LETTER TO THE EDITOR

Electrocardiogram in Takotsubo Syndrome

John E. Madias, M.D., F.A.C.C., F.A.H.A.

From the Icahn School of Medicine at Mount Sinai, New York, NY and the Division of Cardiology, Elmhurst Hospital Center, Elmhurst, NY

Ann Noninvasive Electrocardiol 2015;20(4):405

Dear Editor,

I enjoyed reading the comprehensive review on the electrocardiogram (ECG) in Takotsubo syndrome (TTS) by Duran-Cambra et al.,¹ published ahead of print on November 4, 2014 in the *Journal*. I have the following remarks for the authors' consideration: (1) It is possible that the ST-segment elevation in TTS is due to ischemic injury or "injury" of some kind, akin to what one encounters in pericarditis; the ischemic mechanism is unlikely because many studies have shown normal myocardial perfusion early in the clinical course. Perhaps the ST elevation is of the type seen in association with dyskinesia in chronic myocardial infarction, due to a displacement of the ECG curve, rather than true ischemic injury.² (2) The authors refer to Q waves, "more commonly in leads V₃ and V₄," and this is in keeping with the special topography of the edematous myocardium of the apex, reflected on the mid-precordial leads, which are devoid of any myocardial elements opposite to it, to produce electrical cancellations, as they occur between the septal-lateral, and anterior-posterior regions of the ventricle, and thus leading to transient Q waves. Indeed, the same mechanism is responsible for the observation of negative ST-segment in aVR, or absence of ST-elevation in V₁, of Kosuge et al.,³ resulting from an unopposed ST-segment injury vector, generated by the edematous apex. (3) Myocardial edema most probably is at the roots of the attenuation of the QRS seen early in the clinical

course of TTS,³ but to exploit this observation, and reach a conclusion that one deals with TTS and not an acute myocardial infarction, there is a need to compare the admission ECG of the patient under evaluation, with previous ECGs obtained in other hospitals, or even other geographical locations; this can be feasible via smart phones, smart personal health data cards, and cloud computing.⁴ Because this comparison is often missing, one is only left with a comparison of the admission ECG with subsequently recorded ECGs, revealing often increase in the amplitude of QRS complexes, in ECGs prior to the patient's discharge (seen in most of the leads of Figure 4 of the authors' article, between "admission" and "day 5"), or at follow-up, which observation, of course, does not contribute to our goal of an early diagnosis of TTS.

REFERENCES

1. Duran-Cambra A, Sutil-Vega M, Fiol M, et al. Systematic review of the electrocardiographic changes in the Takotsubo syndrome. *Ann Noninvasive Electrocardiol* 2014, Nov 14 [Epub ahead of print].
2. Madias JE. Is the ST-segment elevation in Takotsubo syndrome partially (or even totally) due to dyskinesia? *Am J Cardiol* 2013;111:778-779.
3. Kosuge M, Ebina T, Hibi K, et al. Simple and accurate electrocardiographic criteria to differentiate Takotsubo cardiomyopathy from anterior acute myocardial infarction. *J Am Coll Cardiol* 2010;55:2514-2516.
4. Madias JE. Transient attenuation of the amplitude of the QRS complexes in the diagnosis of Takotsubo syndrome. *Eur Heart J Acute Cardiovasc Care* 2014;3:28-36.

Address for correspondence: John E. Madias, M.D., Division of Cardiology, Elmhurst Hospital Center, 79-01 Broadway, Elmhurst, NY 11373; Fax: 718-334-5990; E-mail: madiasj@nychhc.org

Conflicts of interest: None.