LETTER TO THE EDITOR

Electrocardiogram peaked T-waves in a patient with reverse Takotsubo syndrome

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I read with great interest the report by Caracioni et al. [1], about a healthy 27-year old man who suffered reverse Takotsubo syndrome (rTTS) attack, complicated by SCAI Stage D cardiogenic shock (CS) in the setting of sepsis, requiring vasopressors and mechanical circulatory support with an Impella CPs device, kidney injury requiring renal replacement therapy, and posterior reversible encephalopathy syndrome. We should not have the slightest doubt that Takotsubo syndrome (TTS) is more frequent that is currently appreciated in the setting of sepsis, with or without CS, and that we should continue to study the TTS/sepsis association with or without CS closely, for an expedited diagnosis of TTS and its proper management [2,3]. The authors should be congratulated for a superb management of their patient, and for their thorough diagnostic assessment of the postulated underlying causes of their patient's illness.

The objective of this letter is to draw attention to an issue totally overlooked by the authors, i.e., the electrocardiogram (ECG) appearances, that are of utmost importance in view of the TTS variant (i.e., rTTS) their patient presented with; Fig. 1 of their paper shows an ECG with "increased T-waves over 25mV, observed in leads V3 and V4, with no pathological T-wave inversions" [1]. Previous work has commented on the relevance of the location of the underlying pathology (i.e myocardial edema) in a TTS variant and its association with the ECG repolarization changes [4,5] which is based on vectorial ECG assessment and previous reports of patients with rTTS [4,5]; accordingly, in patients with the "mid-basal TTS" or rTTS, one is expecting to see localized ST-segment depression in inferior leads II, III, aVF and precordial leads V3-V6, followed by peaked upright T waves and QT prolongation, the opposite of ST-segment elevation followed by progressively deeper T-waves, in the ECG leads displaying initially ST-segment elevation or depression, encountered in patients with the commonest and most prevalent apical TTS variant [4,5].

I have the following questions for the kind consideration by the authors: 1) Did this patient have another ECG from the initial presentation at the other hospital or at the "higher center" to which he was subsequently transferred? 2) Did such ECGs show ST-segment depressions and/or lower amplitude upright T-waves? 3) How many hours transpired between the onset of the patient's symptoms and the ECG of Fig. 1 [1]. 4) Did ECGs recorded subsequently to the ECG of Fig. 1 [1], show progressively taller T-waves? 5) Did any ECGs of this patient reveal QTc prolongation? [4,5] 6) How did the ECG at follow-up look like?

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