverted T-waves, and in what electrocardiogram leads?

Conflict of interest: None declared

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