RESPONSE OF THE AUTHOR

RESPONSE: The Electrocardiographic Changes in Mid-basal (Inverted) Takotsubo Syndrome

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Dear Editor,

I read with great interest the recently published article by the authors Duran-Cambra et al.[1] published on November 4, 2014 in the journal ahead of print regarding systematic review of the electrocardiographic (ECG) changes in takotsubo syndrome (TS). The authors have carefully described the ECG changes in the classical TS, the time course of the ECG changes, and some other changes as QRS voltage attenuation. They have also described different ECG criteria for differentiation between anterior myocardial infarction and TS. With this letter, I would like to complement and highlight some other ECG changes, which also occur and are important to recognize in TS. The localization of the underlying pathology and the left ventricular dysfunction determine the ECG changes in TS. In mid-basal TS, also known as inverted TS, the ECG findings are the reverse of the ST elevation and the giant T-wave inversion seen in mid-apical TS. It is characterized by ST depression localized in the majority of patients with mid-basal TS in inferior leads II, III, aVF, and precordeal leads V_3 through V_6. These ECG changes are followed by peak upright T waves and QT prolongation in some patients. Mid-basal TS with the above-mentioned ECG changes is more frequently triggered by physical stress factors as stroke, pheochromocytoma, and therapeutic or accidental epinephrine administration but it may even be triggered by emotional stress factors. Waller et al.[2] have reported on a case of 46-year-old female with mid-basal TS triggered by subarachnoid hemorrhage and complicated by cardiogenic shock. The ECG in that patient revealed right bundle branch block and anterior precordial lead ST depression. Cho et al.[3] have reported only peak upright T wave and prolonged QT interval (QTc 467 milliseconds) in a patient with mid-basal TS triggered by pheochromocytoma. Magri et al.[4] reported on a patient with epinephrine-triggered mid-basal TS with ST sagging in leads II, III, aVF, V_5, and V_6, and nonspecific diffuse T-wave flattening. Follow-up ECG showed tall upright T waves in leads II, III, aVF, and V_3 through V_6. The tallest T waves were seen on day 3. Cacciotti et al.[5] have reported on emotional-induced midbasal TS showing diffuse ST segment depression and evolved with tall and pointed T waves in leads V_2–V_4. Interestingly, Byer et al.[6] in 1947 had reported on large upright T waves and long QT intervals induced by hypertensive encephalopathy; intracranial diseases are now well-recognized trigger factors for TS.

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